Animesh Agarwal
*Editor*

# Nonunions

## Diagnosis, Evaluation and Management

Springer

*Editor*
Animesh Agarwal

# Nonunions

## Diagnosis, Evaluation and Management

Springer

*Editor*

Animesh Agarwal
Division of Orthopedic Traumatology, Department of Orthopedics,
University of Texas Health Science Center, San Antonio, TX, USA

ISBN 978-1-4939-7176-3 e-ISBN 978-1-4939-7178-7
https://doi.org/10.1007/978-1-4939-7178-7

Library of Congress Control Number: 2017940364

© Springer Science+Business Media LLC 2018

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer Science+Business Media LLC
The registered company address is: 233 Spring Street, New York, NY 10013, U.S.A.

*We must find time to stop and thank the people who make a difference in our lives.*

—John F. Kennedy

*First and foremost, I would like to thank my parents, Jagdish and Kusum, for being extraordinary role models, as well for their hard work and dedication to parenthood. They have supported me throughout my life, both personally and professionally. I am forever grateful. They continue to amaze me now as grandparents.*

*To the most delightful, caring, and resilient children I could have ever had. Thank you Priya, Deven, and Trevor for being so understanding when I am on call or working late. You remind me what is most important in life every time I look at you. The three of you have been and continue to be my inspiration.*

*I want to thank those who have supported me professionally. I am lucky to have trained at an institution with many "giants" of orthopedics, and even more fortunate to have joined them as a faculty member. They have always treated me as an equal, despite having been my attendings. So thank you to Drs. James Heckman, Charles Rockwood, and Fred Corley, who all believed in me and gave me a chance. Thank you to those that trained me during my orthopedic trauma fellowship, Drs. Attila Poka, Robert Ostrum, and Brian Davison for making that year of training educational, fun, and unforgettable.*

*Last but not least, I would be remiss if I did not thank my two research mentors, Drs. Kyriacos Athanasiou and Mauli Agrawal. They were instrumental in fueling my curiosity, teaching me to write scientifically, and steering me towards orthopedics. I wouldn't be where I am today without their encouragement and guidance so early in my career.*

Animesh Agarwal, MD

Boerne, TX, USA

February 2017

# Foreword

It is an honour to be invited to write the foreword to this important contribution to the orthopedic surgical literature. This textbook is an extension of the robust fracture information and knowledge base originally created in the Department of Orthopedics at the Health Science Center in San Antonio by Rockwood and Green in the 1970s. Despite many substantial advances in primary fracture care over the last several decades, our ability to enhance fracture healing has not improved very much; thus, delayed unions and nonunions remain a common clinical challenge. Furthermore, as patients develop greater expectations for very good or even perfect results after injury, they are less willing to accept incomplete fracture healing as the final outcome. Today's patient wants to be able to return to high-level performance following injury and not be sidelined because of a failure of fracture healing. Therefore, when fracture healing does not occur, it is incumbent upon the surgeon to identify the cause and develop an effective solution to restore function in a timely manner. The textbook is a very useful resource to that end.

Dr. Agarwal has compiled a very comprehensive overview of the causes of and the effective treatments for virtually all of the common nonunion patterns. His contributing authors are authoritative and recognized experts in their respective fields, and the treatments for specific nonunions are presented in a very comprehensive, well-illustrated, and user-friendly way.

This textbook clearly advances our understanding of the evaluation and treatment of fracture nonunions, and it will be a very useful guide to the surgeon who must find an effective means to restore function when the normal fracture healing process has failed.

**James D. Heckman**
**Manchester, VT, USA**
**March 2017**

# Preface

An investment in knowledge pays the best interest.
—Benjamin Franklin

Nonunions have been a challenging aspect for orthopedic trauma surgeons. The literature is scarce with recommendations for many of these problems. The principles of nonunion management have been traditionally based on the type of nonunion and its aetiology, whether it is a mechanical or biological one. As orthopedic care has advanced with newer implants—such as locked plating—old problems have been alleviated, but new issues have arisen as well. Although patient factors and injury factors contribute significantly to the development of nonunions, iatrogenic causes are not infrequent. Orthopedic trauma care still requires attention to detail and the basic principles of fracture management to help prevent the surgeon's contribution to nonunion development.

Over the last twenty years of my practice, nonunions have always been a difficult problem to assess. New patient issues such as vitamin D deficiency have come to light. Infection all too often accompanies nonunions and must be evaluated. This text was designed to provide a single reference for the basic principles of nonunion diagnosis, evaluation, and management. Although not every single treatment option can be covered for every single anatomical area and type of nonunion, we hope that this text is useful, not only for managing these difficult problems but also in preventing nonunions from occurring by avoiding surgical causes and mitigating patient risk factors.

The contributors to this text were selected based on their interest and expertise in this subject. Nonunion management is unique in that it is rarely taught; rather, surgeons have learned over the years through their own personal experience, oftentimes by trial and error. Learning a new subject, technique, or gaining new knowledge requires the desire to be more well-informed. To quote the great Woody Allen, "Eighty percent of success is showing up". The contributors have certainly shown up. If you are reading this preface, we hope that you have taken a step towards "showing up", and thus being successful in the management of these difficult and challenging

cases.

**Animesh Agarwal**
**Boerne, TX, USA**

# Contents

# Contributors

**Animesh Agarwal, MD**
Department of Orthopedics, Division of Orthopedic Traumatology, UTHSCSA, San Antonio, TX, USA

**Joseph Borrelli Jr., MD, MBA**
Orthopedic Surgery, BayCare Medical Group, St. Joseph's Hospital-North, Lutz, FL, USA

**Fred G. Corley, MD**
Department of Orthopedics, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

**Brian P. Cunningham, MD**
Department of Orthopedic Surgery, University of Minnesota, Regions Hospital, St. Paul, MN, USA

**Kyle F. Dickson, MD, MBA**
Southwest Orthopedic Group, Baylor University, Houston, TX, USA

**Anthony Ding, MD**
Department of Orthopedic Surgery, San Francisco General Hospital, Orthopedic Trauma Institute, University of California San Francisco, San Francisco, CA, USA

**Ahmad Fashandi, MD**
Department of Orthopedics, University of Virginia, Charlottesville, VA, USA

**Ben S. Francisco, MD**
Department of Orthopedics, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA

**Aaron M. Freilich, MD**
Department of Orthopedics, University of Virginia, Charlottesville, VA, USA

**Samuel E. Galle, MD**
Orthopedic Trauma Service, Orthopedic Trauma & Fracture Clinic, Saint Alphonsus—Boise Campus, Boise, ID, USA

**Roman A. Hayda, MD**
Rhode Island Hospital, Brown University Warren Alpert School of Medicine, Providence, RI, USA

**Thomas F. Higgins, MD**
Department of Orthopedics, University Orthopedic Center, University of Utah Health Sciences, Salt Lake City, UT, USA

**Ethan S. Lea, MD, MSc**
Department of Orthopedic Surgery, University of California at Davis Medical Center, Sacramento, CA, USA

**Mark A. Lee, MD**
Department of Orthopedic Surgery, University of California, Sacramento, CA, USA

**Michael P. Leslie, DO**
Department of Orthopedics and Rehabilitation, Yale School of Medicine, New Haven, CT, USA

**Matthew Lyons, MD**
Department of Orthopedics, University of Virginia, Charlottesville, VA, USA

**Michael D. McKee, MD, FRCS(C)**
Division of Orthopedic Surgery, Department of Surgery, University of Toronto, Toronto, Canada

**Saam Morshed, MD, PhD, MPH**
Department of Orthopedic Surgery, San Francisco General Hospital, Orthopedic Trauma Institute, University of California San Francisco, San Francisco, CA, USA

**Gil R. Ortega, MD, MPH**

Department of Orthopedic Surgery, Mayo Clinic Arizona Orthopedic Residency Program, Mayo Clinic Arizona, HonorHealth Scottsdale Osborn Trauma Center, Scottsdale, AZ, USA

**Kevin J. Pugh, MD**
Orthopedic Trauma and Reconstructive Surgery, Grant Medical Center, Columbus, OH, USA

**Emil H. Schemitsch, MD, FRCS(C)**
Division of Orthopedic Surgery, Department of Surgery, University of Toronto, Toronto, Canada

**Laura A. Schemitsch, MA**
Division of Orthopedic Surgery, Department of Surgery, University of Toronto, Toronto, Canada

**Philip R. Wolinsky, MD**
Department of Orthopedic Surgery, University of California at Davis Medical Center, Sacramento, CA, USA

**David P. Zamorano, MD**
Orthopedic Trauma Service, Orthopedic Trauma & Fracture Clinic, Saint Alphonsus—Boise Campus, Boise, ID, USA

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_1

# 1. Principles of Nonunions

Animesh Agarwal[1]

(1) Division of Orthopedic Traumatology, Department of Orthopedics, University of Texas Health Science Center, San Antonio, TX, USA

**Animesh Agarwal**
**Email:** Agarwal@uthscsa.edu

**Keywords** Nonunion – Diagnosis – Evaluation – Management – Hypertrophic – Atrophic – Risks – Bone graft – Smoking – Vitamin D deficiency

## 1.1 Introduction

Fracture healing is a very unique process in the human body. Bone is a unique tissue in that it can regenerate itself during the process of healing. This requires a very complex process which is regulated by various metabolic and hormonal factors to include various growth factors. These biological processes occur at the cellular level requiring recruitment proliferation and differentiation of many cells including endothelial cells, osteoprogenitor cells, platelets, macrophages, mesenchymal stem cells (MSCs) , and monocytes. These cells secrete various biologically active molecules at the site of injury to facilitate fracture repair. The bone morphogenetic proteins (BMPs) are osteoinductive agents which promote the proliferation and differentiation of undifferentiated cells to become either osteoprogenitor or chondroprogenitor cells. Although our bodies have the inherent capability to repair the fracture, the fracture healing process can be impaired for numerous reasons.

When the fracture healing cascade stalls, a delayed union may develop, but the process may altogether cease. In a delayed union , both clinical evidence and radiographic evidence of healing do progress, but it lags behind what the normal healing time should be for a particular bone. There are however many factors to take into consideration such as the particular bone involved, the specific anatomic regions of the particular bone, the fracture pattern, as well as the method of treatment. There are certainly specific areas within the skeleton that already have a predisposition to impaired healing due to both biologic and mechanical factors such as the subtrochanteric femoral region . Additionally, the treatment method may contribute to a nonunion due to the inadequate mechanical environment provided by the choice of fixation. Often times the diagnosis is more retrospective in nature then prospective. Nonoperative interventions such as various noninvasive stimulation devices or medications can potentially augment the slow fracture healing process.

A delayed union may eventually heal or eventually may become a nonunion. Often times it is difficult to diagnose a nonunion in real time, and much of the time the diagnosis is made retrospectively. If the process stops altogether, a nonunion has developed which may require intervention. The US Food and Drug Administration (FDA) has defined a nonunion as a fracture that is at least nine months old and has not shown any signs of healing progression for at least three consecutive months [1]. From a clinical perspective, we define a nonunion as one in which the normal fracture healing process has ceased, to the extent that, without further treatment, healing will not progress. Thus, the nine-month rule should not be applied to all fractures and be based more upon the clinical presentation and the individual patient [2]. In addition to a lack of clear-cut “time” guidelines for a nonunion, there is difficulty in assessing a fracture for a nonunion based upon radiological findings and a wide disparity exists in orthopedic surgeons’ perceptions of nonunion criteria and time points for nonunions [3]. Additionally, it is well known that there are certain bones that are at a greater risk to go on to a nonunion. This may be due to the location on a certain bone due to vascularity issues or the whole bone itself, e.g., scaphoid. In certain situations, the associated bone loss that occurs clearly exceeds any critical size defect and will not heal with fixation alone, and thus, a nonunion is the expected result. It would be inappropriate to delay intervention in these patients until 9 months per the FDA definition. One can clearly see that the details of each case must be taken into consideration when deeming it a

nonunion.

There has been considerable discussion regarding the costly burden of nonunions financially, but the affects on functional outcome and the quality of life can be devastating. In a study of tibia nonunions, the authors found that these patients had high per patient costs overall with increased healthcare resource usage [4]. In a study by Kanakaris and Giannoudis [5], the increased costs were also associated with humeral and femoral nonunions in addition to tibia nonunions. Not only are there direct costs associated with the treatment, but also significant indirect costs associated with losses in productivity [6]. Earlier treatment based on earlier diagnosis could result in significant financial savings to the healthcare system and society. In addition to the additional cost, there are significant impacts to the quality of life and functional outcome of these patients. In a study evaluating patients that have tibial shaft nonunion s with functional outcome scores, Brinker et al. [7] found that the SF-12 scores (physical and mental) indicated an extremely disabling effect on physical and mental health. The impact on physical health was comparable to that of end-stage hip arthrosis and worse than congestive heart failure. In a follow-up study, Schottel et al. [8] found that all longbone nonunions had a very low health-related quality of life based upon Time Trade-off direct measures to determine utility scores. Long-bone nonunions had a utility score of 0.68 that was well below that of type-1 diabetes (0.88), stroke (0.81), and HIV (0.79). Those with forearm nonunions had the worst quality of life. Unfortunately, even with successful treatment of the nonunion, it has been shown that, at least in respect to tibial nonunions, there is a long-term negative impact on one's quality of life [9]. The indirect burden to society remains unanswered.

It has been estimated that between 5 and 10% all patients will have some difficulty in healing their fracture [6, 7]. It has also been reported that 1 out of 6 fractures that have delayed healing will go onto a nonunion [10]. Additionally, the incidence is also variable depending upon the anatomic area in question. Unfortunately, the overall incidence of delayed union and nonunion following fractures has been thought to be increasing due to various factors including an aging population, increased obesity, diabetes, smoking , vitamin D deficiency , as well as improved survival rates of patients with multiple injuries. These aforementioned factors certainly affect the biological aspect of fracture healing; however, the mechanical aspects of fracture healing can also be problematic. The mechanical factors are often dependent

upon the type of treatment method chosen by the surgeon in discussion with the patient. The mechanical stability that can be achieved at the fracture site is dependent upon the type of stabilization method used whether it be nonoperative or operative means. Cast stabilization of the fracture has the least amount of stability, but can be effective in many fractures that are amenable to nonoperative management. Methods of surgical fixation include open reduction and internal fixation, external fixation, and intramedullary nailing. This multitude of options can lead to a vast spectrum of stability. This affects the type of fracture healing that can occur, either primary or secondary fracture healing, in which callous formation occurs in the latter type. The interplay of biologic factors, including osteogenic cells and the extracellular matrix, which acts as a natural scaffold, and growth factors inherent to fracture hematoma along with the mechanical environment forms the basis of the diamond concept of fracture healing introduced by Giannoudis et al. [11]. All of these factors should be taken into consideration in the management of nonunions as well. Neglect of one of these key cornerstones of fracture healing can doom the treatment of the nonunion.

Many people have tried to elucidate factors, biological markers , or other aspects of the fracture or treatment that could contribute to a nonunion allowing one to potentially predict which fractures or which patients may progress on to a nonunion [12–30]. The establishment of a nonunion on radiographs does not necessarily imply the need for operative intervention. Nonunions maybe asymptomatic, and therefore, both clinical and radiological findings as well as the patient's current function and wishes are necessary to determine the best course of action in the management of a nonunion. Surgical intervention of the original fracture can often times make the diagnosis of a nonunion difficult especially in the absence of associated hardware failure. Thus, the evaluation, diagnosis, and the treatment of a nonunion can be very complicated [10, 31]. It requires a thorough understanding of the original injury and treatment, subsequent treatments as well as patient comorbidities, which may have contributed to the development of the nonunion.

## 1.2 History

Evaluation of a nonunion should begin, first and foremost, with an evaluation of the patient and their medical history. A thorough evaluation and review of

the patient's past medical and surgical history including medications are very important in helping to elucidate the etiology of the nonunion. It is important to take a medical history and assess for vascular disease, malnutrition, diabetes, social history, and metabolic bone disease such as osteoporosis, endocrine disorders, vitamin D deficiency , hepatic and renal disorders, steroid use, and rheumatologic disorders. Many of these comorbidities will be discussed below under "etiology." Social issues such as smoking or illicit drug use are important to note as these things may prevent healing or increase the risk of complications. A thorough and complete physical examination should be performed on all patients presenting with a nonunion. The physical examination should include a general physical which may point to other underlying disorders that may have been overlooked. Detailed examination of the extremity involved should be performed to include an evaluation of the neurovascular status, looking for open wounds (draining sinuses), healed lacerations (indicative of perhaps an open injury), healed incisions, clinical alignment, joint motion, and examination of the presumed nonunion site for motion. Any open wound or draining sinus in proximity to the fracture should lead one to suspect a septic nonunion and is so until proven otherwise. Such open wounds must be taken into consideration, and a soft tissue reconstruction plan will need to be integral to the overall bony reconstruction. Previous incisions may limit options and may dictate how previous hardware is removed. Alternative approaches may need to be employed if the existing soft tissues are scarred in or suboptimal for further surgical intervention. If there is a deformity, correction of the malalignment has to be taken into consideration as well. This includes any leg length discrepancy that may need to be addressed. Joint motion may be limited from arthrofibrosis or a result from a false joint at the nonunion site, or patients may have developed contractures. Any surgical plan must take into consideration the need for lysis of adhesions, soft tissue releases, etc., to insure the best possible overall outcome. In short, preoperative planning taking all these factors into consideration before going down the reconstructive pathway is paramount.

It is extremely important to obtain an accurate history of the original injury mechanism as well as other fracture characteristics. It is important to determine whether or not the fracture was from a high-energy or low-energy injury. The extent of the initial soft tissue injury as well as the amount of periosteal stripping that may have been encountered at the time of surgery or because of the surgery may shed light on the potential cause of the

development of the nonunion. It has been recently suggested that compartment syndrome and associated fasciotomy may be a risk factor for the development of nonunion in tibia fractures [12]. Open fractures obviously have much more soft tissue damage, and the potential for an occult infection and septic nonunion must also be taken into consideration.

A careful evaluation of all previous surgeries is critical, especially the index operation. Review of the operative reports and/or injury radiographs along with the immediate postoperative films can be crucial to understanding the underlying cause. Subsequent interventions should also be evaluated in a similar manner, taking into consideration the pre- and post-op radiographs and the details of the surgical procedure. If bone grafting or biologic adjuncts had been done or used at any time, the type of bone graft or adjunct, the location of harvest of the autogenous bone graft, should be noted. Previous sites of harvest may limit future options. Inadequate fixation or extensive surgical exposures can be large determinants in the development of a nonunion. In fractures treated with intramedullary nails, external fixation , cast stabilization , or bridge plating, a relatively stable construct has been created allowing for callous formation. In cases of open reduction and internal fixation (ORIF) , an environment with absolute stability often is created allowing for primary bone healing without callous formation. The surgical assault obviously affects the amount of soft tissue stripping which can affect the amount of blood supply to the fracture site. Additionally, past surgical interventions and hardware that is present can certainly affect future treatment options for the management of the nonunion.

A thorough evaluation of prior complications should be performed. Any history of infection should increase one’s suspicions for continued infection even in the absence of clinical signs or symptoms. Nerve injuries should be assessed as this may limit the overall outcome of any nonunion reconstruction and may lean one toward a more definitive intervention such as amputation. Previous vascular injuries may require further assessment in terms of viability of the previous repair and a thorough assessment of the vascular status of the limb.

## 1.3 Risk Factors for Nonunion

Biological factors and mechanical factors can contribute to the development of a nonunion. These can be related to the patient or the intervention

performed by the surgeon. If the patient has been referred in, as mentioned previously, it is helpful to obtain previous injury radiographs, computed tomography (CT) scans, and other imaging studies as well as operative reports to understand what was done and why it was done. If you are the index surgeon, it is important to critically asses your own surgical intervention to determine whether things that were done may have contributed to the nonunion. Decision errors can always occur, and what is successful in one patient may not be so in another patient. In any event, risk factors for the development of a nonunion can be classified as patient dependent or independent [10, 25]. Many of the independent factors are more surgeon-dependent factors or injury characteristics.

The injury characteristics unique to a specific fracture location will be discussed in each specific anatomic section, but some generalities can be made. Areas that are known to have tenuous blood supplies have been shown to be at risk of nonunion [10, 28, 32]. Such areas include the femoral neck, subtrochanteric region of the femur, the scaphoid, the talus, the metadiaphyseal region of the fifth metatarsal, and tarsal navicular body. Open fractures with their significant soft tissue stripping clearly have increased risks of nonunion as well as infection [23, 25, 26, 28, 29]. The associated soft tissue injury and muscle loss in severe open injuries can result in loss of the blood supply to the bone resulting in a detrimental effect on the healing process and increasing the risk of infection. Lin showed that functional outcomes in patients with open tibia fractures were worse than those with closed fractures [33]. Westgeest et al. [29] found that fractures which were classified as open grade IIIA injuries were associated with delayed healing and nonunion. Additionally, in this prospective cohort of 736 subjects, all with open long bone fractures, deep infection was associated with delayed healing and nonunions. In a retrospective study of long-bone fractures treated with intramedullary nailing, Malik et al. [23] found that open fractures had a significant association with the development of deep infection which also was associated with the development of a nonunion. In the same study, they alluded that opening of a closed fracture also was a significant contributor to the development of a nonunion, and therefore, opening of the fracture, in cases of intramedullary nailing, be avoided if possible. In the study by Blair et al. [12], fasciotomy for compartment syndrome in tibia fractures, which in essence is opening of the fracture, was also associated with significant increase in both infection and nonunion. In an effort to prevent infection in

open fractures, it is well established that antibiotics be administered as rapidly as possible and hopefully within an hour of the fracture presenting [34]. Often times the open fractures are also associated with significant bone loss and in most cases such defects cannot heal on their own and are expected to become nonunions if left alone. These eventually will require bony reconstruction. The type of reconstruction, timing of bone graft placement, and the source of bone graft is highly variable among orthopedic trauma surgeons [35]. Determining the amount of bone graft for such defects can be problematic, and some have tried to develop quantitative models to determine the amount needed [36]. Other fracture characteristics that need to be assessed include the degree of displacement, the extent of comminution, the amount of cortical apposition at final fixation, and the stability of fixation [24, 25, 28, 32, 37, 38].

Surgeon factors can contribute to either biological reasons for the development of a nonunion or a mechanical one [23, 25, 28, 32]. Contributions to a biological cause include excessive stripping of soft tissues, failure to bone graft at the appropriate time, and inadequate debridement of devitalized/dead bone, which can lead to infection, which then may prevent union. Mechanical factors introduced by the surgeon are related to the method of treatment and/or implant for the original fracture. Fracture stabilization has significant affects on fracture healing. In a literature review by Hildebrand et al. [37], the type and timing of fracture stabilization can alter the systemic inflammatory response after trauma and can affect fracture healing. They also found that the type and stability of the fracture stabilization affects gene expression involved in fracture healing. Relative stability constructs such as intramedullary nailing, cast immobilization, and external fixation allow the fracture to heal by callus formation; however, excessive motion could lead to a hypertrophic nonunion. The rigidity of the fracture fixation has been shown to improve the process of healing [37]. Reaming of the canal in intramedullary nailing can increase the size of the nail and enhance the mechanical stability. The effect of reaming has been looked at extensively [39]. It has been well established that reaming enhances fracture healing and that there is a higher incidence of delayed union and nonunions in unreamed nails with more secondary procedures to obtain union [23]. This is true despite a recent study showing that the functional outcomes in tibia fractures were not affected by reaming [33]. Inadequate internal fixation when one is trying to achieve absolute stability to create an

environment for primary bone healing can also lead to excessive motion and a subsequent nonunion. Niikura et al. [25] reviewed 102 nonunions of which almost 80% were related to or solely caused by inadequate stability or reduction. Conversely, rigidly fixing fracture fragments with gaps or without proper internal fixation techniques such as obtaining compression across fracture planes may delay or even prevent healing [31]. Fixation can be too rigid leading to a failure in healing. If the patient had undergone what was felt to be appropriate fixation with appropriate surgical technique for the fracture in question, then it is important to investigate patient-related factors, both biological and mechanical, that may have contributed to the development of the nonunion. Brinker et al. [13] created an algorithm on when to refer patients for endocrine workups in relation to their nonunion. When evaluating the nonunion, the technical aspects of the fracture fixation should be assessed. If there was no technical error, then it was suggested that perhaps there was a metabolic etiology to the nonunion, and thus, the patient should be referred to an endocrinologist. If technical error was a crucial factor in the etiology, referral was not indicated. However, it is important to still assess metabolic issues even in light of inadequate fixation as many patients still have some deficiencies in bone metabolism [13].

Patient factors contributing to mechanical problems can be related to noncompliance with weight-bearing restrictions or an error in allowing the patient to weight bear too early. The healing process is always a race between hardware failure and fracture healing, and thus, when patients present with a nonunion in conjunction with hardware failure, the time from the original surgery is important in determining what came first—the hardware failure or nonunion, as each one can lead to the other. Often times, with plate failure there is an associated deformity through the nonunion site (Fig. 1.1). In cases of early hardware failure, often times the patient has started weight bearing too early or was allowed to do so. This is more common in cases of plate fixation. In these situations, the fracture has not healed sufficiently to handle the body weight and the implant is taking all the stress leading to early failure. Failure can be in the form of screw loosening, implant breakage, or bending. Depending on the fracture pattern and amount of comminution as well as the location, it may still unite. In the lower extremity more so than the upper, the alignment may gradually worsen as stability is lost and a mal-aligned nonunion can develop. In some instances, especially where there is comminution, as the angulation worsens resulting in more bony contact, the

fracture may unite resulting in a malunion. In late cases of hardware failure, the fracture may have healed sufficiently to handle some weight in addition to the implant and may have maintained the alignment. After a while, the implant undergoes fatigue failure as the micromotion from the loading leads to failure of the implant at a stress riser such as a hole in the plate. The alignment is often times maintained, but the patient has pain and discomfort which necessitates surgical intervention. Loss of fixation can also occur without weight-bearing issues. This is often the case in patients with poor bone quality such as in those with comorbidities such as diabetes or osteoporosis. It is important to know whether patients have these conditions as special surgical and fixation techniques may need to be employed to obtain improved fixation by the judicious use of locked, fixed angle, or load-sharing devices such as intramedullary nails when appropriate.


(a)
(b)
(c)
UPRIGHT
R
(d)
(e)
(f)
(g)
(h)
(i)
(j)
(k)
(l)
(m)

***Fig. 1.1*** Patient with right ankle injury treated with open reduction and internal fixation of fibula and closed treatment of distal tibia fracture. Referred for nonunion after progressive deformity developed. **a**–**c** Three views (anteroposterior [AP], mortise, lateral) of the *right ankle* show failure of the fibula hardware and mal-alignment with nonunion of both the tibia and fibula. Patient underwent hardware removal and cultures. **d**–**f** Three views (AP, lateral, and mortise) of the *right ankle* after hardware removal. Due to the malalignment and stiff nonunion, a Taylor spatial frame (TSF) was applied to allow correction and healing of the nonunion. **g**–**h** AP and lateral after TSF applied to *right ankle* prior to correction. **i**–**j** AP and lateral with TSF showing full correction of the deformity and realignment of the limb. **k**–**m** Three views (AP, lateral, and mortise) of the *right ankle* 1 year after consolidation of nonunion and removal of TSF

Patient medical factors contributing to a biological cause for the nonunion are many and can be problematic not only from the original fracture standpoint but also for the treatment of an established nonunion [10, 13, 25, 32, 40]. Established diseases such as vascular disease, rheumatologic disease, and s/p organ transplantation cannot be affected, but their effects on fracture healing and subsequent management of the nonunion need to be taken into consideration. Perhaps their steroids or immunosuppressive agents can be held for short time period which would allow for surgical intervention and healing, and such decisions should be made in conjunction with the patients' appropriate other physicians. A multidisciplinary approach is needed to get many of these patients healed.

Although there are many endocrine abnormalities that can affect the musculoskeletal system, such as thyroid and parathyroid disorders, hypogonadism, and calcium imbalances to name a few [13], diabetes has had the most attention due to the high prevalence in the population. Diabetes has been shown to prolong healing times for fractures [40, 41]. It is also well documented that patients with diabetes have increased complications when dealing with musculoskeletal conditions, especially with fractures [32, 42, 43]. In a nationwide population based study out of Taiwan, diabetics were found to have an increased incidence of fractures as well as more adverse events and a higher mortality after fractures [42]. The addition of neuropathic complications can make even simple fractures that require surgery end up being disastrous for the patient. Wukich et al. [43] showed that patients with ankle fractures that had complicated diabetes had a 3.8 times increased risk of overall complications and a 3.4 times increased risk of malunion and nonunion compared to uncomplicated diabetic patients. These patients were also 5 times more likely to require revision surgery or arthrodesis. Diabetics need to understand that glucose control is extremely important for them to

avoid diabetic complications of end organ damage, neuropathy, nephropathy, and peripheral arterial disease to minimize further musculoskeletal complications [32]. Diabetics should be treated with prolonged immobilization and delayed weight bearing compared to the nondiabetic to aid in avoiding complications. Additionally, many of these patients require additional fixation for otherwise straightforward fractures to try and prevent the late complications that occur with these injuries.

Vitamin D deficiency or insufficiency has been linked to nonunions, but a clear causal link is difficult to establish [13, 40]. Both the 25-OH vitamin D and 1, 25 $OH_2$ vitamin D levels can be monitored, but the 25-OH level is the one that is important. Patients with 25-OH levels <20 are considered insufficient and between 20 and 30 deficient. It is not clear however whether higher levels than simply above the 30 level are needed in patients with fractures. Brinker et al. [13] showed that a preponderance of their nonunion patients had vitamin D deficiency. They had 37 patients that were evaluated for a metabolic or endocrine abnormality of which 68% (25 of 37) had vitamin D deficiency . It has become increasingly clear that many patients are vitamin D deficient or insufficient. In a meta-analysis of the literature, it was found that the pooled prevalence of hypovitaminosis was 77.5% in young trauma patients and 73% in geriatric fragility fracture patients [44]. In a follow-up study, the same authors showed that there is a lack of consensus in prescribing vitamin D to fracture patients. They found that 66% of surgeons tended to prescribe vitamin D to fragility fracture patients compared to 25.7% to nonfragility fracture patients [45]. The lack of prescribing in this population needs to be re-examined since the prevalence of low vitamin D in young trauma patients is high. Low vitamin D is more prevalent than previously thought and is widespread in patients of all orthopedic subspecialties and not just orthopedic trauma [46]. Management of vitamin D is easily done via replacement therapy and has been shown to be successful in raising serum levels [44]. In a study to evaluate the cost benefit of both calcium and vitamin D supplementation in all fracture patients, the cost of an 8-week course of treatment was determined and compared to the cost savings assuming just a 5% reduction in nonunions. This would result in a potential cost savings of $65,866 annually [47]. Many dosages of replacement therapy are available, but the authors' preference is for high-dose (50,000 IU) vitamin D weekly for six months along with calcium supplementation. The target is to obtain a 25-OH level in the 40–60 range. Patients with low vitamin D can

also develop secondary hyperparathyroidism and should also have a parathyroid hormone (PTH) level drawn when evaluating for a nonunion. The high PTH can contribute to the development of a nonunion [13]. In most cases, the high PTH will resolve with appropriate vitamin D replacement therapy.

Osteoporosis has also been linked to the development of nonunions [32]. The issues with osteoporotic bone healing are both biologic and mechanical [48]. By definition, osteoporotic bone is bone with less bone mass and as such is at an increased risk for fracture. The biologic changes that occur with osteoporosis, including a diminished level of mesenchymal stem cells and thus osteoblasts, a decrease in the chondrogenic potential of the periosteum and other alterations in the fracture healing pathway results in a less than robust fracture healing process [32, 40, 48]. Additionally, because of the lower bone mass, the fixation in such bone can be problematic and as such can lead to inadequate fixation and fracture stability. The result can be a nonunion. Many specialized techniques have been described in the management of osteoporotic fractures and should be employed when dealing with nonunions especially if mechanical failure was a significant contributor to the development of the nonunion. Locked plating, use of load-sharing devices, use of fixed angle devices, augmentation of fixation with cement or bone graft substitutes, adjunctive use of structural bone grafts, and preservation of soft tissue can assist in the management of these fractures and nonunions [48]. Although most osteoporotic individuals are elderly, age is an independent factor which can negatively affect fracture healing also resulting in delayed unions or nonunions [49]. This decline in healing potential can be attributed to hormonal changes, changes at the cellular level of fracture healing signaling, and diminished mesenchymal stem cells which all may also occur with osteoporosis. The true etiology still requires much more investigation due to the complex interplay that occurs in fracture healing and the overlap in physiology with aging and osteoporosis. Another confounding factor is that patients with osteoporosis are often being pharmacologically treated for their osteoporosis. The most common are the bisphosphonates , which are anti-resorptive agents and inhibit osteoclast function. The interference with remodeling of the bone has resulted in an unwanted side effect and resultant “atypical” femoral fractures. It is advised that these medications be discontinued during the fracture healing process [32, 48]. It is also important to evaluate all their medications and the potential effects that

they may have on bone metabolism.

One of the most common class of medications that many patients take, both prescription and over the counter, are the nonsteroidal anti-inflammatory drugs (NSAIDs) . Recently, the use of NSAIDs during fracture healing has come under intense scrutiny [49–51]. Early reports of the use of NSAIDs in animal fracture healing models showed a clear deleterious effect [32, 49–51]. The doses required were very high. The mechanisms by which they are theorized to inhibit fracture healing include inhibition of prostaglandin synthesis and reduction of osteoblast activity, both of which result in an impaired fracture healing response [32]. Prostaglandins are needed during the inflammatory phase of fracture healing and help start the osteogenic response [6, 31, 38, 49]. Although a few clinical studies have shown a loose association between the use of NSAIDs and nonunions, it is controversial [50]. Kurmis et al. [51] performed a systematic analysis of over 300 relevant papers and concluded that there was not significant evidence to indicate a negative effect on fracture healing from the short-term use of NSAIDs after a fracture. Most of the clinical studies published in relation to NSAIDs in fracture healing were Level 5 evidence or expert opinion only [28]. Therefore, it is hard to make a recommendation on the use of NSAIDs both in terms of timing and duration immediately after a fracture. Due to the lack of guidelines and unknown true effects on fracture healing, the author's practice is to avoid NSAIDS for the first 4–6 weeks after a fracture. This is especially true of Indomethacin. Additionally, we do not use Toradol (intravenous or *per os*) intra-operatively or immediately postoperatively for acute fracture cases. NSAID use after repair of nonunions has not been investigated to our knowledge.

Since inflammation is one of the initiating factors for bone healing, it has been suggested that perhaps healing may be altered in the polytrauma patient as well [32, 37, 40, 50]. These patients undergo a prolonged state of inflammation [40]. It is thought that the increased inflammation could delay fracture healing through a variety of cellular responses [20, 50]. Additionally, many of these patients also have multiple fractures that may require operative intervention. The post-op rehab protocol on one fracture may result in delayed stimulation of another fracture with resultant delayed healing or even nonunion. Other system injuries also may have an effect on fracture healing as well. A literature review by Hildebrand et al. [37] found that isolated hemorrhagic shock, chest trauma, severe soft tissue injury, and systemic

inflammation can all affect fracture healing. Finally, it has been suggested that the American Society of Anesthesiologist (ASA) classification, which indicates overall health, was associated with nonunion development—the higher the ASA, the increase in probability of a nonunion [23].

Smoking has been clearly shown to inhibit fracture healing and result in both delayed unions and nonunions as well as increase the overall complications in the management of fractures [10, 21, 24, 28, 29, 32, 46, 49, 52–54]. Smoking has also been linked to an increase in fracture rates of the hip, distal radius, spine, and other osteoporotic fractures [52]. The exact mechanism and offending agent has not been clearly elucidated. Nicotine is one of more than 4000 chemicals that exist in cigarette smoke [46]. It has been shown to cause vasoconstriction (resultant hypoxia), platelet adhesion, and reduced cell proliferation for healing. All of these physiologic changes result in a negative effect on both wound and fracture healing [52]. However, it is not clear exactly which chemical is responsible for all the negative effects. Animal studies have had conflicting results with some studies, with nicotine alone, not showing the negative effects that are seen with smoking, whereas others have shown deleterious effects [49]. Nevertheless, the clinical literature overwhelmingly supports the increased risk of delayed unions, nonunions, and wound complications seen with smoking [21, 24, 52–54]. Scolaro et al. [54], in a systematic review of the literature, showed that smokers were over 2 times more likely (statistically significant) to develop a nonunion than nonsmokers. This was especially true in open fractures and tibia fractures. There was also a trend for longer healing times and infections (deep and superficial) in the smoking group. In a separate systematic review of the literature, Patel et al. [53] also found a negative effect of smoking on bone healing. They also looked at each study in relation to the bone or procedure in question. All the tibia fracture studies, except for one treated with external fixation, showed a clear increased risk of nonunion from smoking. This was also true in distraction osteogenesis, fibula fractures, ulna osteotomy healing, subtalar and ankle arthrodesis, and elective foot surgery. Fractures of the femoral diaphysis were not statistically significantly affected by smoking. In contrast, Hernigou and Schuind [21], in their retrospective study looking at diaphyseal fractures, found that smoking was significantly associated with nonunions (OR 8.25) in the femur, as well as the tibia and humerus. Westgeest et al. [29] found that in a prospective cohort study of open long-bone fractures, smoking (OR 1.73) was significantly associated

with developing a nonunion. Murray et al. [24] looked at their series of diaphyseal clavicular fractures. They found that smoking was the strongest predictor of a nonunion (OR 3.76) and recommended that smoking cessation be an integral part of any treatment. However, getting patients to stop smoking is extremely difficult. The first step is acknowledging that smoking is bad for one's health. Matuszewski et al. [55] performed a cross-sectional cohort survey study and found that smokers did not understand the negative effects of smoking on their general health or on fracture care. On a positive note, the orthopedic trauma patients surveyed seemed interested in smoking cessation more so than what was expected. They recommended formal education for smoking cessation. It is well accepted and has been shown that preoperative smoking cessation can reduce both pulmonary and wound complications postoperatively [46]. Educating the patients on the ill effects of smoking on fracture healing is part of our "discussion" with the patient being evaluated for nonunions. It is the author's policy to not perform nonunion surgery on active smokers as long as the management can be done on an elective basis (aseptic nonunions). Both serum and urinary levels of cotine and nicotine are monitored to insure patient compliance. Although many feel that smoking cessation is the primary care physician's responsibility, as an orthopedist it behooves us to play an active role to help maximize the patient's outcome and minimize complications from any surgical intervention.

When evaluating a patient for a nonunion management, one must assess for the presence of the risk factors above. There are certainly more comorbidities than can affect fracture healing, but these are the most prevalent. These risk factors and/or co-morbidities should be improved upon or corrected if feasible. Many are injury or treatment related, but knowing those details can help devise an appropriate treatment plan for the nonunion.

---

## 1.4 General Principles

### 1.4.1 Diagnosis

The diagnosis of a nonunion is highly controversial because no gold standard exists for healing assessment [6, 15]. In a multinational survey of orthopedic surgeons, there was a 73 and 53% consensus that a lack of standardization in the definition for a delayed union and nonunion, respectively, existed.

However, they did agree (88%) that the diagnosis should be done based on clinical evaluation and plain radiographs [3]. Pain on weight bearing was felt to be the most consistent predictor of delayed union and nonunion.

The diagnosis of a nonunion should be made on a series of radiographs in addition to the clinical picture. Often the fracture healing may be delayed, but critical evaluation of radiographs 6–8 weeks apart may show some improvement indicating progress. If the X-rays show no progress on two sets of consecutive images and the patient is having pain, then nonunion has probably been established assuming sufficient time has initially passed. The problem arises in the patient without symptoms but clear radiographic evidence of a nonunion. Many of these patients, because they lack symptoms, may not return in fear of needing surgery. The problem occurs when they return after hardware failure with new-onset pain and/or deformity. The time passed based on the FDA definition may not have been reached, but if cessation of all healing is indicated by plain radiographs and the patient is symptomatic, then intervention is probably warranted.

### 1.4.2 Radiographic Evaluation and Scoring

After the history and physical, evaluation should always begin with plain radiographs. It still remains the most common method of assessing for fracture union. However, just as in the lack of standardization of definitions, there is a lack of consensus on radiographic criteria as well. Dijkman et al. [56] reviewed the literature to look at radiographic criteria used in studies. They found that bridging of the fracture by bone, callus, or trabeculae was used 53% of the time. Bridging of the fracture across three cortices 27% of the time and loss of fracture lines was 18% of the time. The best interobserver reliability was found to be the number of cortices bridged by callus.

Despite the issues with radiological criteria , standard orthogonal views (*anteroposterior* and lateral) of the bone in question should be obtained. If the patient is referred in, previous studies are desired for comparison. In some cases, the fracture is actually progressing and reassurance is all that is needed. They may have a delayed union, but radiographic evidence of healing is occurring. The length, alignment, and rotation of the limb should be appropriately evaluated. In the lower extremity, if there is an associated deformity, then additional full-length radiographs (± ruler) from the hip to the knee are obtained to assess the mechanical axis of the limb (Fig. 1.2).

Restoring the mechanical axis of the limb can aid in healing of the nonunion and should be part of the preoperative plan. If it appears to be short, then a scannogram (Fig. 1.3) or full-length radiographs with a ruler should be obtained (see Fig. 1.2 ). Oblique radiographs can aid in the diagnosis as well, if the standard *anteroposterior* and lateral do not clearly show the nonunion due to the obliquity of the original fracture or because of overlying hardware (internal or external fixation). Such views can also better define the plane of maximum deformity when that plane is not in the usual sagittal or coronal plane. Rotation can be assessed clinically in some situations; otherwise, a CT scan may be needed (Fig. 1.4).

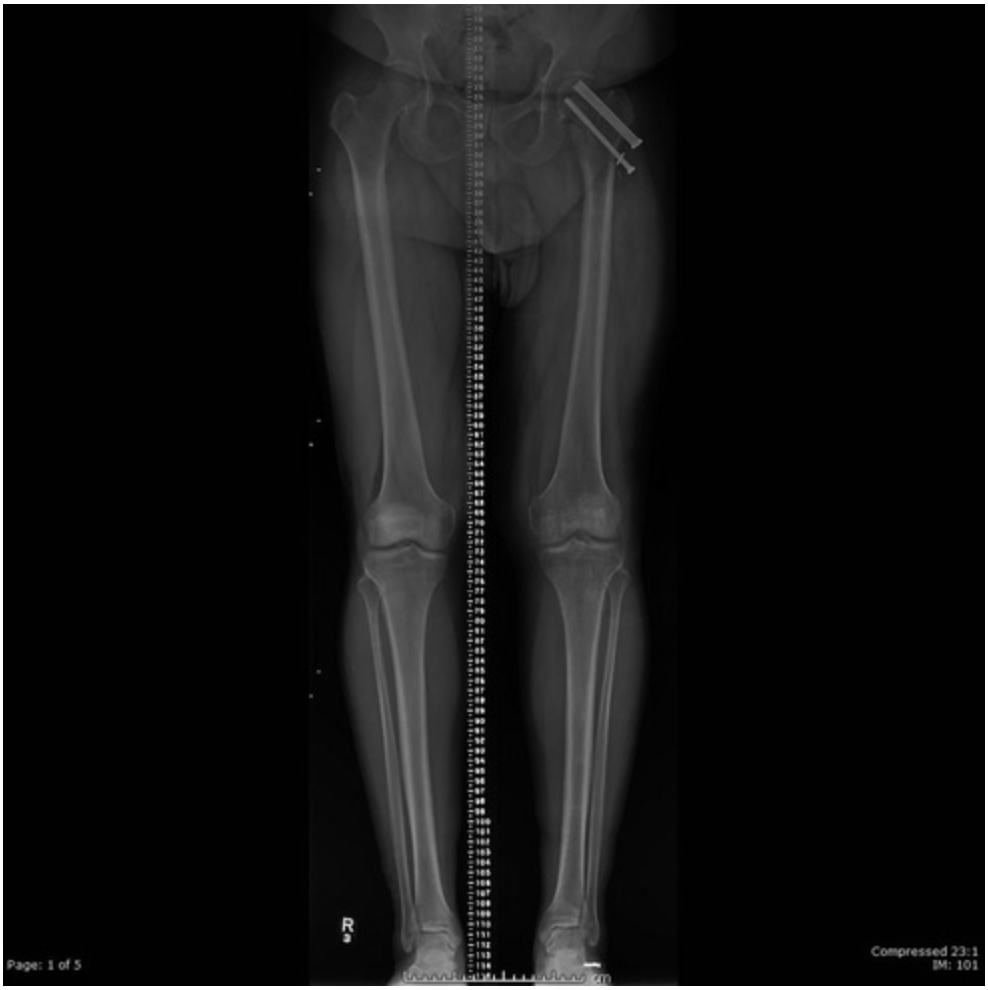



***Fig. 1.2*** Patient with a long leg film obtained for evaluation of his mechanical axis with a *left femoral neck* nonunion . A ruler can be used also to evaluate for leg length discrepancy

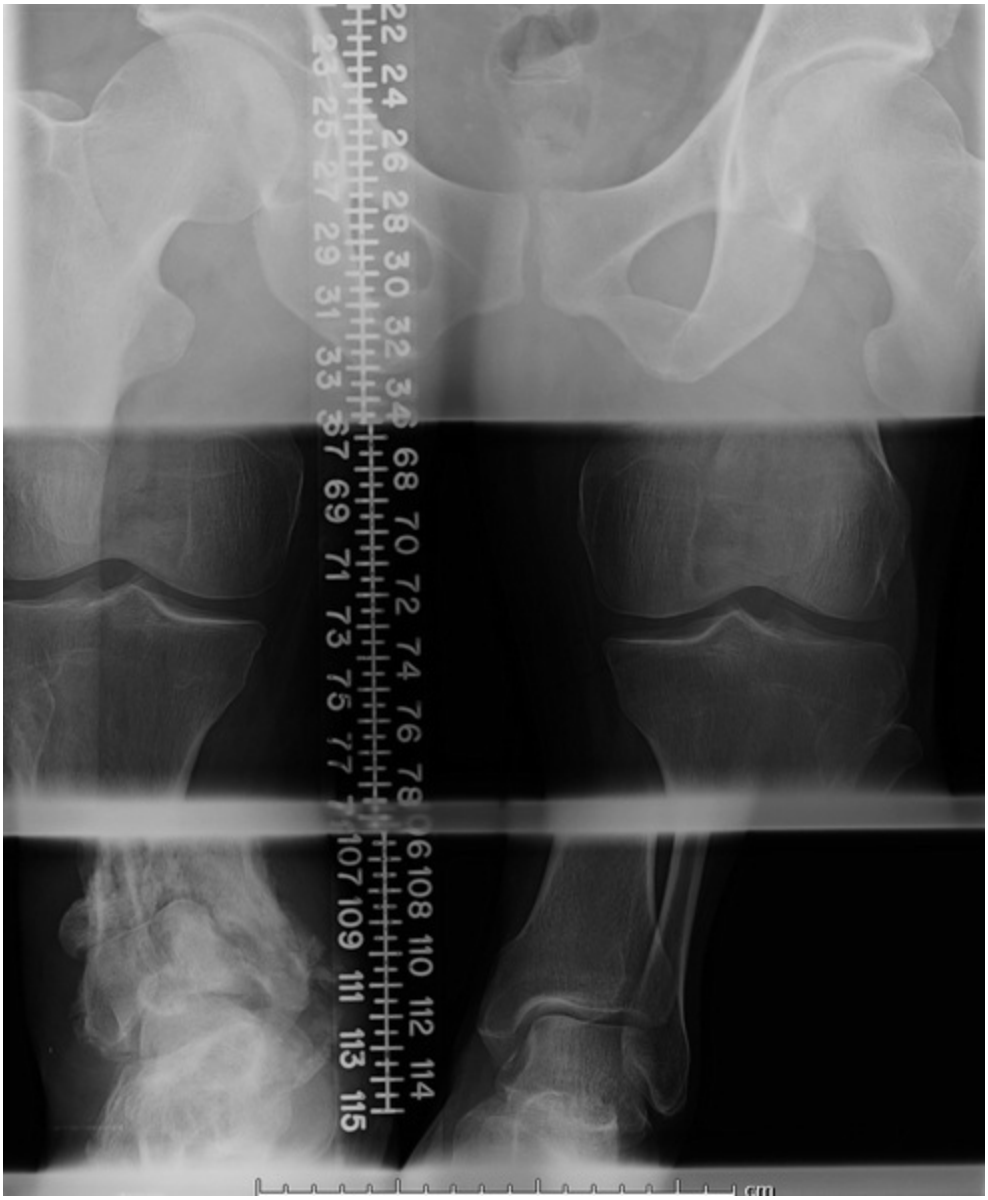


***Fig. 1.3*** Patient with a *right ankle* injury and delayed presentation. Scannogram was obtained to evaluate the amount of leg length discrepancy

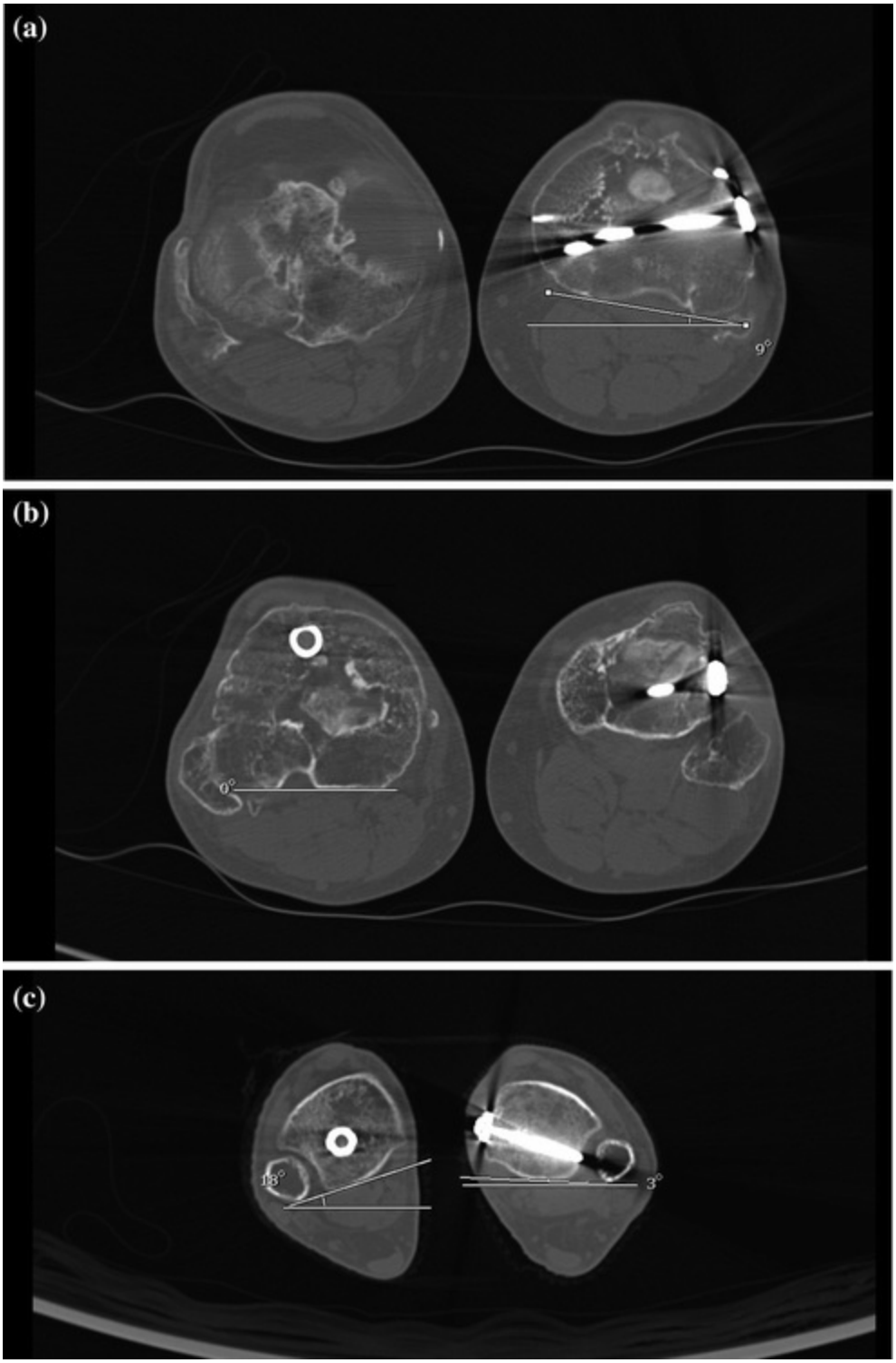



***Fig. 1.4*** ( **a**–**c**. Computed tomography scan images showing how to measure malrotation of left tibia—24° internal rotation compared to *right side*

When looking at the plain radiographs, the absence of bridging bone or

callous at the fracture site, sclerotic fracture edges, bone resorption, or persistent fracture lines all may indicate a nonunion. It is imperative to also critically assess the implants and the initial fixation strategy to insure that the original type of healing wanted—primary versus secondary—was being achieved. Often times absolute stability was desired, yet there is callus formation on the radiographs (Fig. 1.5). This may indicate either excessive motion suggesting hardware failure or that the fixation was not as rigid as one wanted, allowing sufficient motion for callous formation. The fracture however may go on to heal. In other situations, it may go on to a nonunion with or without hardware loosening or breakage. Additionally, the radiographs should be assessed for periosteal reaction, loosening/lysis around hardware, and broken implants. Comparison to previous radiographs cannot be overemphasized.

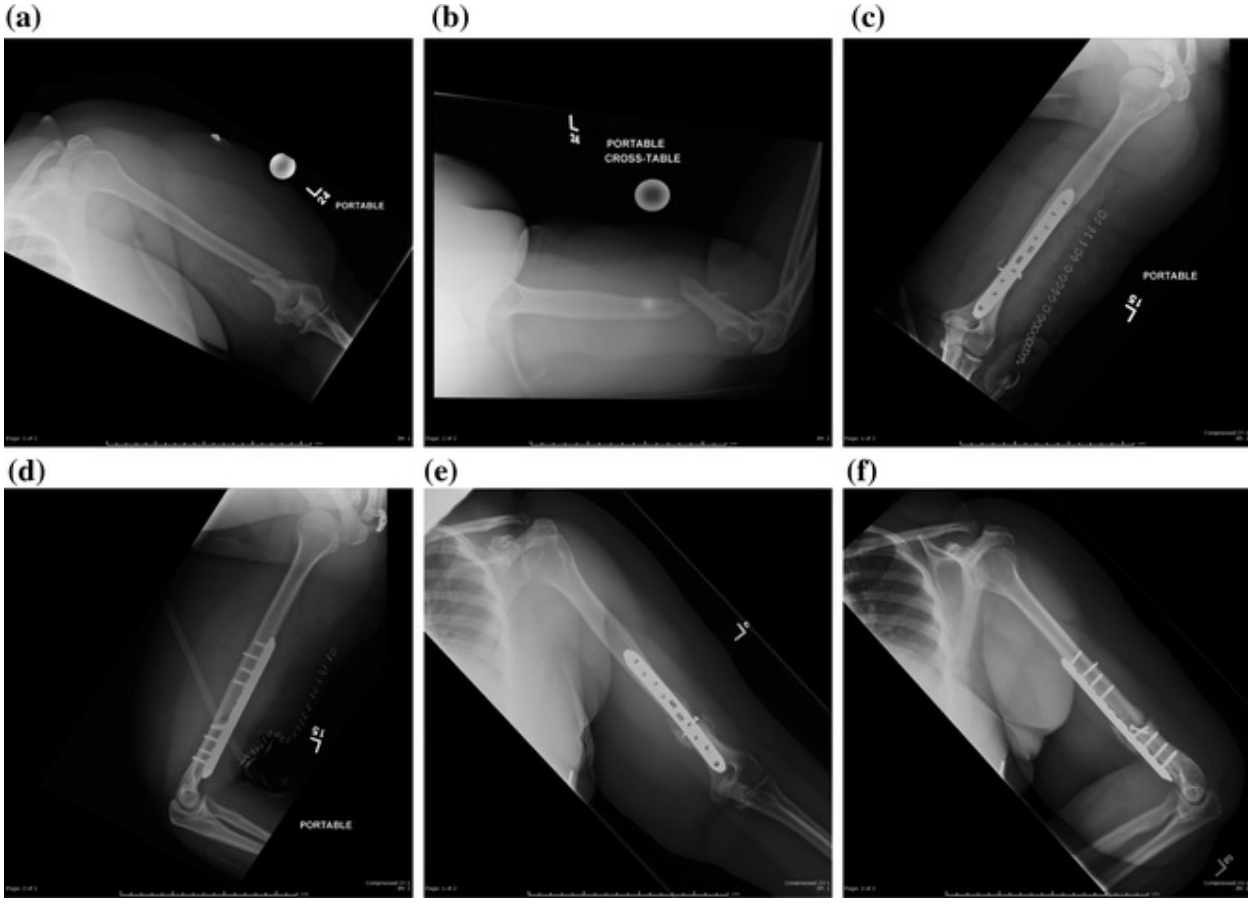



***Fig. 1.5*** **a, b**. Injury radiographs (anteroposterior [AP] and lateral) of patient with *left* humerus fracture after a motor vehicle collision. **c, d** Postoperative radiographs (AP and lateral) after open reduction and internal fixation performed in an effort to obtain absolute stability. **e, f** Follow-up radiographs (AP and lateral) showing unintended callus formation due to micromotion despite attempt

at rigid fixation—infection workup was negative and patient went on to consolidate

As mentioned above, plain radiography alone is often times not a reliable tool for assessing fracture healing due to the lack of consistency among surgeons and interpretation of the films. It is clear that better ways of assessing fracture healing are needed [6]. Several clinical trials all have shown poor agreement between surgeons [15, 56]. Many have proposed criteria to standardize fracture healing assessment [57, 58]. One such assessment tool is the Radiographic Union Scale for Tibial (RUST) developed by Koolstra and his colleagues [58]. This scoring system assesses the presence or absence of fracture callus and the visibility of the fracture line on each of the four cortices. The scale is from 1 to 3 and based on callus and fracture line visibility at each cortex. A one is the absence of callus and a visible fracture line. A two is the presence of callus, but the fracture line is still visible. A three is for callus and the absence of a fracture line. The minimum score is 4, and the maximum is 12. This has shown to improve agreement for assessing union only in tibia fractures treated with intramedullary nails. Whelan et al. [59] showed an overall inter-observer reliability of 86% and intra-observer reliability of 88%. The RUST score has not been correlated with functional outcomes to date.

A similar scoring system was developed by Bhandari et al. [57] for use in hip fractures. The Radiographic Union Score in Hip Fractures or RUSH was developed to improve agreement in the assessment of femoral neck fractures. In a similar manner to the RUST, the RUSH evaluates cortical bridging on each of the four cortices as well as disappearance of the fracture line and an independent score is given. A one is given for no cortical bridging, two for some cortical bridging, and a three for complete cortical bridging. If the fracture line is visible, a one is given, a two for some evidence of the fracture line, and a three for no evidence of the fracture line. Two other aspects of femoral neck fractures are scored, the trabecular index based on consolidation and the disappearance of the fracture line. A score of 1–3 is assigned as well to each component. The overall minimum is 10 and maximum is 30. Their initial study showed that the RUSH improved agreement among reviewers regardless of subspecialty, but their agreement did not improve over time. A very important shortcoming was that the reviewer's assessment was found to be potentially inaccurate without information regarding the time of the radiograph. They had 6 of 7 patients deemed as being healed at 2 weeks,

which is not possible. Chiavaras et al. [60] extended the RUSH score to evaluate intertrochanteric hip fractures and evaluate agreement between radiologists and orthopedic surgeons. They found that the RUSH score did improve the overall agreement regarding fracture healing from fair to substantial between the two specialties.

Although scoring systems can be beneficial in determining union and providing a more objective measurement over time, the real issue is their use in predicting a nonunion. Recently, Frank et al. [19] did a study to assess the utility of the RUSH score to help define femoral neck fracture nonunion . They retrospectively pulled 250 cases from the FAITH hip fracture trial all of which had 6-month hip radiographs. They determined the RUSH score at 6 months for each case. They found that if the RUSH score at 6 months was <18, it had 100% specificity and a positive predictive value of 100% for a nonunion. They all had a 10 times greater risk of undergoing reoperation for a nonunion. If the patient does develop a nonunion of the femoral neck, a valgus intertrochanteric osteotomy is an option to obtain union. Varghese et al. [61] evaluated a group of 40 patients who underwent the procedure for a femoral neck nonunion developing after neglected fractures. They evaluated the presenting nonunion film for a radiographic index they called the neck resorption ratio (NRR) to determine whether that could predict nonunion of the valgus intertrochanteric osteotomy. The NRR is determined by measuring the length of the fractured head and neck fragment and comparing it to the length of the intact neck on the contralateral side (measured from the tip of the head to the intertrochanteric line). The NRR was found to be the most important factor in predicting union in their series. All patients that had a pre-op NRR of >0.52 had union. Taking this parameter into consideration before making treatment decisions in femoral neck nonunions may allow one to consider a more definitive treatment and avoid a repeat nonunion situation.

Although utilizing a score to predict nonunion after a reconstructive procedure can be useful, a score to predict nonunions for acute fractures would have greater applicability. The Nonunion Risk Determination (NURD) Score was developed by O'Halloran et al. [26]. The authors retrospectively reviewed all tibial shaft fractures at their institution over a 7-year period treated with an intramedullary nail. They had 382 patients with 56 nonunions. Factors were evaluated and they developed a logistic regression model to include seven of these factors. They assigned points to these seven factors. The NURD score gave 1 point for male gender, 2 points for open fractures, 3

points for chronic conditions, 4 points for compartment syndrome, and 5 points for flaps. Additionally, 1 point per ASA grade was given as well as for each 25% reduction of cortical contact (100% = 0; 75% = 1; 50% = 2; 25% = 3). If the injury was low energy or spiral, one point was subtracted for each factor. They found that a NURD score of 0–5 had a 2% chance of nonunion versus a 61% chance if the score was >12. The score was felt to be a potential nonunion prediction model that clinicians could utilize to determine which patients had a higher risk of nonunion. If such scores could be developed and validated for other bones, prediction of nonunions could be commonplace and allow for earlier intervention.

In our practice, comparative plain radiographs over time, and the clinical picture and evaluation of the patient are sufficient to diagnose a nonunion. However, in some situations, plain radiographs may not allow complete evaluation of the nonunion site because of the hardware. In these cases, a CT scan, with metal suppression if hardware is present, can be obtained to further evaluate the nonunion site as well as look for areas of sequestered dead bone or areas of bone deficits that may require bone grafting. CT scans have been shown to have high sensitivity but moderate specificity with about a 90% accuracy for the detection of nonunions [62]. Sagittal and coronal reconstructions to include 3D reconstructions can help with visualization (Fig. 1.6). Many times, these fractures are “clinically” healed, but patients have symptomatic hardware. A CT scan can also aid in looking at the integrity of the bony consolidation and for defects within the “healed” construct. In some situations, the patient can be considered as having an implant dependent union; e.g., there is some central bone loss but sufficient bridging that the bone has healed around these deficiencies, but the strength of the bone may be reliant upon the associated hardware.

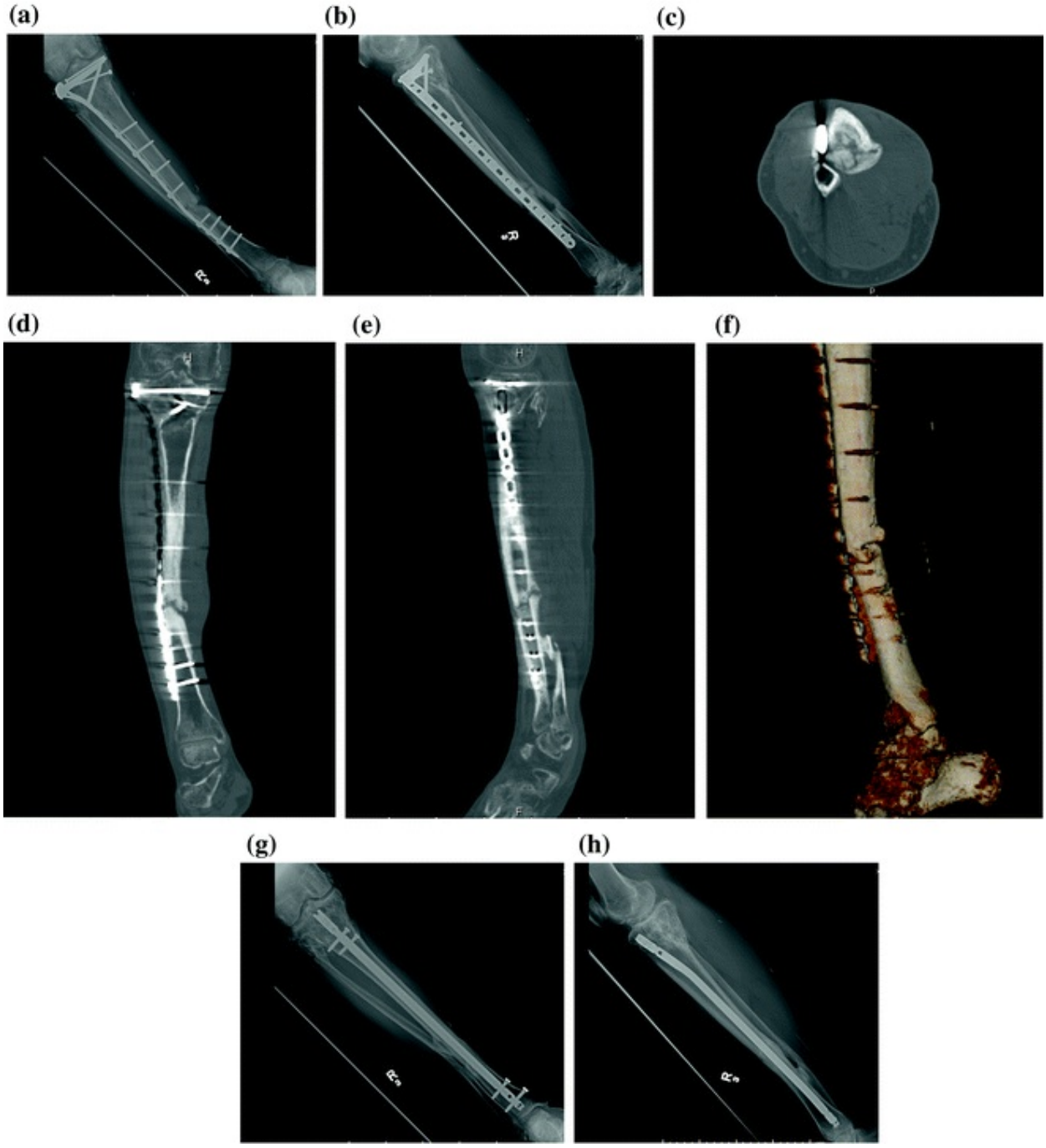



***Fig. 1.6*** Patient referred for nonunion 9 months after treatment for tibial plateau and tibia shaft fracture treated with open reduction and internal fixation. **a, b** Anteroposterior (AP) and lateral radiographs of the tibia show consolidation of the plateau component. There is hardware failure and nonunion of the tibial shaft. **c**–**f** Computed tomography scan images (axial, coronal, sagittal, and 3D reconstructions) which show the subtle hypertrophic nature of the nonunion. **g, h** One year after treatment of hypertrophic nonunion with hardware removal and subsequent reamed nailing (AP and lateral)

Ultrasonography (US) has been shown to have some utility in diagnosing

nonunions [63]. In a study by Moed et al. [64] in which tibia fractures treated with an intramedullary nail were evaluated, the authors showed a sensitivity of 100% and a positive predictive value of 97% in detecting healing of the fracture site. They also could predict healing of these injuries much earlier (38 days versus 127 days) than plain radiography. Chachan et al. [14] in their prospective diagnostic follow-up study showed that ultrasound was able to predict fracture healing 2 weeks earlier than plain radiographs. More importantly, it was able to predict nonunions 8.5 weeks earlier. Despite the earlier detection for a nonunion, US has not become widespread in its use. The benefits of no radiation have not outweighed the primary issues of user dependency, time required for the study and additional cost. Three-dimensional ultrasound is a newer technology that may have added benefits of being able to measure the vascularity not only in the surrounding soft tissue but the fracture itself, as well as providing more information on the progression of healing [63].

Fluoroscopy is another imaging modality that can be used primarily to assess motion at a fracture site to determine healing. This is most useful in the patient treated without internal fixation and when there is a question of the healed status of the injury. It can also be useful in cases where external fixation has been used since the external fixation can be loosened without complete removal and the fracture site stressed. If there is motion, the external fixation can easily be “tightened” and “reset.” In our practice, this is usually done in conjunction with anticipated external fixation removal after definitive management of a fracture or in reconstructive cases, where determining the “laxity” of a nonunion can guide treatment.

Magnetic resonance imaging (MRI) with gadolinium can be used to assess the nonunion site for infection and more importantly for devascularized bone or a sequestrum [31, 65]. Additionally, because of its ability to detect marrow changes, it is very sensitive for osteomyelitis. Osteomyelitis usually shows decreased marrow signal on T-1 images but increased signal on T-2 images (Fig. 1.7a–d). The MRI also allows one to determine the extent of bony involvement [31] in such cases because of the marrow changes which is crucial in determining the best reconstructive option based upon the anticipated length of resection required to eradicate the osteomyelitis.

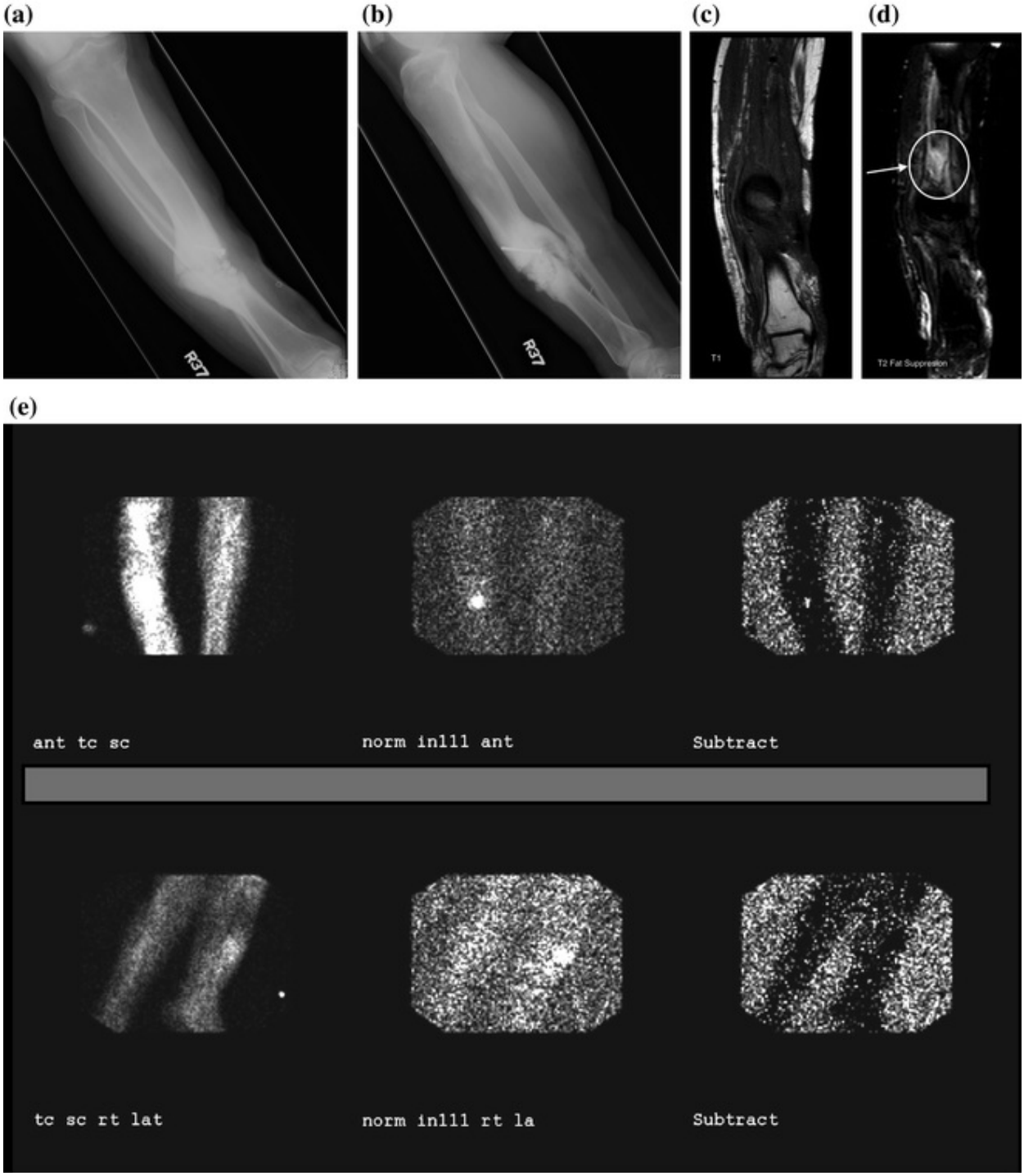



***Fig. 1.7*** **a, b** Anteroposterior and lateral radiographs of patient with infected nonunion of right tibia. **c, d** Magnetic resonnance images of tibia showing increased signal on T2 image indicating osteomyelitis. **e** Nuclear medicine studies showing increased uptake on indium study suggesting the presence of infection

Nuclear medicine studies (Fig. 1.7e) have been historically used to aid in

the detection of infection as well, but over time their utility has been questioned [66]. They are still of use in evaluating the nonunion site for infection and/or biologic activity [31]. Leukocyte-labeled studies have been shown to have appropriate diagnostic accuracy for osteomyelitis in the peripheral skeleton [67]. The traditional technetium bone scan will have increased signal on any biological bone activity, and hence, any fracture site that is biologically active should have uptake. Thus, it really is not used for the evaluation of healing although, in cases of avascular or nonviable fractures sites, e.g., the atrophic nonunion , decreased or no uptake may be the case. Our use is usually for the suspected infected cases when the clinical signs of an infection are absent but laboratory markers—erythrocyte sedimentation rate (ESR) , C-reactive protein (CRP) , or white blood cell (WBC) count —may be elevated. In these cases, a bone scan is obtained which is usually positive. If by chance it is negative for uptake, then no other imaging is done and concern becomes for an atrophic nonunion. After a positive bone scan, an indium (tagged WBC) scan is performed. If this is positive at the fracture/nonunion site, then there is increased suspicion for infection. If it is negative for uptake at the site, then infection is less likely but unfortunately never completely ruled out. The final study done after a positive Indium scan, is the sulfa colloid marrow scan. The areas of uptake are then compared to the indium scan. If the areas of uptake are concordant with the indium scan , the uptake is deemed to be secondary to the associated marrow changes and not infection. Conversely, if the areas of uptake on the indium scan do not coincide with uptake on the sulfa colloid (discordant), then it is thought to be suggestive of an infection [31]. The specificity and sensitivity of such imaging studies has been controversial. Stucken et al. [66] showed that not utilizing the nuclear medicine tests actually improved their predicted probabilities of infection based on laboratory studies alone. The latest imaging modality, which has shown some promise to aid in the detection of infection or osteomyelitis, has been the positron emission tomography (PET) scan ± CT scan. A fluorodeoxyglucose PET scan has been shown to have the highest diagnostic accuracy for excluding or confirming the diagnosis of chronic osteomyelitis [31, 67]. This could aid in the evaluation of the presence of infection in a nonunion.

### 1.4.3 Laboratory Evaluation

Laboratory studies  can assist in determining the etiology of the nonunion or

at least look at conditions that may have contributed to the development of the nonunion. All patients should be evaluated with a CBC with differential, ESR, and CRP. These are utilized to evaluate for infection but realizing that the ESR and CRP are simply indicators of inflammation and can be elevated in the absence of an infected nonunion. Conversely, normal markers do not necessarily rule out an infection either and are usually the case in indolent infections. A standardized protocol to rule out infection was assessed by Stucken et al. [66] to evaluate the efficacy of laboratory studies (WBC, CRP, ESR) and nuclear medicine studies. They found that the ESR and the CRP were both independently accurate predictors of infection. With all three tests being positive, the predicted probability of an infection was 100%. If the nuclear medicine studies were included, the probability went down to 86% for three positive tests.

As mentioned before, in cases where the original surgery was deemed to be highly contributory to the development of the nonunion, more extensive laboratory studies may not be needed. In the cases where the technical aspects seemed to be sound and the reason for the nonunion unclear, other laboratory studies may point to an underlying metabolic abnormality as the etiology [13]. These patients would probably benefit from an endocrinology workup if feasible. Often times in our practice, these are unfunded trauma patients and the workup is often left to the orthopedic trauma surgeon to do the full evaluation. Many of these patients may also have sustained fragility fractures which also warrant laboratory workup. These underlying metabolic disorders include vitamin D deficiency, hypothyroidism, hypogonadism, hypocalcemia, and overall poor nutritional status. Brinker et al. [13] showed that 31 of 37 of their patients with a nonunion had some type of metabolic abnormality with vitamin D deficiency being the most common. The laboratory studies, in addition to the above, should include serum 25-hydroxy-vitamin D, calcium, phosphorus, alkaline phosphatase, thyroid function tests, parathyroid hormone level, hormone levels (testosterone, estrogen, and follicle stimulating hormone), and albumin and cortisol levels [13]. Vitamin D deficiency has been set at 20–30 mg/dl, and <20 are considered insufficient.

In cases of infected nonunions, it is helpful to obtain results of previous cultures if available to determine the previous organism(s). At the time of surgery, especially in cases of staged procedures, which is often the situation in dealing with infected nonunions [66], deep tissue cultures and bone

biopsies can help determine the presence or absence of an infection as well as the offending organism. Preoperative antibiotics should be withheld until after intra-operative cultures are obtained. It is also recommended to cease any antibiotics for at least two weeks, if possible, to maximize the chance of identifying the organism. The first stage is usually to remove previous hardware, to obtain a better idea of the nonunion site, and to get biopsies and cultures. Antibiotic beads can be placed in the interim prior to the second stage.

There has been an increasing interest in looking for serologic markers that may help to predict fracture healing and therefore potentially predict nonunions [16, 20, 22, 27, 30, 68]. Although a full review and discussion of these markers is beyond the scope of this chapter, it is important to mention that they exist and have future implications in predicting fracture healing. These biomarkers are either factors that regulate the healing process itself or bone turnover markers that are extracellular matrix components related to degradation or production during the repair process [15]. The local or systemic factors regulating the healing process include vascular endothelial growth factor (VEGF) and transforming growth factor-beta (TGF-β) . Serum TGF-β has been found to be an indicator of healing versus nonhealing with 4 week levels being much lower in a group of patients that had a delayed union [20]. The bone turnover markers can be divided into one of three categories: 1. bone formation markers, 2. bone resorption markers, and 3. osteoclast regulatory proteins [16, 20, 22]. The bone formation markers indicate osteoblastic activity and as such are fragments of type-I and type-III pro-collagen that are released during the formation of type-III collagen (PIIINP, PICP, PIIINP). Osteocalcin (OC) and bone-specific alkaline phosphatase (BSAP) are also measures of osteoblastic activity. Bone resorption markers include those that measure the degradation of type-I collagen (CTX, NTX, ICTP, pyridinoline, deoxypyridinoline). Tartrate-resistant acid phosphatase (TRAcP) and cathepsin K (CK) are noncollagenous markers that also measure bone resorption but are osteoclast regulatory proteins. Other osteoclast regulatory proteins include receptor activator of nuclear factor NF-kB ligand (RANKL) and osteoprotegerin (OPG). The marker activity of only a handful of these have been evaluated in various fractures and shown some promise in predicting fracture healing [30]. Fischer et al. [18] evaluated a number of cytokines—TGF-β, platelet-derived growth factor (PDGF-AB), insulin-like growth factor 1 (IGF-1)—in patients

with long-bone nonunions treated both successfully and unsuccessfully with the Masquelet technique and compared them to a group with normal bone healing. They found temporal variations of these cytokines in the three groups, with high expressions of IGF-1 corresponding to a successful Masquelet treatment. They demonstrated significant differences in cytokine expression between normal fracture healing and the nonunion treatment groups. If the time profiles of each of these markers can be fully understood, then perhaps variations in these markers from what may be considered the normal in fracture healing may provide insight into which fractures will go on to a nonunion [27]. Earlier detection and subsequent earlier treatment could result in substantial cost savings [20].

## 1.5 Definitions and Classification

As mentioned previously, the US FDA defined a nonunion as a fracture that is at least nine months old and has not shown any signs of healing progression for at least three consecutive months [1]. This however cannot be applied to every fracture, and all nonunions are not the same. Harwood and Ferguson [31] proposed more sensible definitions. They suggested that a nonunion be defined as “a symptomatic fracture with no potential to heal without intervention.” A delayed union was defined as “a fracture in which healing has not occurred in the expected time and the outcome remains uncertain.”

The most common classification was original described by Weber and Cech [69] in 1976 and has survived for 40 years. It was based on the viability and healing potential of the nonunion. From a vascular viewpoint, that corresponds to either a hypervascular or avascular environment [2, 31, 65, 69, 70]. This is based on the appearance of the fracture site on plain radiographs after a period of time when improvement in the fracture healing has ceased. The hypervascular nonunions have been further subdivided into a descriptive classification as an “elephant foot,” “horse hoof/foot,” and oligotrophic nonunion. The avascular nonunions have been further subdivided into the torsion wedge, comminuted, defect, or atrophic nonunion. In addition, the pseudarthrosis has been described [2, 31, 69]. Any of these types of nonunions can be aseptic or septic. If septic, the infection has to be eradicated and any osteomyelitis addressed usually with bone resection. Additionally, these may or may not have a deformity that is associated with it, and if

present, any management needs to address the malalignment.

The hypervascular “elephant foot” nonunion (Fig. 1.8) is based on the appearance of the bone ends. These hypertrophic nonunion s exhibit abundant callus formation and are due to excessive motion at the fracture site from inadequate stability [31, 65]. The motion precludes union of the fracture ends. These are well vascularized and generally do not require a bone graft. These require enhanced mechanical stability, which may involve revision of the hardware or additional fixation. The “horse hoof/foot” nonunion is also hypertrophic but much less so. It usually occurs in a situation of inadequate or unstable plate fixation constructs but can occur with nails [2] (see Figs. 1.6 and 1.9). The “oligotrophic” nonunion albeit hypervascular is not hypertrophic on radiographic appearance (Fig. 1.10). The callus is absent, and some absorption occurs but the ends are viable [2]. It is often times due to inadequate reduction or distraction at the fracture site. Revision of the fixation is dependent upon the integrity of the hardware and need for cortical apposition. All three of these nonunions generally require revision fixation with the aim of improving stability. Bone grafts and other biologic adjuncts are not needed except possibly in the case of the oligotrophic nonunion [70].

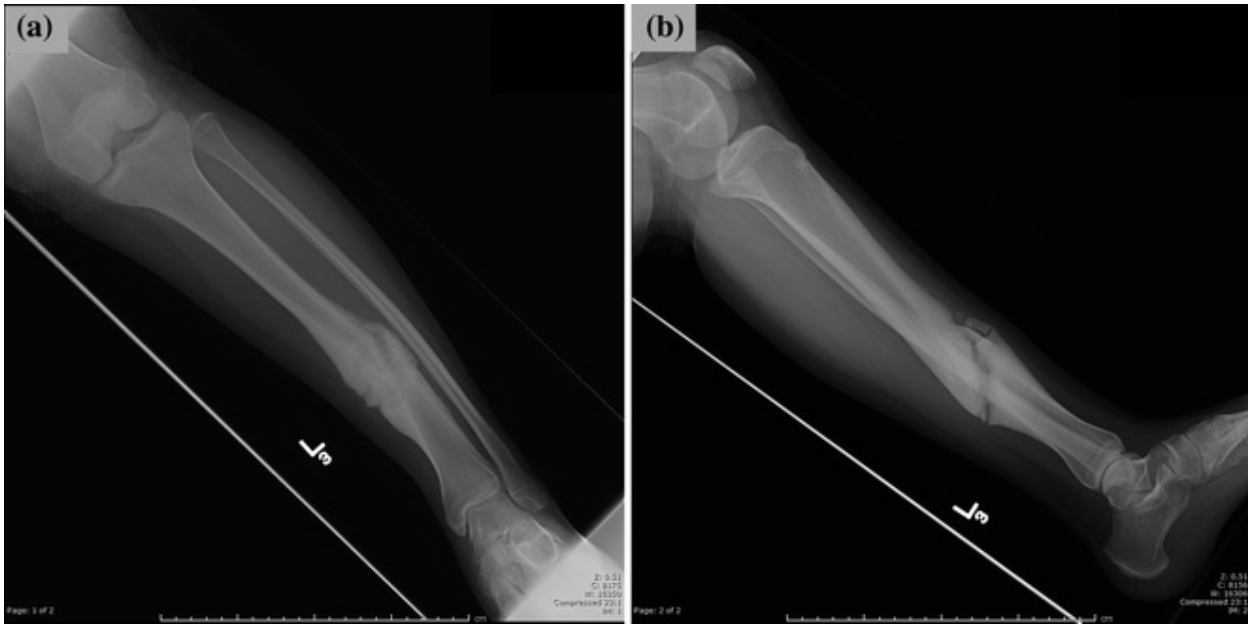



***Fig. 1.8*** **a, b** Anteroposterior and lateral radiographs of a patient with a low energy left tibia fracture treated with cast immobilization that went onto a hypertrophic (“elephant foot”) nonunion

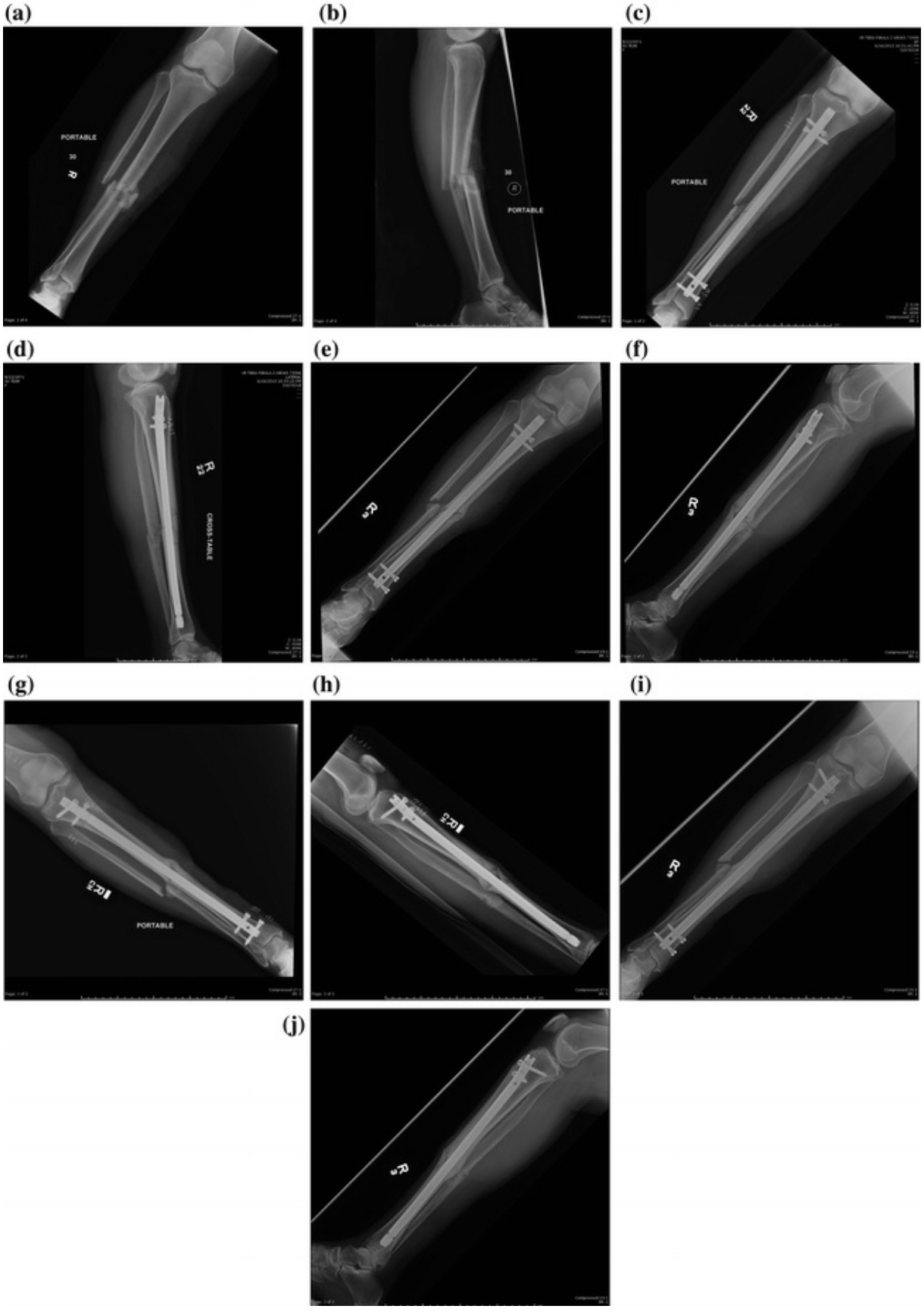

(a)
PORTABLE
(b)
PORTABLE
(c)
PORTABLE
(d)
CROSS TABLE
(e)
(f)
(g)
PORTABLE
(h)
(i)
(j)

***Fig. 1.9*** Patient with a right grade I open tibia fracture treated initially with irrigation and debridement and reamed intramedullary (IM) nailing. **a, b** Injury, anteroposterior (AP) and lateral. **c, d** Postop AP and lateral, follow-up after 10 months showing development of a hypertrophic nonunion and subsequent exchange nailing with union. **e, f** Nonunion AP and lateral. **g, h** Exchange IM nail, AP and lateral. **i, j** Healed AP and lateral


(a)
(b)
(c)
(d)
(e)
(f)

***Fig. 1.10*** Patient with a segmental tibial shaft treated with an intramedullary nail but with distraction noted at the proximal fracture. **a, b** Anteroposterior (AP) and lateral. Patient developed an oligotrophic nonunion at 8 months at both sites. **c, d** Nonunion, AP, and lateral. Patient underwent dynamization with removal of both distal locking screws and subsequently healed. **e, f** Healed, AP and lateral

All of the avascular nonunion subtypes can be considered as having atrophic ends as all are deficient in callus formation, have undergone some resorption, or have significant bone loss at the time of injury [2, 31, 65, 69, 70] (Fig. 1.11). These generally require a biologic stimulus to heal the nonunion with varying degrees of fixation (or revision fixation) and/or soft tissue reconstruction [70]. If the hardware placed appears to be intact and appropriate, then a biologic stimulus may be all that is needed. This is usually in the form of autogenous bone grafting although various bone graft substitutes have been used. Other adjunctive treatments have also been described [70] and will be discussed later.

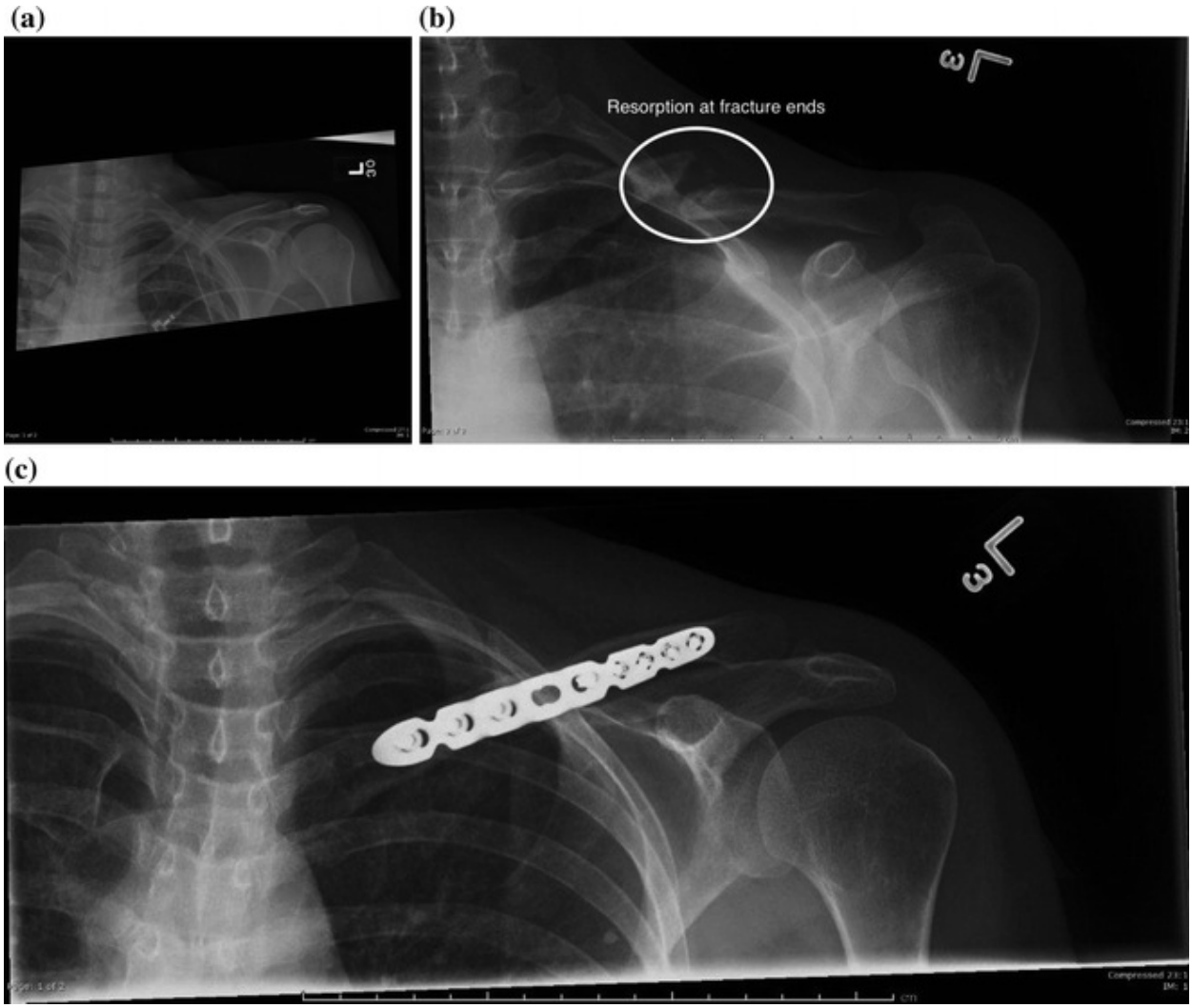



***Fig. 1.11*** Patient with left clavicle fracture without shortening and minimal elevation initially treated non-operatively. **a** Injury radiograph. **b** Nonunion radiograph after 3 months showing resorption and established atrophic nonunion. **c** Healed clavicle fracture after treatment with open reduction and internal fixation and bone graft

A pseudarthrosis is a nonunion that chronically develops into a joint-like appearance with a hypertrophic callus or can be atrophic on radiographs with gross motion [2] (Fig. 1.12). Despite the obvious instability, these are surprisingly nonpainful. In fact, this was defined by Harwood and Ferguson [31] as “a painless fracture that has failed to unite and has no potential to do so without intervention.” These all require surgery. The cavity at the fracture site is usually filled with a synovial lining creating a “false joint.” These require resection of this cavity along with stabilization and bone grafting.


***Fig. 1.12*** Patient with left tibia fracture treated with closed management and development of pseudarthrosis of tibia but healed fibula. (**a, b)** Pseudarthrosis anteroposterior (AP) and lateral; Patient treated with reamed intramedullary nailing of pseudarthrosis after resection synovial cavity with subsequent union.(**c, d**) Healed AP and lateral

Other classification schemes have been reported as well [71–73]. The classical Ilizarov description has been to define nonunions based upon the amount of motion at the site, as stiff, slack, or lax [71]. These correspond to the previously described radiographic appearances as well. It is however important to take into consideration that motion can only be assessed in the absence of intact hardware or adjacent intact structures, e.g., an intact fibula in the case of the tibia. The stiff nonunion (hypertrophic) generally has no detectable motion on stress examination. The slack nonunion (oligotrophic-hypertrophic) has some motion hinging at the fracture site. The lax nonunion (atrophic) has free movement at the fracture site. This classification is often used in the management of nonunions with external fixation [71, 74–76].

Biasibetti et al. [72] reported their classification based on radiographic evaluation. Their preference is for the use of external fixation in the management of these nonunions. They defined nonunions as types 1–4. The type 1 nonunions are the classic hypertrophic nonunion s that require mechanical stabilization by compression. The type 2 nonunions are those with large oblique fragments where axial compression would result in shear and torsion with negative affects on consolidation. Type 3 nonunions are those that were comminuted injuries, have significant defects, or are atrophic. These require both mechanical stability and biologic stimulation. The type 4 is the infected nonunion.

The management of nonunions is extremely complicated, and failure rates have been reported around the 20% level. Despite classification schemes and scoring systems to better provide improved agreement on when to diagnose a nonunion, treatment guidelines are lacking. In an effort to provide such guidelines, Calori et al. [73] in 2008 proposed a new scoring system to classify nonunions and dictate the level of care that the nonunion requires. This scoring system takes into consideration the bone, soft tissues, and the patient to determine the best course of action. The maximum score would be 100 (scored points × 2). The scoring system is very comprehensive and looks at all the issues previously mentioned including the fracture characteristics, adequacy of original treatment, defects, alignment, soft tissue integrity, and patient risk factors. The higher the score, the more difficult it was felt to obtain union. Those with a score up to 25 were felt to have a straightforward nonunion that could be managed by standard techniques. Those with scores from 26 to 50 should have more specialized care. In addition to specialized care, specialized treatment was also required if the score was 51–75. They

recommended consideration for amputation for any score above 75. Although this score looks to have some promise, it has not been validated to our knowledge.

Careful assessment of the radiographs over time can help classify the type of nonunion. The type of nonunion can then help determine the cause of the nonunion suggesting either a biologic or mechanical etiology. Taking all of these previous factors that have been discussed can help determine the best course of action to take in managing the nonunion. Classification and scoring systems can certainly be helpful.

---

## 1.6 Management Principles

In general, the management principles for the treatment of the nonunion are common to all sites and are based on the classification. The goals in treatment of the nonunion are universal: 1. healing the nonunion, 2. restoring function, and 3. eliminating pain. There are two basic tenets in accomplishing these goals—maximize the biology and re-establish appropriate mechanical integrity of the nonunion environment. Maximizing the biology of the environment can be looked at from two perspectives: local and systemic. Locally, it is important to enhance the biology at the nonunion site and eradicate infection if present. Systemically, the patient's comorbidities must be minimized or corrected if feasible. The mechanical integrity of the nonunion environment can be looked at from a local point of view as well as from the entire limb point of view if u will. Keep in mind that improvement of the local mechanical stability also improves the local biology at the nonunion site to promote union. It has also been suggested that nonunions should be treated with polytherapy, insuring the nonunion site is enhanced with osteoprogenitor cells, growth factors, and an adequate osteoconductive scaffold in cases of adequate stability [77]. This is certainly an aggressive approach and may be warranted if these three aspects of the diamond concept [11] are lacking.

### 1.6.1 Biological Environment: Systemic

After a careful evaluation of the patient and causes for the nonunion, any metabolic abnormalities should be addressed. Vitamin D deficiency or insufficiency should be corrected with replacement vitamin D therapy. Our

preference is to start patients at 50,000 units of vitamin D weekly for at least 6 months. Levels should be obtained after 4–6 weeks to insure a proper response. In patients with associated secondary hyperparathyroidism, vitamin D replacement should solve the high PTH level. Patients should also be given calcium supplementation along with the vitamin D. Smoking cessation counseling should be initiated in efforts to minimize or even stop smoking to aid in the healing process after reconstruction. Diabetes should be as well controlled as feasible. All comorbidities should be optimized prior to intervention if time allows.

### 1.6.2 Biological Environment: Local

Infection should be ruled in or out prior to any definitive management. If infection is present, the decision between a one-stage and two-stage treatment plan must be made [78]. If a two-stage approach is deemed necessary, the first step is to remove existing hardware, evaluate the nonunion site, and obtain cultures and/or biopsies to determine the presence and extent of the infection. The presence of osteomyelitis must be determined as well as the extent of bony involvement. If there is an obvious infection, the infection must be cleared up prior to wound closure and certainly before definitive management for the nonunion including bone grafting [78]. There are varied opinions on when timing of the bone graft should occur from immediate to 6 weeks after the resection [35]. If osteomyelitis is present or suspected and confirmed by biopsy, the amount of bony resection that needs to be performed to eradicate the infection has to be determined. As mentioned before, MRI is quite useful since marrow changes can help delineate the extent of osteomyelitis. Reconstruction options for bony defects are discussed in Chap. 15, but include the Masquelet technique with massive bone grafting, distraction osteogenesis (bone transport) [71, 74], and vascularized or nonvascularized bone grafts (Figs. 1.13 and 1.14). Struijs et al. [79] reviewed the literature on the management of infected long-bone nonunions. The majority were case series, and definitive conclusions and recommendations could not be made. However, it was clear that appropriate debridement is universally required as a basis for any further treatment. The majority of the first-stage treatment methodologies, when significant bone resection is required for associated osteomyelitis, included bone transport techniques with the Ilizarov fixator after the debridement with 70–100% union results. In a retrospective review of utilizing a single-stage treatment protocol for

“presumptive” aseptic nonunions, the authors had success in preventing secondary surgery in 72% of culture positive cases [80]. If preoperatively, the history and clinical examination do not indicate an infection, a single-stage protocol of withholding antibiotics, removing the implant, open debridement or canal reaming, sending cultures followed by antibiotics and revision ORIF or exchange nailing was performed. They had positive cultures in 28.7% and out of those 28% needed secondary surgery. Overall, they felt that a single-stage protocol is warranted in cases where the nonunion is considered aseptic preoperatively. In obvious cases of infected nonunions, a single-stage protocol is not utilized. The best two-stage results (93–100% union with recurrent infection rate up to 18%) seemed to be with debridement, antibiotic beads, and planned secondary fixation. In a study by Obremskey et al. [35] where they surveyed members of the Orthopedic Trauma Association (OTA), almost 90% of surgeons used some sort of antibiotic cement spacer before bone grafting in a segmental defect. In any event, treatment needs to be individualized for the patient. Such specialized techniques as using antibiotic impregnated cement-coated nails for the interim stabilization in infected long-bone nonunions are mainstay of treatment. Recently, Scolaro and Mehta [81] described the use of antibiotic impregnated cement-coated locking plates in the use of infected peri-articular nonunions. In a similar fashion to utilizing the antibiotic impregnated cement-coated nail to stabilize the nonunion temporarily while the infection is cleared, a plate was used in peri-articular nonunions for the same reason with success, albeit a small case series. In addition, the infection must be addressed with appropriate antibiotics. The duration of and mode of delivery (intravenous and *per os*) also should be based on the organism, bone penetration of the antibiotics, and the retention of potentially contaminated hardware. Much of the treatment plan is based on the surgeon’s experience since each case can be unique. Amputation should always be discussed with patients that have infected nonunions [71, 73]. If infection is present, the reconstructive path can be long and difficult. Many patients have already undergone numerous surgeries over several years to no avail in resolving the infection or nonunion.

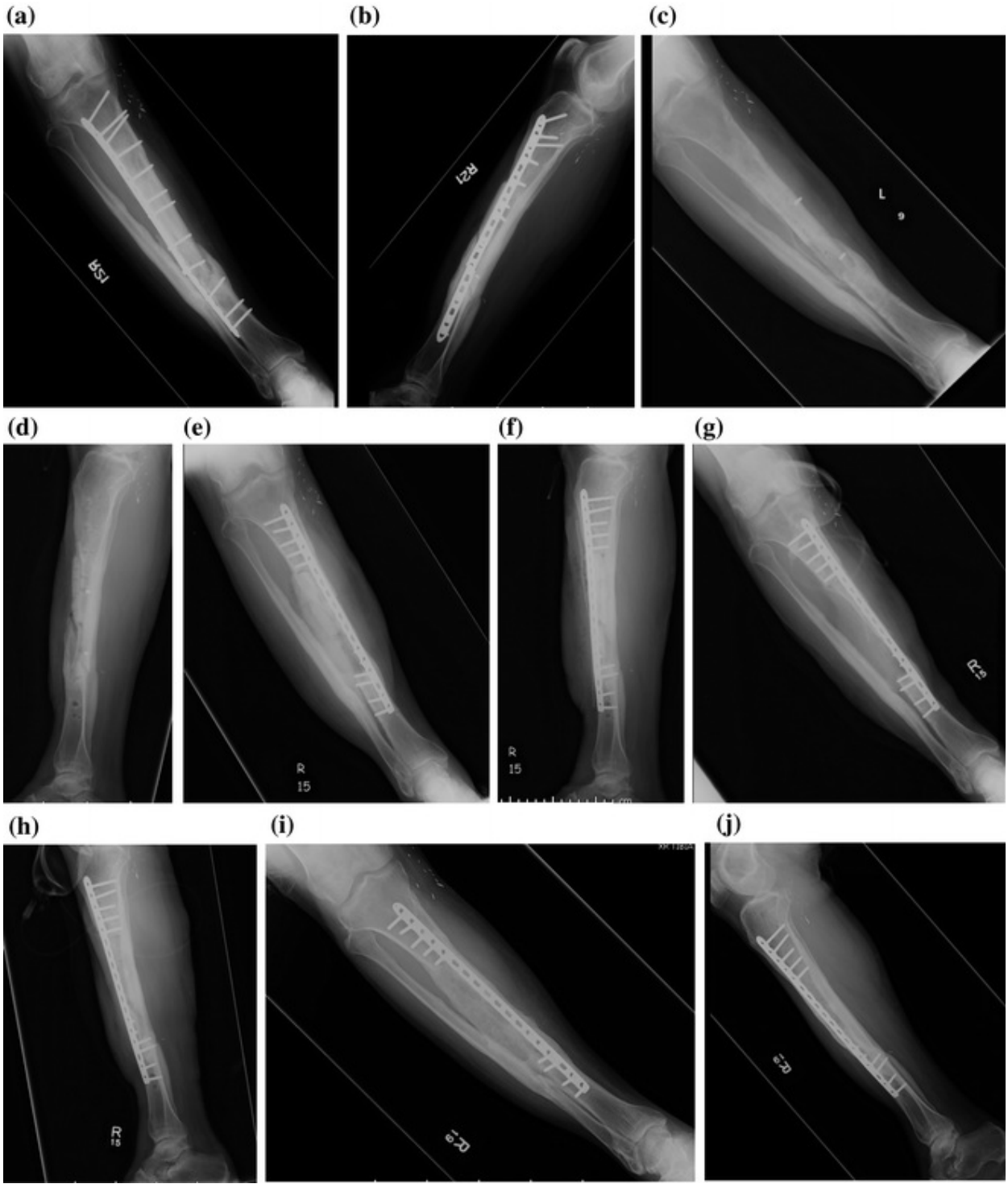



***Fig. 1.13*** Patient presented with a year history of draining sinus tracts after open reduction and internal fixation (ORIF) of grade IIIB right open tibia fracture concerning for infected nonunion. **a, b** Infected Nonunion anteroposterior (AP) and lateral. Patient underwent infection workup, which was consistent with osteomyelitis. The patient then had hardware removal with evaluation and biopsy of the bone. **c, d** AP and lateral after hardware removal. Patient then underwent resection of osteomyelitis with plating and cement spacer placement to create membrane in anticipation of Masquelet procedure. **e, f** AP and

lateral after ORIF and cement placement. Bone grafting into membrane, which was obtained from ipsilateral femur, using reamer-irrigator-aspirator system (RIA$^{TM}$). **g, h** AP and lateral after cement removal and bone grafting. Patient went on to heal with complete consolidation of the bone graft. **i, j**. One-year follow-up AP and lateral of tibia


***Fig. 1.14*** Patient referred after sustaining right Grade IIIB open femur fracture with massive bone loss.

Initial treatment was irrigation and debridement and retrograde intramedullary nail with placement of bone cement in defect in anticipation of Masquelet technique. **a, b** Presenting anteroposterior (AP) and lateral showing bone defect with cement. Patient underwent bone harvesting from contra-lateral femur using the reamer-irrigator-aspirator system and then placed into defect after cement removed. **c, d** Initial postop AP and lateral radiographs after bone grafting. Patient went on to consolidate after the bone grafting with complete healing across the defect. **e, f** AP and lateral showing consolidation and incorporation of bone graft into defect

Atrophic or oligotrophic nonunions require a biologic stimulus to reinitiate the healing process. This is usually in the form of a bone graft. Autogenous bone graft remains the gold standard [31, 78, 82–84] because it is osteogenic, osteoinductive, and osteoconductive, with the iliac crest (ICBG) as the most common site of harvest historically [31, 84, 85]. In a retrospective study of long-bone nonunions by Flierl et al. [86], they compared the success rates of 5 different groups: autograft, allograft, autograft + allograft, recombinant human bone morphogenetic protein-2 (rhBMP-2) ± adjunctive bone grafting. The autograft was superior in union time, had the lowest rates of surgical revisions and revision bone grafting, and had a lower new-onset postoperative infection rate. Obremskey et al. [35] surveyed members of the OTA and 92% use autograft for bone grafting procedures in nonunions. The site from where the bone graft was obtained varied, however, with 50.9% of the respondents picking the reamer-irrigator-aspirator system (RIA$^{TM}$) , 49.9% chose anterior crest, and 24.8% for posterior crest (more than 1 choice was allowed). Only 20.8% of surgeons used allograft and BMP as an alternative bone graft. Since the invention of the RIA$^{TM}$ system, it has been used more and more for harvesting autogenous bone graft. Those that have used it cite lower complications and lower comorbidity than ICBG harvesting.

Dimitrou et al. [85] performed a systematic review of the literature and found that the overall complication rate for RIA$^{TM}$ was 6% compared to 19.4% for ICBG harvesting. They also showed that there were differences between the anterior and posterior crest harvest sites. The anterior crest had significantly higher rates of infection, hematoma formation, fracture, and hypertrophic scar formation but significantly lower rates of chronic donor site pain and sensory disturbances. In a separate clinical study by Loeffler et al. [87], they prospectively enrolled 92 patients undergoing anterior ICBG for nonunions. They had a 3% infection rate and only 2% rate of chronic pain. They felt that anterior ICBG harvesting was well tolerated. In addition to comparing complication rates between RIA$^{TM}$ and ICBG, there has been

concern that the bone graft quality (cellular constituents and biochemical characteristics) from the intramedullary canal is not as good. Sagi et al. [88], in a prospective study, harvested bone graft from both the medullary canal (RIA$^{TM}$) and the iliac crest from the same individual for nonunion procedures. They evaluated the graft histologically and performed transcriptional profiling for biochemical markers that are known to be expressed during fracture healing. The transcriptional profiles were found to be very similar. The RIA$^{TM}$ graft was found to have greater regenerative characteristics as well as mesenchymal stem cells. This suggests that RIA$^{TM}$ bone graft may actually be better.

Dawson et al. [89] looked at the union rates between RIA$^{TM}$ and ICBG in a prospective randomized study for nonunions or a post-traumatic defect that required operative intervention. They had 113 patients for the final statistics, 57 patients received ICBG, and 56 had RIA$^{TM}$ grafting. The union rates were similar as were the rates of donor site complications, but the RIA$^{TM}$ had larger volumes of graft (anterior ICBG SS $\ll$ than posterior ICBG NSS < RIA$^{TM}$) and had significantly less donor site pain. Autogenous bone graft remains the gold standard, but the choice of harvest site is still at the surgeons' discretion. Adjunctive bone substitutes are sometimes required for recalcitrant nonunions or if more volume is needed. The author prefers to use RIA$^{TM}$ especially if large volumes of bone graft are required or the canal is being accessed due to the implants being used (intramedullary nails). Other local donor sites have been utilized for small amounts of graft depending on the site of the nonunion, e.g., distal radius metaphysis for forearm nonunions or proximal tibia metaphysis for distal tibia/fibula nonunions. If the patient is obese, the preference is also for RIA$^{TM}$ to avoid wound complications with the large soft tissue envelope.

Another alternative for large bone defects besides massive autogenous cancellous bone grafts are the vascularized bone grafts [90]. In certain situations, a vascularized graft may be the best option. Not only can they provide better structural support but can promote healing due to the added blood supply. Numerous types of vascularized bone grafts exist [91], but it does require a surgeon with microvascular skills and they tend to have more issues. Historically, it was felt that if large structural grafts were used (>6 cm), then it should be vascularized. Allsopp et al. [90] in reviewing the literature found no evidence to support this perception nor did it support that

the success was superior to nonvascularized grafts. The technique is still useful and a valuable part of one's armamentarium. It may be beneficial in particular situations such as nonunions complicated with osteonecrosis, e.g., the femoral neck or scaphoid [91].

One of the advantages of autogenous bone grafts is that it contains the patient's own osteoprogenitor cells which aid in its osteogenic potential. Other sources that can provide osteogenesis are bone marrow and peripheral blood. Bone marrow (BMA) can be aspirated providing a source of osteoprogenitor cells that have been shown to provide a biologic stimulus to aid healing in nonunions [31, 70, 82–84, 92–95]. It is considered both osteogenic and osteoinductive. Peripheral blood can be obtained and then centrifuged by a variety of commercially available proprietary systems that separate out the platelet rich plasma (PRP), which has shown mixed results in aiding facture healing [31, 50, 84]. The PRP is only considered osteoinductive.

A large bore needle is inserted into the iliac crest in order to aspirate the bone marrow. The aspirate can then be directly injected into the nonunion site under fluoroscopic guidance [70, 94]. The technique is useful when the retained hardware is intact and stable. Braly et al. [92] published their case series in eleven consecutive patients that presented with delayed union or a nonunion of the distal tibia metaphysis that were initially treated with ORIF. They had 9 of 11 patients heal within six months. They found it to be a safe and inexpensive, minimally invasive treatment. However, the use of BMA is limited because of the small number of stem cells obtained [82, 84]. In an effort to increase the number of stem cells, multiple aspirations and cell concentration techniques have been described [83]. Other future concepts include culturing aspirated cells to increase the numbers and combining these cultured cells with specific scaffolds during surgery or in the laboratory creating hybrid constructs for implantation [83, 93, 95].

To obtain PRP, peripheral blood is obtained from the patient. The amount of blood required depends on the commercial PRP concentration centrifuge systems available. The blood is placed in the centrifuge and the PRP separates out. It can then be drawn into a syringe and injected at the nonunion site [31]. The PRP contains numerous growth factors but in low concentrations [84]. The clinical outcome in the use of PRP has been extremely varied and thus has not gained wide acceptance.

Bone morphogenetic proteins (BMPs) have been considered the most

important growth factor in bone formation and healing. Although there are many BMPs that have been described, only three have been shown to stimulate stem cell differentiation into the osteoblast lineage in vitro—BMP 2, 4, and 7 [96, 97]. Only recombinant BMP 2 (rhBMP-2:Infuse; Medtronic Sofamor Danek, Memphis, TN) and 7 (rhBMP-7: Osteogenic Protein-1 [OP-1]; Stryker, Kalamazoo, MI) have been cleared by the FDA for clinical use, with rhBMP-2 indicated for open tibia fractures and rhBMP-7 for recalcitrant long bone nonunions [98, 99]. There has been extensive research looking at these molecules as a way to repair nonunions and accelerate the fracture healing process [96, 97, 99]. Friedlaender et al. [100] reported on their prospective, randomized control multicenter study utilizing OP-1 in the management of tibial nonunions. All patients had an intramedullary rod and either OP-1 or autograft. At 9-month follow-up, there was a clinical success rate of 85% and a radiographically healed rate of 84% in the autogenous bone graft group compared to 81% and 75%, respectively, in the OP-1 group. There was no statistically significant difference between the two patients, although 20% in the autograft group had chronic pain at the donor site. OP-1 was felt to be safe and effective for tibial nonunions. Dimitriou et al. [101] utilized rhBMP-7 in 25 consecutive patients with 26 nonunions in various locations. They had success in 24 of the 26 nonunions; however, 16 of these successes also had autograft in addition to the BMP. Only 8 cases had rhBMP-7 alone. In an observational retrospective, nonrandomized study by Ronga et al. [102], they also looked at the use of rhBMP-7 in long-bone nonunions. They had an 88.8% success rate with an average healing time of 7.9 months. Their group was mixed in that 38 cases had the BMP alone, 11 cases were BMP with an osteoconductive agent, 50 cases with an autograft, and a composite graft in 6. In both these studies, the only conclusion that could be made was that the use of rhBMP-7 was safe and effective and could be utilized with autograft. Giannoudis et al. [103] specifically looked at the effect of BMP-7 with autograft. They retrospectively reviewed their prospective database of patients treated for atrophic nonunions in which both BMP-7 and autograft were used in all patients at different anatomic sites. Revision of the fixation was also performed in 77.8% of cases. They had a 100% union rate. They concluded that although autograft was the gold standard, the BMP-7 could enhance the osteoinductive capacity of the graft. Morison et al. [104] looked at the use of rhBMP-7 in atrophic long-bone nonunions in the upper extremity. They used BMP alone but with plate

fixation. However, they did state that if local autogenous bone was available, it was morselized and added but autogenous bone graft was not harvested. They had an 89% success rate.

As mentioned before, the other BMP that is available is rhBMP-2. This has been primarily studied in open tibia fractures with little to no data in nonunions to our knowledge. Jones et al. [105] in the BESTT-ALL trial used BMP-2 with allograft versus autograft in open tibia fracture bone defects. The average size of the defect was 4 cm (1–7 cm). The success rates were not statistically significantly different with a 67% union rate in the autograft group and 87% in the BMP-2+ allograft group. In a study by Aro et al. [106], rhBMP-2 was used in conjunction with reamed intramedullary nail fixation of open tibia fractures and compared to reamed nailing alone. They found that the use of BMP-2 did not accelerate the rate of fracture healing, despite the trend toward faster healing at the 13-week mark in the BMP-2 group. This difference normalized at 20 weeks, where 68% of the BMP-2 group and 67% of the nail alone were healed.

Multiple reviews of the literature [97, 98, 107] have all concluded that although there was good clinical data on the effectiveness of BMPs, it was as good but not better than autogenous bone graft. The use of BMPs can be expensive, but a cost–benefit analysis has shown that their use can potentially provide a cost savings in both nonunions and open fractures [97–99, 103]. More prospective, randomized clinical studies are needed to determine the true effectiveness of BMPs in both nonunions and acute fractures.

There are many other bone graft substitutes, either derived from human sources or manmade, that are commercially available [31, 82, 84]. They include the calcium phosphate substances, bioactive glass, coral, allograft, and demineralized bone matrix (DBM). The synthetic substitutes and allograft are strictly osteoconductive, whereas the DBM is both osteoconductive and osteoinductive, although the osteoconductive potential is highly variable based on the company [84]. Most of these materials are best used as graft extenders in the management of nonunions. No good clinical studies exist evaluating these materials in the treatment of nonunions.

It is also important to note that in cases of open injuries, soft tissue management is integral to the initial treatment. Poor soft tissues and inadequate vascularity may contribute to the development of a nonunion. In all cases of nonunions, the local soft tissue environment should be appropriately assessed. If the soft tissues are deficient or damaged, it is

important to obtain good soft tissue coverage through the use of either local or free tissue flaps [31].

### 1.6.3 Mechanical Environment: Local

The fixation may or may not need revision depending on the technical considerations with respect to the management of the initial fracture and its integrity. If appropriate and intact, then the biologic stimulus may be insufficient. If the original fixation was inadequate or has failed, the construct should be appropriately revised and the need for bone graft assessed based upon the initial healing response. The radiographic appearance of the nonunion should be used to classify the site, which can aid in the management and determination of the need for bone graft.

In cases of hypertrophic nonunion s, stability is needed (see Figs. 1.6g, h and 1.15). The best treatment is based largely in part due to the initial implant used for the original fracture. In cases of fractures previously treated with intramedullary nails, exchange nailing is regarded as the method of choice for both the femur [65, 108–110] and tibia [107, 108, 111] (see Fig. 1.9g–j). Nail dynamization is best reserved for cases of static locking and oligotrophic nonunions to stimulate the healing response [65] (see Fig. 1.10e, f). Care must be taken in cases of comminuted or oblique fractures, where dynamization could lead to unacceptable shortening or loss of rotation. In a review of the literature on aseptic tibial nonunions, Kanakaris et al. [107] in 2007 concluded that exchange nailing was the method of choice based upon better than 90% union rates. In looking at the literature on femoral nonunions, Crowley et al. [109] also found excellent rates with exchange nailing and felt that it remained the gold standard despite good results with adjunctive plate fixation. Swanson et al. [110, 111] reported their excellent results utilizing a systematic approach in both femoral and tibial nonunions regardless of classification. All patients had correction of any metabolic or endocrine abnormalities. The atrophic nonunions did not have open bone grafting. The femurs underwent secondary dynamization in 28% of the cases and the tibias in 7% of cases. There were 4 cases (9%) that had partial fibulectomy at the same time as the exchange nailing in the tibia cases. They had a 100% union rate in femurs and 98% union rate in tibias. In both studies, they routinely exchanged nails with a size at least 2 mm larger in diameter in static mode but used a different manufacturer’s nail. The use of a different manufacturer’s nail was felt to be important to optimize screw purchase since

the screw locations/trajectories would be different. Other more recalcitrant long bone hypertrophic nonunions that have failed exchange nailing may be better off with adjunct plate fixation, which has been shown to be effective in these situations especially for the femur [65, 108, 109] (Fig. 1.16). In cases of plate fixation and hypertrophic nonunion s, often times the entire construct needs to be removed and completely revised. In these cases, the hardware has often failed with resultant mal-alignment. The healing actually may continue because of the excessive motion causing increased callus and a stiff nonunion. Many times, these can be managed with distraction osteogenesis utilizing external fixation, which also allows for correction of the deformity at the same time and subsequent healing of the nonunion [74–76, 112] (Fig. 1.1d–m). The frames can provide an excellent mechanically stable environment to provide healing. Distraction osteogenesis, by applying an Ilizarov circular fixator, was used in a case series of 16 hypertrophic mal-aligned nonunions [75]. They had complete correction of the deformity and 100% union. Feldman et al. [112] used the Taylor spatial frame (TSF) in conjunction with bone grafting to heal 5 atrophic nonunions in addition to two hypertrophic nonunions with 100% success. Schoenleber and Hutson [76] reported their results on eight patients utilizing either an Ilizarov fixator or the TSF for distraction osteogenesis also with 100% success. Other times, revision of the internal fixation is warranted to either a new plate construct, or in some cases depending upon the anatomic site in question, intramedullary devices can be successful in obtaining union if the intramedullary canal is still patent or can be re-established.

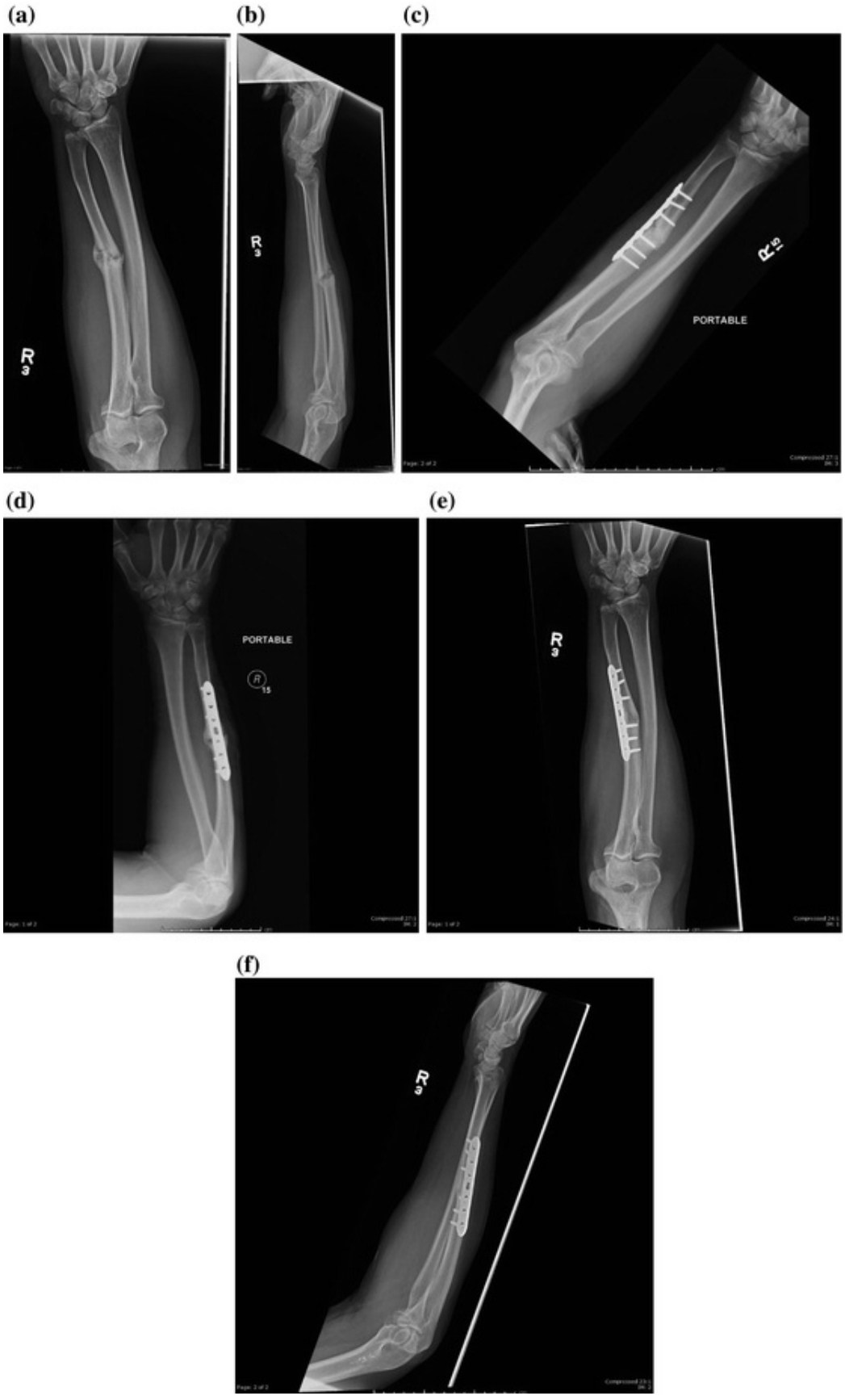

(a)
(b)
(c)
PORTABLE
(d)
PORTABLE
(e)
(f)

***Fig. 1.15*** Patient sustained a “nightstick” fracture to right ulna after an assault and was treated with cast immobilization and subsequent bracing but developed painful nonunion (**a, b**) anteroposterior (AP) and lateral of established hypertrophic nonunion. Patient required stability and underwent open reduction and internal fixation (ORIF) with plate fixation. **c, d** AP and lateral after ORIF. The fracture healed once stability was obtained. **e, f** One-year follow-up AP and lateral showing the healed ulna


(a)
(b)
(c)
(d)
(e)
(f)
(g)
PORTABLE
(h)
PORTABLE
(i)
(j)

***Fig. 1.16*** Patient referred for nonunion of left femur that had undergone four prior surgeries over a two year period. **a, b** Presenting anteroposterior (AP) and lateral radiographs showing oligotrophic nonunion. Patient underwent exchange nailing with bone graft harvesting from the same femur using the reamer-irrigator-aspirator (RIA$^{TM}$) system and placement of bone graft at the fracture site. **c, d** Postop AP and lateral radiographs after exchange intramedullary nail and bone grafting. Patient continued with pain and now a hypertrophic nonunion 8 months later. **e, f** AP and lateral showing development/conversion into a hypertrophic nonunion. Patient with persistent pain and instability at fracture site now with recalcitrant nonunion. Adjunctive plating was performed to provide increased stability. **g, h** Postoperative AP and lateral radiographs after plating; Patient subsequently had resolution of his pain with complete healing of the nonunion. **i, j** One-year follow-up AP and lateral radiographs showing complete consolidation of the nonunion site

In cases of atrophic nonunions with hardware failure, both the local mechanical and biological environment (see above) need to be addressed. Revision fixation is warranted if the hardware has failed or was inadequate at the outset. Anticipation of the need for early bone grafting in cases of bone loss can help prevent hardware failure and can lead to a successful outcome.

## 1.6.4 Mechanical Environment: Limb

As mentioned, any associated deformity must be addressed when dealing with the nonunion. Repair of the nonunion without correction of any deformity, especially the mechanical alignment in the lower extremity, will often fail to restore the proper biomechanics and result in persistence of the nonunion [31]. Correction of the biomechanics is crucial for many nonunions such as the femoral neck, where valgus intertrochanteric osteotomy can be successful as long as excessive valgus alignment is avoided [61]. The length, alignment, and rotation should always be assessed in patients and addressed at the time the nonunion is if there are issues. If the nonunion does heal despite ignoring the malalignment, a malunion will be created which can in and of itself be problematic for the patient [61]. It is imperative to fully evaluate the associated deformity with the appropriate radiographs (see previous section), scannogram for length, and CT scan for rotational issues if warranted. All bones in the particular limb should be assessed for any mal-alignment with long limb standing films. A detailed physical examination should be performed to assess for any compensatory changes in the adjacent joints.

## 1.6.5 Adjunct Therapies

In addition to the surgical management of nonunions, noninvasive interventions in the form of bone stimulators have been used to help facilitate fracture healing acutely as well in cases of delayed unions or nonunions [108]. They come in three forms: 1. ultrasound , 2. extracorporeal shock waves (ESWT), and 3. electrical stimulation [70]. The clinical data are varied.

Low-intensity pulsed ultrasound (LIPUS) has the most clinical data and has been shown to enhance bone healing safely [113–119]. The literature has shown that ultrasound can reduce the healing times of fresh fractures of the radius and tibia, can offset the negative effects of smoking and age on fracture healing, and can be effective in the treatment of delayed unions and nonunions [114–119] (Fig. 1.17). Heckman et al. [115] evaluated the use of LIPUS in acute tibia fractures. They had 67 fractures that were all treated with long leg immobilization. They showed that LIPUS significantly decreased the time to clinical union and overall union (clinical and radiographic) compared to the nontreatment group. Cook et al. [116] looked at patients that had either tibia fractures or distal radius fractures and that smoked to see whether LIPUS showed a difference in healing with smokers. In the tibia fracture groups, they showed a significant 41% reduction in healing time in the smokers and a 26% significant reduction in healing time in the nonsmokers. Smokers had a significant 51% reduction in healing time and nonsmokers a significant 34% reduction in healing time for the patients with distal radius fractures. Nolte et al. [118] reported their results in 29 cases of nonunions treated with LIPUS . They had an 86% success rate with an average treatment time of 22 weeks. This was a heterogeneous group of nonunions including both a variety of anatomic locations and types of nonunion. In a review of the literature, Watanabe et al. [119] showed that the reported success rates seemed to be better for more subcutaneous bones than the deeper bones in both delayed unions and nonunions. Overall rates in prospective cohort studies were reported as anywhere from 55 to 100%. It has also been suggested that LIPUS has utility in distraction osteogenesis and can reduce the time required for maturation of the callus [114, 119].

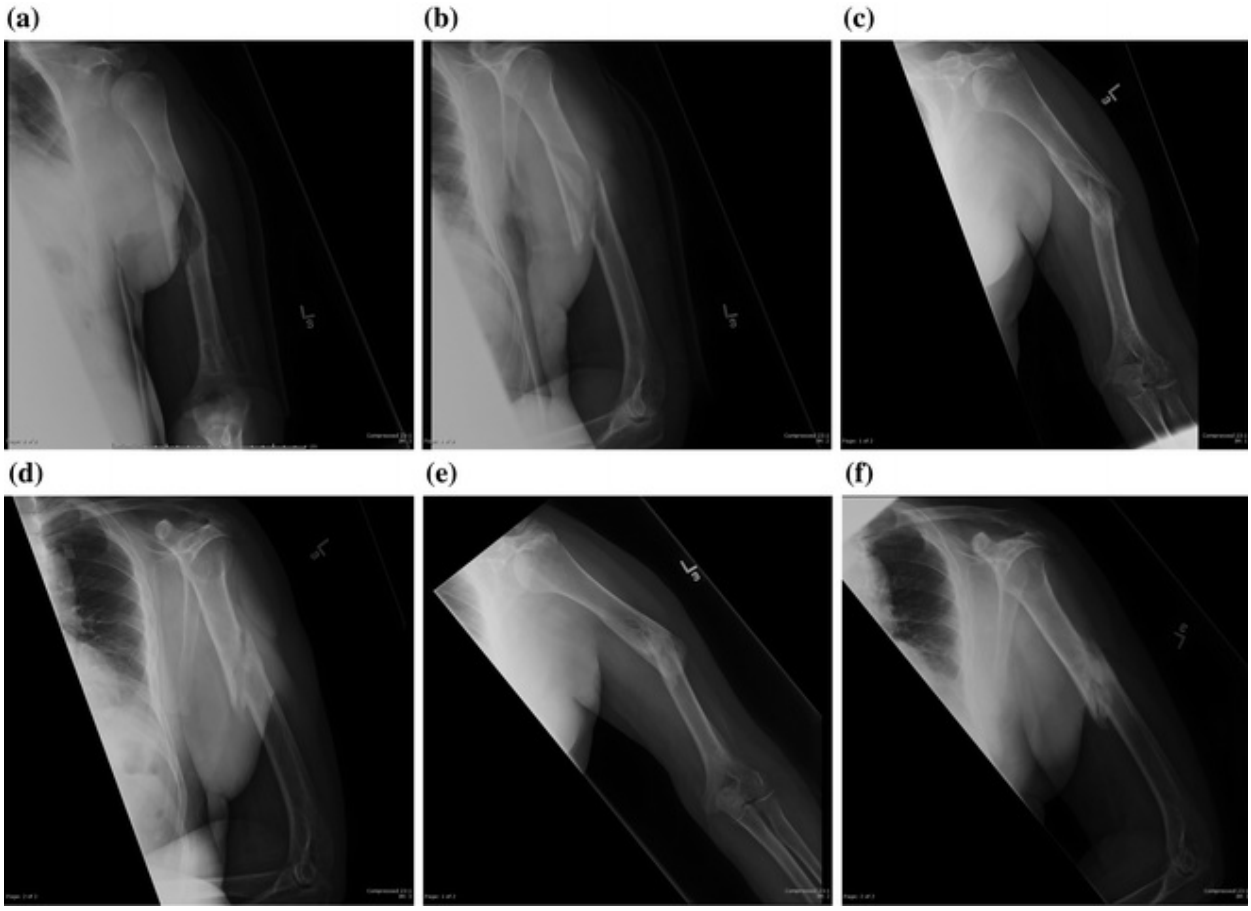



***Fig. 1.17*** Elderly male presented with left humeral shaft fracture after low energy fall. Patient had significant medical comorbidities and decision was made to manage the patient with bracing. **a, b** Injury anteroposterior (AP) and lateral radiographs in brace showing excellent alignment. Patient continued with mild discomfort and radiographs showed persistent nonunion. **c, d** AP and lateral radiographs showing nonunion. Discussion with patient and family was to try alternative methods due to high surgical risks. Ultrasound bone stimulator (Exogen$^{TM}$) was started and patient went on to heal the fracture. **e, f** AP and lateral radiographs 6 months later showing healed fracture

Another form of ultrasound therapy is ESWT. In ESWT, shock waves that are single high-amplitude sound waves which are generated by various means. It has been evaluated in the treatment of delayed unions and nonunions. Zelle et al. [120] in a systematic review of the literature found ten-level 4 studies using ESWT for this purpose. The overall union rate was 76% and was found to be significantly higher in hypertrophic nonunions (76%) than atrophic nonunions (29%). They concluded that the cumulative data suggests that ESWT can stimulate the healing process; however, further studies are warranted due to the level of evidence in these studies.

There are several types of electrical stimulation available: (1) capacitively

coupled electric field (CCEF) , (2) pulsed electromagnetic fields (PEMF) , (3) direct current (DC) (more invasive), and 4. combined magnetic fields (CMF) [70, 121]. In 2008, Mollon et al. [122] performed a meta-analysis of randomized control trials looking at the use of electrical stimulation in long bone fracture healing. They could not show a benefit of its use in improving the rate of union in fresh fractures, delayed unions or nonunions. They did cite the heterogeneity in the studies as a reason for the lack of recommendations either way. In a subsequent review of the literature in 2010, Goldstein et al. [123] reviewed 4 separate meta-analyses on electrical stimulation in fracture healing. That review also concluded that no clear benefit to the use of electrical stimulation was seen. They felt that the meta-analysis by Mollon et al. [122] was the most methodologically rigorous. It was clear that better studies were needed. Adie et al. [124] published their multicenter, double-blind randomized trial on the use of PEMF stimulation for acute tibial shaft fractures. They showed that the use of PEMF did not reduce the number of secondary procedures needed for delayed unions or nonunions. Additionally, it did not improve union rates or patient-reported functional outcomes in acute tibial shaft fractures.

The studies available on adjunct therapies indicate that LIPUS has a much more positive response in delayed unions and nonunions as well as in certain fresh fractures. However, none of the studies provide guidelines as to when it should or should not be used. They can be of benefit in patients that may not be in the best health to undergo surgical procedures. The clinical decision-making should be based on one's experience, patient's needs and wants, and the type of nonunion.

## 1.7 Summary

The best management in treating nonunions is their prevention. Adhering to basic AO principles of fracture fixation and limiting the soft tissue dissection are paramount to a good result. Iatrogenic causes have been shown to be a significant contributor to nonunion development [25]. The soft fracture callus that begins to form right away has healing potential as shown by Danoff et al. [125] in an animal model. They created a mid-shaft femoral shaft fracture in rats and stabilized it with intramedullary nailing. They exposed the fracture site at seven days and created three study groups. In the first group, none of the soft callus was removed. In the second, the soft callus was removed. The

final group had the callus removed and then replaced. The callus removal group showed significant evidence of delayed healing. Replacing the callus mitigated the negative effect on the healing. They recommended replacing the soft callus on all ORIF procedures. In addition to limiting the biologic insult of surgery, all fracture patients should be critically evaluated for comorbidities that may also contribute to nonunion development as mentioned before. Early bone grafting when appropriate should be performed to aid fracture healing when defects are present and promote healing and prevent hardware failure. If a nonunion presents, reassessment of the patient is required. Critical evaluation of the initial treatment should be performed. If there clearly were issues with the mechanical environment, metabolic causes may not need to be sought after; however, vitamin D insufficiency and sufficiency are more prevalent than thought. If the initial fixation was appropriate, then a metabolic workup is warranted. There should be careful planning of the treatment for the nonunion. All patient aspects of the nonunion must be addressed to include deformities, metabolic issues, biology, and stability of the nonunion site.

---

# References


1. Food and Drug Administration. U.S. Department of Health and Human Services. Guidance document for industry and CDRH staff for the preparation of investigational device exemptions and premarket approval applications for bone growth stimulator devices; draft; availability. [Docket No. 98D-0238]. Rockville, MD: U. S. Food and Drug Administration; 1998.

2. Frölke JP, Patka P., Patka P. Definition and classification of fracture non-unions. Injury. 2007;38(Suppl 21):S19–S22. Erratum in: Injury. 2007;38(10)1224.

3. Bhandari M, Fong K, Sprague S, Williams D, Petrisor B. Variability in the definition and perceived causes of delayed unions and nonunions. J Bone Joint Surg Am. 2012;94(15):e1091–6.

4. Antonova E, Le TK, Burge R, Mershon J. Tibia shaft fractures: costly burden of nonunions. BMC. 213:14:42.

5. Kanakaris NK, Giannoudis PV. The health economics of the treatment of long-bone non-unions. Injury. 2007;38(Suppl 2):S77–84.

6. Hak DJ, Fitzpatrick D, Bishop JA, Marsh JL, Tilp S, Schnettler R, et al. Delayed union and nonunions: epidemiology, clinical issues, and financial aspects. Injury. 2014;45(Suppl2):S3–7.

7. Brinker MR, Hanus BD, Sen M, O'Connor P. The devastating effects of tibial nonunion on health-related quality of life. J Bone Joint Surg Am. 2013;95(24):2170–6.

8. Schottel PC, O'Connor P, Brinker MR. Time trade-off as a measure of health-related quality of life: long bone nonunions have a devastating impact. J Bone Joint Surg Am. 2015;97(17):1406–10.

9. Wichlas F, Tsitsilonis S, Disch AC, Haas NP, Hartmann C, Graef F, Schwabe P. Long-term functional outcome and quality of life after successful surgical treatment of tibial nonunions. Int Orthop. 2015;39(3):521–5.

10. Bishop JA, Palanca AA, Bellino MJ, Lowenberg DW. Assessment of compromised fracture healing. J Amer Acad Orthop Surg. 2012;20(5):273–82.

11. Giannoudis PV, Einhorn TA, marsh D. Fracture healing: The diamond concept. Injury. 2007;38(Suppl 4):S3-S6400-7.

12. Blair JA, Stoops TK, Doarn MC, Kemper D, Erdogan M, Griffing R, Sagi HC. Infection and non-union following fasciotomy for compartment syndrome associated with tibia fractures: a matched cohort comparison. J Orthop Trauma. 2016;30(7):392–6.

13. Brinker MR, O'Connor DP, Monla YT, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trauma. 2007;21(8):557–70.

14. Chachan S, Tudu B, Sahu B. Ultrasound monitoring of fracture healing: is this the end of radiography in fracture follow-ups? J Ortho Trauma. 2015;29(3):e133–8.

15. Cook, GE, Bates BD, Tornetta P, McKee MD, Morshed, Slobogean GP, Schemitsch EH. Assessment of fracture repair. J Orthop Trauma. 2015;29(Suppl 12):S57-61.

16. Cox G, Einhorn TA, Tzioupis C, Giannoudis PV. Bone-turnover markers in fracture healing. J Bone Joint Surg Br. 2010;92(3):329–34.

17. Dimitriou R, Kanakaris N, Soucacos PN, Giannoudis PV. Genetic predisposition to non-union: evidence today. Injury. 2013;44(Suppl 1):S50–3.

18. Fischer C, Doll J, Tanner M, Bruckner T, Zimmermann Helbig L, et al. Quantification of TGF-B1, PDGF and IGF-1 cytokine expression after fracture treatment vs. non-union therapy via masquelet. Injury. 2016;47(2):342–9.

19. Frank T, Osterhoff G, Sprague S, Garibaldi A, Bhandari M. Slobogean GP; FAITH Investigators. The radiographic union score for hip (RUSH) identifies radiographic nonunion of femoral neck fractures. Clin Orthop Relat Res. 2016;474(6):1396–404.
[PubMedCentral]

20. Hankenson KD, Zimmerman G, Marcucio R. Biological perspectives of delayed fracture healing. Injury. 2014;45(Suppl 2):S8–15.
[PubMedCentral]

21. Hernigou J, Schuind F. Smoking as a predictor of negative outcome in diaphyseal fracture healing. Int Orthop. 2013;37(5):883–7.
[PubMedCentral]

22. Kurdy NM. Serology of abnormal fracture healing: the role of PIIINP, PICP, and BsALP. J Orthop Trauma. 2000;14(1):48–53.

23. Malik MH, Harwood P, Diggle P, Khan SA. Factors affecting rates of infection and nonunion in intramedullary nailing. J Bone Joint Surg Am. 2004;86(4):556–60.

24. Murray IR, Foster CJ, Eros A, Robinson CM. Risk factors for nonunion after nonoperative treatment of displaced midshaft fractures of the clavicle. J Bone Joint Surg Am. 2013;95(13):1153–8.

25. Niikura T, Lee SY, Sakai Y, Nishida K, Kuroda R, Kurosaka M. Causative factors of fracture nonunion: the proportions of mechanical, biological, patient-dependent, and patient-independent factors. J Ortho Sci. 2014;19(1):120–4.

26. O'Halloran K, Coale M, Costales T, Zerhusen T Jr, Castillo RC, Nascone JW, O'Toole RV. Will my tibial fracture heal? Predicting nonunion at the time of definitive fixation based on commonly available variables. Clin Orthop Relat Res. 2016;474(6):1385–95. [PubMedCentral]

27. Pountos I, Georgouli T, Pneumaticos S, Giannoudis PV. Fracture non-union: can biomarkers predict outcome? Injury. 2013;44(12):1725–32.

28. Santolini E, West R, Giannoudis P. Risk factors for long bone fracture non-union: a stratification approach based on the level of existing scientific evidence. Injury. 2015;46(Suppl 8):S8–19.

29. Westgeest J, Kin BSC, Weber D, Dulai SK, Bergman JW, Buckley R, Beaupre LA. Factors associated with development of nonunion or delayed healing after an open long bone fracture: a prospective cohort study of 736 subjects. J Orthop Trauma. 2016;30(3):149–55.

30. Zimmermann G, Muller U, Wentzensen A. The value of laboratory and imaging studies in the evaluation of long-bone non-unions. Injury. 2007;38(Suppl 2):S33–7.

31. Harwood PJ, Ferguson DO. An update on fracture healing and non-union. Orthop Trauma. 2015;29(4):228–42.

32. Calori, GM, Albisetti W, Agus A, Iori S, Tagliabue L. Risk factors contributing to fracture non-unions. Injury. 2007;38S:S11–S18. THERE IS NO CITATION 19.

33. Lin CA, Swiontkowski M, Bhandari M, Walter SD, Schemitsch EH, Sanders D, Tornetta P 3rd. Reaming does not affect functional outcomes after open and closed tibial shaft fractures: the results of a randomized controlled trial. J Orthop Trauma. 2016;30(3):142–8. [PubMedCentral]

34. Obremskey W, Molina C, Collinge C, Nana A, Tornetta P 3rd, Sagi C, et al. Evidence-based quality value and safety committee orthopaedic trauma association, writing committee. Current practice in the management of open fractures among orthopaedic trauma surgeons. Part A: Initial management. A survey of orthopaedic trauma surgeons. J Orthop Trauma. 2014;28(8):e198–202.

35. Obremskey W, Molina C, Collinge C, Tornetta P III, Sagi C, Schmidt A, et al. Evidence-based quality value and safety committee orthopaedic trauma association, writing committee current practice in the management of open fractures among orthopaedic trauma surgeons: Part B: Management of segmental long bone defects. A survey of Orthopaedic Trauma Association members. J Orthop Trauma. 2014;28(8):e203–7.

36. Dye NB, Vagts C, Manson TT, O'Toole RV. Estimation of tibial shaft defect volume using standard radiographs: development and validation of a novel technique. Injury. 2015;46(2):299–307.

37. Hildebrand F, Griensven MV, Huber-Lang, M, Flohe SB, Andruszkow H, Marzi I, Pape HC; Trauma research network for the German Society of Trauma, DGU. Is there an impact of concomitant injuries and timing of fixation of major fractures on fracture healing? A focused review of clinical and experimental evidence. J Orthop Trauma. 2016;30(3):104–112.

38. Marsell R, Einhorn TA. The biology of fracture healing. Injury. 2011;42(6):551–5. [PubMedCentral]

39. Tzioupis C, Giannoudis PV. Prevalence of long-bone non-unions. Injury. 2007;38(Suppl 2):S3-9. Erratum in Injury. 2007;38(10):1224.

40. Borrelli J Jr, Pape C, Hak D, Hsu J, Lin S, Giannoudis P, Lane J. Physiological challenges of bone repair. J Orthop Trauma. 2012;26(12):708–11.

41. Loder RT. The influence of diabetes mellitus on the healing of closed fractures. Clin Orthop Relat Res. 1988;232:210–6.

42. Liao CC, Lin CS, Shih CC, Yeh CC, Chang YC, Lee YW, Chen TL. Increased risk of fracture and postfracture adverse events in patients with diabetes: two nationwide population-based retrospective cohort studies. Diab Care. 2014;37(8):2246–52.

43. Wukich DK, Joseph A, Ryan M, Ramirez C, Irrgang J. Outcomes of ankle fractures in patients with uncomplicated versus complicated diabetes. Foot Ankle Int. 2011;32(2):120–30.

44. Sprague S, Petrisor B, Scott T, Devji T, Phillips M, Spurr H, et al. What is the role of vitamin D supplementation in acute fracture patients? A systematic review and meta-analysis of the prevalence of hypovitaminosis D and supplementation efficacy. J Orthop Trauma. 2016;30(2):53–63.

45. Sprague S, Bhandari M, Devji T, Scott T, Petrisor B, McKay P, Slobogean GP. Prescription of vitamin D to fracture patients: a lack of consensus and evidence. J Orthop Trauma. 2016;30(2):e64–9.

46. Stephens BF, Murphy A, Mihalko WM. The effects of nutritional deficiencies, smoking, and systemic disease on orthopaedic outcomes. J Bone Joint Surg Am. 2013;95(23):2153–7.

47. Childs BR, Andres BA, Vallier HA. Economic benefit of calcium and vitamin D supplementation: does it outweigh the cost of nonunions? J Orthop Trauma. 2016;30(8):e285–8.

48. Hobby B, Lee MA. Managing atrophic nonunion in the geriatric population: incidence, distribution, and causes. Orthop Clin North Am. 2013;44(2):251–6.

49. Kwong FNK, Harris MB. Recent developments in the biology of fracture repair. J Am Acad Orthop Surg. 2008;16(11):619–25.

50. Giannoudis PV, Hak D, Sanders D, Donohoe E, Tosounidis T, Bahney C. Inflammation, bone healing, and anti-inflammatory drugs: an update. J Orthop Trauma. 2015;29(Suppl):S6–9.

51. Kurmis AP, Kurmis TP, O'Brien JX, Dalen T. The effect of nonsteroidal anti-inflammatory drug administration on acute phase fracture-healing: a review. J Bone Joint Surg Am. 2012;94(9):815–23.

52. Lee JJ, Patel R, Biermann S, Dougherty PJ. The musculoskeletal effects of cigarette smoking. J Bone Joint Surg Am. 2013;95(9):850–9.

53. Patel RA, Wilson RF, Patel PA, Palmer RM. The effect of smoking on bone healing. Bone Joint Res. 2013;2(6):102–11.
[PubMedCentral]

54. Scolaro JA, Schenker ML, Yannascoli S, Baldwin K, Mehta S, Ahn J. Cigarette smoking increases complications following fracture. J Bone Joint Surg Am. 2014;96(8):674–81.

55. Matuszewski PE, Boulton CL, O'Toole RV. Orthopaedic trauma patients and smoking: knowledge deficits and interest in quitting. Injury. 2016;47(6):1206–11.

56. Dijkman BG, Sprague S, Schemitsch EH, Bhandari M. When is a fracture healed? Radiographic and clinical criteria revisited. J Orthop Trauma. 2010;24(Suppl 3):S76–80.

57. Bhandari M, Chiavaras M, Ayeni O, Chakraverrty R, Parasu N, Choudur H, Bains S, Sprague S, Petrisor B. Assessment of radiographic fracture healing in patients with operatively treated femoral neck fractures. J Orthop Trauma. 2013;27(9):e213–9.

58. Koolstra BW, Dijkman BG, Sprague S, Schemitsch EH, Bhandari M. The radiographic union scale in tibia fractures: reliability and validity. J Orthop Trauma. 2010;24(Suppl 1):S81–6.

59. Whelan DB, Bhandari M, Stephen D, Kreder H, McKee MD, Zdero R, Schemitsch EH. Development of the radiographic union score for tibial fractures for the assessment of tibial fracture healing after intramedullary fixation. J Trauma. 2010;68(3):629–32.

60. Chiavaras MM, Bains S, Choudur H, Parasu N, Jacobson J, Ayeni O, et al. The radiographic union score for hip (RUSH): the use of a checklist to evaluate hip fracture healing improves agreement between radiologists and orthopaedic surgeons. Skeletal Radiol. 2013;42(8):1079–88.

61. Varghese VD, Boopalan PR, Titus VTK, Oommen AT, Jepegnanam TS. Indices affecting outcome of neglected femoral neck fractures after valgus intertrochanteric osteotomy. J Orthop Trauma. 2014;28(7):410–6.

62. Bhattacharyya T, Bouchard KA, Phadke A, Meigs JB, Kassarjian A, Salamipour H. The accuracy of computed tomography for the diagnosis of tibial nonunion. J Bone Joint Surg Am. 2006;88(4):692–7.

63. Augat P, Morgan EF, Lujan TJ, MacGillivray TJ, Cheung WH. Imaging techniques for the assessment of fracture repair. Injury. 2014;45(Suppl 2):S16–22.

64. Moed BR, Subramanian S, van Holsbeeck M, Watson JT, Cramer KE, Karges DE, et al. Ultrasound for the diagnosis of tibial fracture healing after static interlocked nailing without reaming: clinical results. J Orthop Trauma. 1998;12(3):206–13.

65. Gelalis ID, Politis AN, Arnaoutoglou CM, Korompilias AV, Pakos EE, Vekris MD, et al.

Diagnostic and treatment modalities in nonunions of the femoral shaft. A review. Injury. 2012;43(7):980–8.

66. Stucken C, Olszewski DC, Creevy WR, Murakami AM, Tornetta P. Preoperative diagnosis of infection in patients with nonunions. J Bone Joint Surg Am. 2013;95(15):1409–12.

67. Termaat MF, Raijmakers PG, Scholten HJ, Bakker FC, Patka P, Haarman HJ. The accuracy of diagnostic imaging for the assessmemt of chronic osteomyelitis: a systemic review and meta-analysis. J Bone Joint Surg Am. 2005;87(11):2464–71.

68. Stoffel K, Engler H, Kuster M, Riesen W. Changes in biochemical markers after lower limb fractures. Clin Chem. 2007;53(1):131–4.

69. Weber BG, Cech O. Pseudarthrosis, pathology, biomechanics, therapy, results. Bern: Hans Huber; 1976.

70. Panagiotis M. Classification of non-union. Injury. 2005;36(Suppl 4):S30–7.

71. Atkins RM. Principles of management of septic non-union of fracture. Injury. 2007;38(Suppl 2):S23–32.

72. Biasibetti A, Aloj D, Di Gregorio G, Masse A, Salomone C. Mechanical and biological treatment of long-bone non-unions. Injury. 2005;36(Suppl 4):S45–50.

73. Calori GM, Phillips M, Jeetle S, Tagliabue L, Giannoudis PV. Classification of non-union: need for new scoring system? Injury. 2008;39(Suppl 2):S59–63.

74. Kanellopoulos AD, Soucacos PN. Management of nonunion with distraction osteogenesis. Injury. 2006;37(Suppl 1):S51–5.

75. Kocaoglu M, Eralp L, Sen C, Cakmak M, Dincyurek H, Goksan SB. Management of stiff hypertrophic nonunions by distraction osteogenesis. J Orthop Trauma. 2003;17(8):543–8.

76. Schoenleber SJ, Hutson JJ Jr. Treatment of hypertrophic distal tibia nonunion and early malunion with callus distraction. Foot Ankle Int. 2015;36(4):400–7.

77. Calori GM, Mazza E, Colombo M, Ripamonti C, Tagliabue L. Treatment of long bone non-unions with polytherapy: indications and clinical results. Injury. 2011;42(6):587–90.

78. Egol KA, Nauth A, Lee M, Pape HC, Watson JT, Borrelli J Jr. Bone grafting: sourcing, timing, strategies, and alternatives. J Orthop Trauma. 2015;29(Suppl 12):S10–4.

79. Struijs PAA, Poolman RW, Bhandari M. Infected nonunion of the long bones. J Orthop Trauma. 2007;21(7):507–11.

80. Amorosa LF, Buirs LD, Bexkens R, Wellman DS, Kloen P, Lorich DG, Helfet DL. A single-stage treatment protocol for presumptive aseptic diaphyseal nonunions: a review of outcomes. J Orthop Trauma. 2013;27(10):582–6.

81. Scolaro JA, Mehta S. Stabilisation of infected peri-articular nonunions with an antibiotic impregnated cement coated loaking plate: technique and indications. Injury. 2016;47(6):1353–6.

82.

Mahendra A, Maclean AD. Available biological treatments for complex non-unions. Injury. 2007;38(Suppl 4):S7–12.

83. Rosset P, Deschaseaux F, Layrolle P. Cell therapy for bone repair. Orthop Traumatol Surg Res. 2014;100(Suppl 1):S107–S112. Review.

84. Sen MK, Miclau T. Autologous iliac crest bone graft: should it still be the gold standard for treating nonunions? Injury. 2007;38(Suppl; 1):S75–S80.

85. Dimitriou R, Mataliotakis GI, Angoules AG, Kanakaris NK, Giannoudis PV. Complications following autologous bone graft harvesting from the iliac crest and using the RIA: a systematic review. Injury. 2011;42(Suppl 2):S3–15.

86. Flierl MA, Smith WR, Mauffrey C, Irgit K, Williams AE, Ross E, et al. Outcomes and complication rates of different bone grafting modalities in long bone fracture nonunions: a retrospective cohort study in 182 patients. J Ortho Surg Res. 2013;8:33.

87. Loeffler BJ, Kellam JF, Sims SH, Bosse MJ. Prospective observational study of donor-site morbidity following anterior iliac crest bone-grafting in orthopaedic trauma reconstruction patients. J Bone Joint Surg Am. 2012;94(18):1649–54.

88. Sagi HC, Young ML, Gerstenfeld L, Einhorn TA, Tornetta P. Qualitative and quantitative differences between bone graft obtained from the medullary canal (with a reamer/irrigator/aspirator) and the iliac crest of the same patient. J Bone Joint Surg Am. 2012;94(23):2128–35.

89. Dawson J, Kiner D, Gardner W II, Swafford R, Nowotarski PJ. The reamer- irrigator-aspirator (RIA) as a device for harvesting bone graft compared with iliac crest bone graft (ICBG): Union rates and complications. 2014;28(10):586–90.

90. Allsopp BJ, Hunter-Smith DJ, Rozen WM. Vascularized versus nonvascularized bone grafts: what is the evidence? Clin Orthop Relat Res. 2016;474(5):1319–27. [PubMedCentral]

91. Soucacos PN, Dailiana Z, Beris AE, Johnson EO. Vascularized bone grafts for the management of non-union. Injury. 2006;37(Suppl 1):S41–50.

92. Braly HL, O'Connor DP, Brinker MR. Percutaneous autologous bone marrow injection in the treatment of distal meta-diaphyseal tibial nonunions and delayed unions. J Orthop Trauma. 2013;27(9):527–34.

93. Gómez-Barrena E, Rosset P, Lozano D, Stanovici J, Ermthaller C, Gerbhard F. Bone fracture healing: cell therapy in delayed unions and nonunions. Bone. 2015;70:93–101.

94. Homma Y, Zimmermann G, Hernigou P. Cellular therapies for the treatment of non-union: the past, present and future. Injury. 2013;44(Suppl 1):S46–9.

95. Tseng SS, Lee MA, Reddi AH. Nonunions and the potential of stem cells in fracture healing. J Bone Joint Surg Am. 2008;90(Suppl 1):92–8.

96. De Biase P, Capanna R. Clinical applications of BMPs. Injury. 2005;36(Suppl 3):S43–6.

97. Giannoudis PV, Dinopoulos HT. BMPs: options, indications, and effectiveness. J Orthop Trauma. 2010;24(Suppl 3):S9–16.

98. Obert L, Deschaseaux F, Garbuio P. Critical analysis and efficacy of BMPs in long bones non-union. Injury. 2005;36(Suppl 3):S38–42.

99. Schmidmaier G, Schwabe P, Wildemann B, Haas NP. Use of bone morphogenetic proteins for treatment of non-unions and future perspectives. Injury. 2007;38(Suppl 4):S35–41.

100. Friedlaender GE, Perry CR, Cole JD, Cook SD, Cierny G, Muschler GF, et al. Osteogenic protein-1 (Bone morphogenetic protein-7) in the treatement of tibial nonunions. J Bone Joint Surg Am. 2001;83-A (Suppl 1 Pt 2):S151–S158.

101. Dimitriou R, Dahabreh Z, Katsoulis E, Matthews SJ, Branfoot T, Giannoudis PV. Application of recombinant BMP-7 on persistent upper and lower limb non-unions. Injury. 2005;36(Suppl 4):S51–9.

102. Ronga M, Baldo F, Zappala G. Cherubino P; BMP-7 Italian Observational Study (BIOS) Group. Recombinant human bone morphogenetic protein-7 for treatment of long bone non-union: an observational, retrospective, non-randomized study of 105 patients. Injury. 2006;37(Suppl 3):S51–6.

103. Giannoudis PV, Kanakaris NK, Dimitriou R, Gill I, Kolimarala V, Montgomery RJ. The synergistic effect of autograft and BMP-7 in the treatment of atrophic nonunions. Clin Orthop Relat Res. 2009;467(12):3239–48.
[PubMedCentral]

104. Morison Z, Vicente M, Schemitsch EH, McKee MD. The treatment of atrophic, recalcitrant long-bone nonunion in the upper extremity with human recombinant bone morphogenetic protein-7 (rhBMP-7) and plate fixation: a retrospective review. Injury. 2016;47(2):356–63.

105. Jones AL, Bucholz RW, Bosse MJ, Mirza SK, Lyon TR, Webb LX, et al. BMP-2 evaluation in surgery for Tibial Trauma-Allograft (BESTT-ALL) Study Group. Recombinant human BMP-2 and allograft compared with autogenous bone graft for reconstruction of diaphyseal tibial fractures with cortical defects. A randomized, controlled trial. J Bone Joint Surg Am. 2006;88(7):1431–41.

106. Aro HT, Govender S, Pate AD, Hernigou P, de Gregorio AP, Popescu GI, et al. Recombinant human bone morphogenetic protein-2: a randomized trial in open tibial fractures treated with reamed nail fixation. J Bone J Surg Am. 2011;93(9):801–8.

107. Kanakaris NK, Paliobeis C, Manidakis N, Giannoudis PV. Biological enhancement of tibial diaphyseal aseptic non-unions: the efficacy of autologous bone grafting, BMPs and reaming by-products. Injury. 2007;38(Suppl 2):S65–S75. Erratum in Injuryl;207;28(10):1224.

108. Brinker MR, O'Connor DP. Management of aseptic tibial and femoral diaphyseal nonunions without bony defects. Orthop Clin N Am. 2016;47(1):67–75.

109. Crowley DJ, Kanakaris NK, Giannoudis PV. Femoral diaphyseal aseptic non-unions: is there an ideal method of treatment? Injury. 2007;38(Suppl 2):S55–63.

110. Swanson EA, Garrard EC, Bernstein DT, O'Connor DP, Brinker MR. Results of a systematic approach to exchange nailing for the treatment of aseptic femoral nonunions. J Orthop Trauma. 2015;29(1):21–7.

111. Swanson EA, Garrard EC, O'Connor DP, Brinker MR. Results of a systematic approach to exchange nailing for the treatment of aseptic tibial nonunions. J Orthop Trauma. 2015;29(1):28–35.

112. Feldman DS, Shin SS, Madan S, Koval KJ. Correction of tibial malunion and nonunion with six-axis analysis deformity correction using the Taylor spatial frame. J Orthop Trauma. 2003;17(8):549–54.

113. Khan Y, Laurencin CT. Fracture repair with ultrasound: clinical and cell-based evaluation. J Bone Joint Surg Am. 2008;90(Suppl 1):138–44.

114. Malizos KN, Hantes ME, Protopappas V, Papachristos A. Low intensity pulsed ultrasound for bone healing: an overview. Injury. 2006;37(Suppl 1):S56–62.

115. Heckman JD, Ryaby JP, McCabe J, Frey JJ, Kilcoyne RF. Acceleration of tibial fracture healing by non-invasive, low-intensity pulsed ultrasound. J Bone Joint Surg Am. 1994;76(1):26–34.

116. Cook SD, Ryaby JP, McCabe J, Frey JJ, Heckman JD, Kristiansen TK. Acceleration of tibia and distal radius fracture healing in patients who smoke. Clin Orth Rel Res. 1997;337:198–207.

117. Rubin C, Bolander M, Ryaby JP, Hadjiargyrou M. The use of low-intensity ultrasound to accelerate the healing of fractures. J Bone Joint Surg Am. 2001;83(2):259–70.

118. Nolte PA, van der Krans A, Patka P, Janssen IM, Ryaby JP, Albers GH. Low-intenisty pulsed ultrasound in the treatment of nonunions. J Trauma. 2001;51(4):693–702.

119. Watanabe Y, Matsushita T, Bhandari M, Zdero R, Schemitsch EH. Ultrasound for fracture healing: current evidence. J Orthop Trauma. 2010;24(Suppl 3):S56–61.

120. Zelle BA, Gollwitzer H, Zlowodzki M, Buhren V. Extracorporeal shock wave therapy: current evidence. J Orthop Trauma. 2010;24(Suppl 3):S66–70.

121. Karamitros AE, Kalentzos VN, Soucacos PN. Electric stimulation and hyperbaric oxygen therapy in the treatment fo nounions. Injury. 2006;37(Suppl 1):S63–73.

122. Mollon B, da Silva V, Busse JW, Einhorn TA, Bhandari M. Electrical stimulation for long-bone fracture-healing: a meta-analysis of randomized controlled trials. J Bone Joint Surg Am. 2008;90(11):2322–30.

123. Goldstein C, Sprague S, Petrisor BA. Electrical stimulation for fracture healing: clinical evidence. J Orthop Trauma. 2010;24(Suppl 3):S62–5.

124. Adie S, Harris IA, Naylor JM, Rae H, Dao A, Yong S, Ying V. Pulsed electromagnetic field stimulation for acute tibial shaft fractures. J Bone Joint Surg Am. 2011;93(7):1569–76.

125. Danoff JR, Aurégan JC, Coyle RM, Burky RE, Rosenwasser MP. Augmentation of fracture healing using soft callus. J Orthop Trauma. 2016;30(3):113–8.

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_2

# 2. Fracture Healing

Saam Morshed[1] and Anthony Ding[1]

(1) Zuckerberg San Francisco General Hospital, Orthopedic Trauma Institute, Department of Orthopedic Surgery, University of California San Francisco, 2550 23rd Street, Building 9, 2nd Floor, San Francisco, CA 94110, USA

**Saam Morshed ( MD, PhD, MPH)**
**Email:** saam.morshed@ucsf.edu

**Keywords** Fracture healing – Nonunion – Delayed union

## 2.1 Introduction

Musculoskeletal injury is one of the leading causes of disability and dysfunction worldwide. In the USA alone, the burden of fracture care in an aging population is projected to exceed $25 billion in costs [1–3]. Achieving fracture union is paramount to patient recovery, return to activity, and quality of life following injury. While the majority of fractures will heal uneventfully, a small but significant number will demonstrate impaired healing [4]. When fractures fail to heal, they place a substantial burden on the patient and on the healthcare system [5–7]. Brinker and O'Connor [5] showed that fracture nonunion is more burdensome than many chronic medical conditions, including chronic obstructive pulmonary disease and congestive heart failure.

Fracture healing is a complex, highly orchestrated regenerative process to restore skeletal integrity. The response following injury involves tightly coordinated temporal and spatial interactions among cytokines, growth

factors, progenitor cells, and adjacent tissues. The intricacy of fracture healing incorporates multiple pathways and interdependent processes; disruption in key steps can delay or terminate healing altogether.

The causative factors underlying nonunion are often multifactorial. Injury patterns, patient factors, and even interventions all have substantial implications toward successful repair. A thorough understanding of the normal healing process, and where it goes awry, is essential to the diagnostic and therapeutic approach in treating nonunions.

The purpose of this chapter is to provide the conceptual framework for understanding fracture healing and its modulating factors in the context of nonunion management. The first part discusses the physiology of fracture healing—its biology, mechanics, and assessment. The second part focuses on modulators of healing—patient-related factors, comorbidities, injury patterns, surgical intervention, and biologic augmentation—that may promote or impair fracture union.

## 2.2 Physiology of Fracture Healing

Despite its complexity, fracture healing is driven by fundamental principles. Fractures all require a viable pool of progenitor cells, an osteoconductive scaffold (extracellular matrix), signaling molecules and their receptors, a vascular supply, and a suitable mechanical milieu to heal. Failure in one or more of these domains impairs successful healing [8–12]. The ability to achieve fracture healing hinges on the interdependency between the mechanics and the biology at the fracture site. The mechanical environment dictates the biologic response to skeletal injury, and there must be sufficient stability to promote bony healing. As healing progresses, extracellular matrix is laid across the fracture site, which lends further mechanical support to the fracture.

### 2.2.1 Biology of Fracture Healing

The healing response depends on the temporal and spatial interactions among four main tissue types: cortical bone, bone marrow , periosteum, and surrounding soft tissue. Ossification, the process of bone tissue formation both in normal development and in skeletal injury, is a key process in fracture healing. Endochondral ossification utilizes a cartilage scaffold to form bone,

whereas intramembranous ossification forms bone without a cartilage scaffold.

There are two main pathways of fracture healing: *direct healing* and *indirect healing*. Direct, or primary, healing allows for direct remodeling of lamellar bone. It involves only intramembranous ossification in the formation of bone. Indirect, or secondary, healing relies on forming a cartilage callus scaffold, through which bone forms and remodels into its mature lamellar structure. Whether a fracture heals by direct or indirect means is determined early by its biologic and physical environment [13, 14]. Initial stability influences the inflammatory response following injury and can thus influence the mode of repair. Rigid stability follows a direct healing pathway, whereas relative stability leads to indirect healing. Additionally, as with most biologic phenomenon, fracture healing represents a spectrum with varying degrees of direct and indirect healing happening simultaneously, depending on the anatomical location and the mechanical environment.

#### *2.2.1.1 Direct Fracture Healing*

Direct or primary healing regenerates lamellar bone across the fracture without a cartilage scaffold. To do so, several conditions must exist. First, the cortical bone must be anatomically reduced and apposed. Second, the fragments must be rigidly fixed, allowing minimal interfragmentary strain (<5%) [15–19]. Gaps must be small, less than 1 mm [17]. Because these conditions usually do not occur naturally, direct healing is primarily achieved by operative fixation [9]. These fixation methods include compression plating, lag screw fixation (Fig. 2.1), and multiplanar external fixation. Failure to meet the above conditions can impair the healing process. Achieving rigid stability in the setting of comminution or a large fracture gap prohibits callus formation across the fracture site. Failing to respect the biology around the fracture site through extensive dissection and excessive soft tissue stripping likewise discourages healing (Fig. 2.2).


***Fig. 2.1*** Primary healing with absolute stability. The patient is a 26-year-old woman who was struck by a motor vehicle and sustained a Grade III open right distal tibia fracture. **a** Injury radiographs. **b**, **c** Initial irrigation and debridement of the fracture site, spanning external fixation, and lag screw fixation. **d**, **e** Definitive fixation with lag screw fixation, neutralization plate. **f**, **g** 3-month follow-up, showing progressive healing of tibia without callus formation and healing of fibula with callus. **h**, **i** 1-year follow-up showing complete healing of tibia and fibula


(a)
(b)
(c)
(d)
(e)
(f)

***Fig. 2.2*** Impaired healing with absolute stability. The patient is a 41-year-old man who sustained an open right distal tibia fracture that was initially treated with open reduction internal fixation at an outside facility. **a**, **b** 6-month postoperative radiographs demonstrate persistent fracture lines with little evidence of healing as well as hardware failure, consistent with nonunion. **c**, **d** Nonunion repair with removal of hardware and intramedullary nailing. **e**, **f** 6-month postoperative radiographs with healing of fracture

Contact healing occurs in the absence of gapping, where cortices are directly apposed. “Cutting cones” lay down new osteons longitudinally across the fracture site. Osteoclasts form the tip of the cone, resorb injured bone, and create new Haversian canals (Fig. 2.3) [8]. New blood vessels, branching from endosteal and periosteal circulation, penetrate the canals and deliver osteoblastic precursors. Osteoblasts form the end of the cutting cone unit, laying down new bone that will eventually mature into its lamellar structure (Fig. 2.4) [8, 9, 13]. There is limited contribution from the surrounding periosteum and soft tissues.

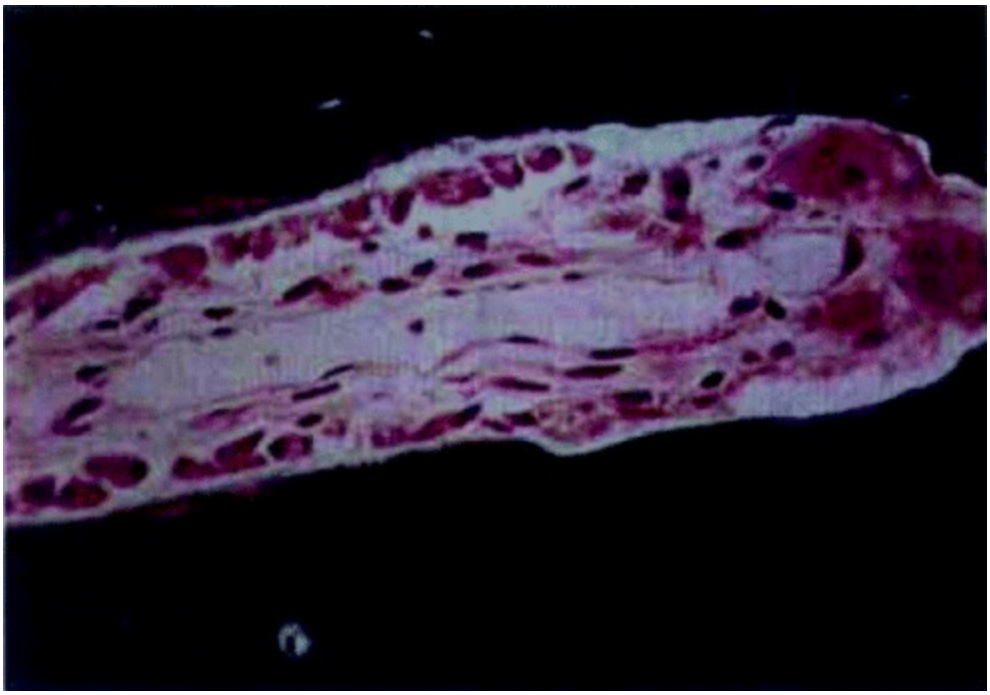


***Fig. 2.3*** Cutting cones. Low power photomicrograph of a “cutting cone” in direct bone healing and remodeling. Multinucleated osteoclasts (*right)* form the leading edge of the cone, followed by osteoblasts (*left*) forming new bone. From Einhorn [8], with permission

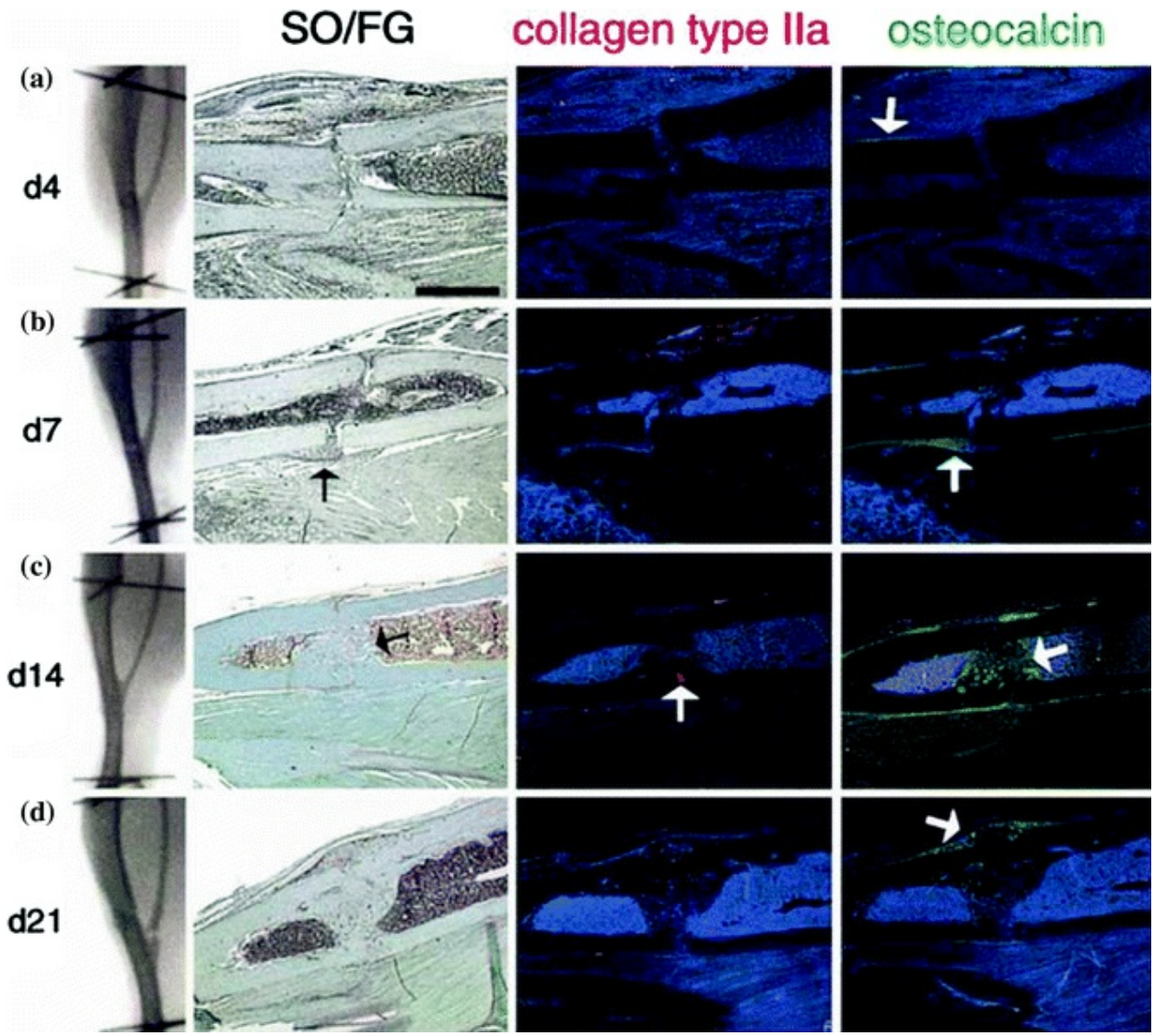



***Fig. 2.4*** Healing of stabilized fracture. Progressive healing of a stabilized tibia fracture in a mouse model demonstrates no callus formation on serial radiographs (day 4 through day 21) or on histological staining. In the presence of new bone formation (*green*), there is minimal staining for collagen type IIa expression (*red*), a marker of chondrogenesis. (*SO/FG* Safranin O/Fast Green stain). From Thompson et al. [13], with permission

Gap healing occurs with small gaps less than 0.8–1 mm under similar rigid conditions. Unlike in contact healing, hematoma initially fills the gap. It is quickly replaced with woven bone in the first 1–2 weeks. Woven bone is then replaced by lamellar repair bone, though this interposed bone is oriented perpendicular to the long bone axis. While stronger than cartilage, this bone bridge is biomechanically weaker at its interface with the normal bone due to its orthogonal orientation. At 6–8 weeks, the repair bone undergoes

secondary remodeling. Cutting cones from the neighboring cortices traverse and replace the repaired bone to reconstitute the canalicular system, recreate the longitudinal lamellar structure, and ultimately restore skeletal integrity. No cartilaginous callus is formed [9, 20].

#### *2.2.1.2 Indirect Fracture Healing*

Indirect fracture healing regenerates bone through a cartilage callus scaffold (Fig. 2.5) [13]. It still requires a relatively stable environment, but it does not require rigid stability or anatomical reduction. Rather, micromotion, to an extent, stimulates the healing response. Indirect healing is the predominant mechanism in most fractures treated by nonoperative means. It is also achieved by interventions that allow for relative stability. These include intramedullary nailing of long bone fractures (Fig. 2.6), external fixation (Fig. 2.7), bridge plating (Fig. 2.8), and splinting, bracing, or casting.

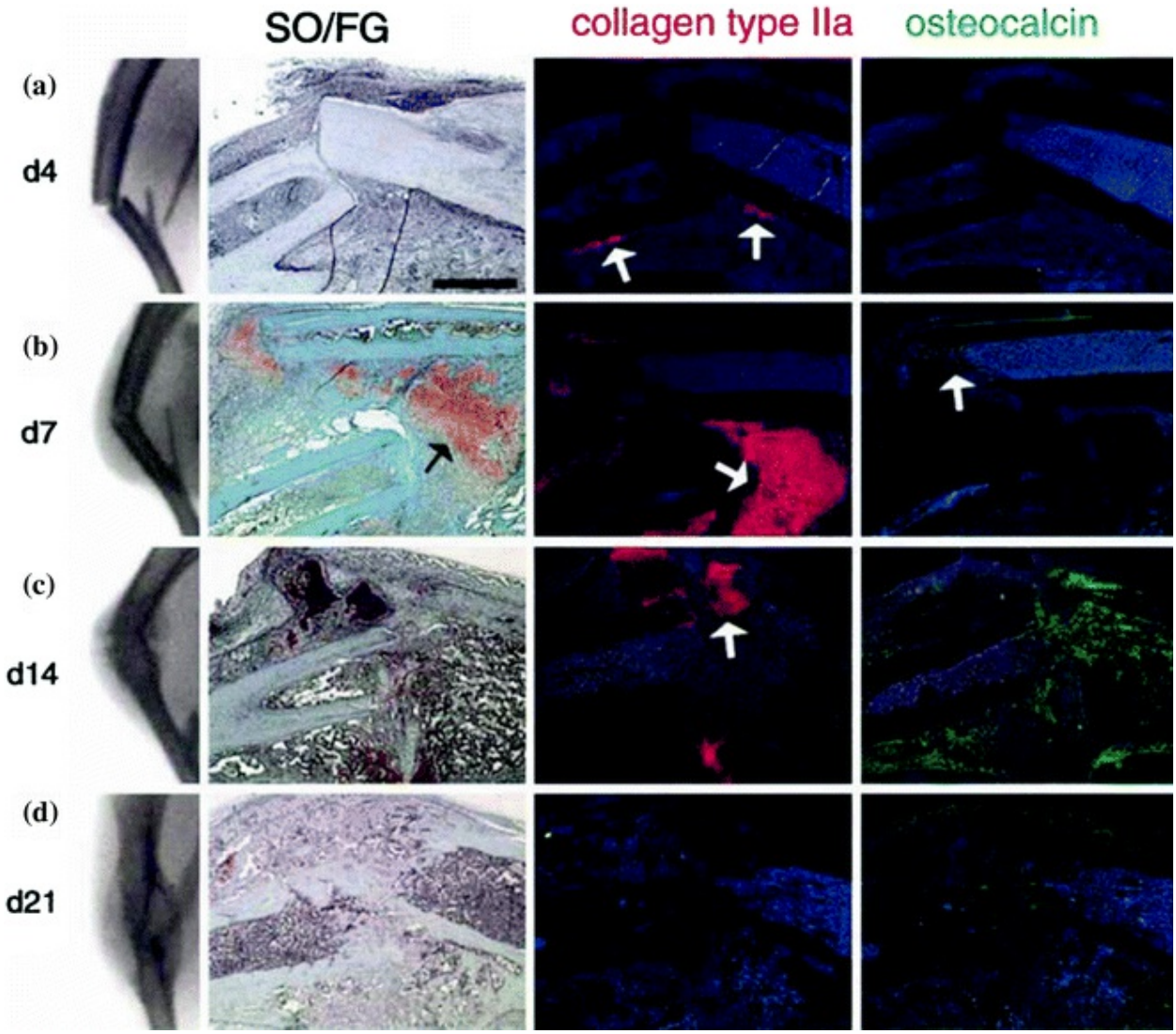



***Fig. 2.5*** Healing in unstabilized fractures. In contrast to stabilized fractures, progressive healing of a stabilized tibia fracture in a mouse model demonstrates abundant callus formation on serial radiographs and on histological staining. Safranin O/Fast Green staining demonstrates abundant collagen type IIa expression (*red*), consistent with robust chondrogenesis. From Thompson et al. [13], with permission


***Fig. 2.6*** Secondary healing with intramedullary device. The patient is a 23-year-old man who was struck by a motor vehicle at high speed and sustained right tibial and fibular shaft fractures with associated compartment syndrome. **a**, **b** Initial injury radiographs. **c**, **d** Immediate postoperative radiographs following tibia intramedullary nailing. **e**, **f** 2-month follow-up, demonstrate callus formation. **g**, **h** 9-month follow-up, with progressive callus formation and bone bridging across the tibial fracture. There is some callus at the fibula fracture ends, but no bone bridging across the fracture site. **i**, **j** 3-year follow-up, with complete healing of tibial fracture, and nonunion of fibular fracture


***Fig. 2.7*** Secondary healing with external fixator . The patient is a 51-year-old man who was struck by a vehicle and sustained a Schatzker VI left tibial plateau fracture. **a**, **b** Initial injury radiographs. **c**, **d** Definitive treatment with spanning external fixation. **e**, **f** 10-week follow-up, with interval removal of external fixator and cast application. There is bridging bone and progressive healing across the fracture

site


(a)
(b)
(c)
(d)
(e)
(f)

***Fig. 2.8*** Secondary healing with bridge plating. The patient is a 62-year-old man who was involved in a motorcycle crash. He sustained a Grade I open left tibia fracture. **a**, **b** Initial injury radiographs. **c**, **d** Initial management consisted of external fixation, followed by bridge plating across the fracture. **e**, **f** 17-month follow-up after bridge plating, demonstrating bone healing across fracture site

Three fundamental phases of indirect healing have been described [21]: inflammatory, reparative, and remodeling. Trauma initiates the acute inflammatory phase, and, through the release of mediators, cytokines, and growth factors, recruits progenitor cells responsible for initiating repair. In the reparative phase, progenitor cells lay down cartilaginous and bony callus, facilitate neoangiogenesis, and replace callus with woven bone. The remodeling phase replaces the woven bone with a mature lamellar bone structure.

## Inflammatory Phase

Injury disrupts skeletal architecture, blood vessels, periosteum, and adjacent soft tissue. The response to injury initiates the inflammatory phase, characterized by the release of cytokines and chemoattractants that together initiate healing and recruit progenitor cells.

Following injury, hematoma occupies the fracture site. Fracture hematoma serves two key functions. It provides a physical scaffold for subsequent occupation by progenitor cells, granulation tissue, and ultimately callus. Furthermore, the hematoma itself contains progenitor cells, cytokines, and growth factors that directly participate in the healing process [22, 23]. Recent studies have identified higher levels of factors and signaling molecules in fracture hematoma. These include macrophage colony-stimulating factor (M-CSF) , transforming growth factor-beta (TGF-β), and interleukins (IL), all of which have important roles in stimulating fracture healing (Table 2.1) [24–27].

***Table 2.1*** Cytokines and their roles in fracture healing

| Cytokine | Effect |
|---|---|
| IL-1 | Stimulates chemotaxis of inflammatory cells, MSCs<br>Promotes VEGF production and angiogenesis |
| IL-6 | Stimulates chemotaxis of inflammatory cells, MSCs<br>Promotes VEGF production and angiogenesis |
| | |

| | |
|---|---|
| PDGF | Released by platelets and inflammatory cells<br>Stimulates chemotaxis of inflammatory cells and osteoblasts |
| TNF-α | Recruits MSCs during inflammatory phase<br>Regulates chondrocyte apoptosis, resorption of cartilage callus<br>Regulates bone remodeling, osteoclastogenesis<br>Stimulates chondrogenic and osteogenic differentiation |
| FGF | Promote differentiation of fibroblasts, chondrocytes, myocytes, and osteoblasts |
| TGF-β | Stimulates chemotaxis and proliferation of MSCs<br>Stimulates proliferation of chondrogenic and osteogenic cells<br>Induces production of extracellular matrix |
| MMP | Degrades chondral and osseous extracellular matrix |
| VEGF | Mediates neoangiogenesis |
| angiopoietin | Regulates formation of larger vessels and branching of collateral branches from existing vessels |
| BMP | Promote osteoblast differentiation and osteogenesis<br>Upregulates extracellular matrix production<br>Stimulate VEGF production |
| M-CSF | Secreted by osteoblasts to induce osteoclast differentiation and proliferation<br>Upregulates RANK expression |
| OPG | Inhibits osteoclast differentiation and activation<br>Inhibits osteoclast-mediated resorption |
| RANKL | Stimulates osteoclastogenesis, osteoclast activation through its receptor RANK |
| Sclerostin | BMP antagonist |

*IL* interleukin; *PDGF* platelet-derived growth factor; *TNF-α* tumor necrosis factor-alpha; *FGF* fibroblast growth factor; *TGF-β* transforming growth factor-beta; *MMP* matrix metalloproteinase; *VEGF* vascular endothelial growth factor; *BMP* bone morphogenetic protein; *OPG* osteoprotegerin; *RANK* receptor-activated NF-κβ; *RANKL* receptor-activated NF-κβ ligand. From Tsiridis et al. [24] with permission

The initial inflammatory response occurs immediately after injury and lasts several days. The response is marked by infiltration of macrophages, platelets, polymorphonuclear leukocytes, and lymphocytes into the fracture site. These secrete proinflammatory cytokines including interleukins (IL-1, IL-6), platelet-derived growth factor (PDGF) , and tumor necrosis factor-alpha (TNF-α) . These factors recruit other inflammatory cells, promote angiogenesis, recruit progenitor stem cells, and induce their differentiation.

## Reparative Phase

The reparative phase is characterized by the deposition of extracellular matrix across the fracture site. It involves a tightly regulated sequence of events that ultimately stabilizes the fracture site with bridging bone. Following the inflammatory phase, this phase begins with the recruitment of mesenchymal stem cells. These progenitors differentiate into osteogenic and chondrogenic cell lines, which produce soft cartilaginous callus as a scaffold for bone healing. Vascular ingrowth prompts the maturation of the fracture callus; the soft callus undergoes mineralization, resorption, and ultimately replacement by hard callus. The end result provides a stable bridge of bone across the fracture site.

### *Recruitment of Mesenchymal Stem Cells*

The recruitment of MSCs is an essential component of fracture healing. MSCs reside throughout the body, including the periosteum, bone marrow , trabecular bone, muscle, and systemic circulation [28]. Periosteal- and bone marrow-derived MSCs were traditionally thought to be the primary sources of progenitor cells in early fracture repair [29]. However, current data suggests that other sources of MSCs, namely from muscle and systemic circulation, may also contribute to the progenitor cell population [28, 30].

Inflammation at the time of injury releases a number of chemokines, growth factors, and signals to recruit MSCs and other inflammatory cells. In the early phase, TNF-α, IL-1, and IL-6 play key roles in chemotaxis, mesenchymal stem cell (MSC) recruitment, and osteogenic and chondrogenic differentiation [14]. Peak levels of IL-1 and IL-6 are reached within the first 24 h, and then decline precipitously after 72 h. IL-1 and IL-6 contribute to chemotaxis of other inflammatory cells and of MSCs and promote angiogenesis via vascular endothelial growth factor (VEGF) production [31]. TNF-α and IL-6 promote recruitment and differentiation of muscle-derived stromal cells. TNA-α, at low concentrations, also stimulates chondrogenic and osteogenic differentiation [32–34] (see Table 2.1). In vivo injection of TNF-α accelerates fracture healing and callus mineralization [32]. Conversely, the absence of TNF-α signaling appears to delay both chondrogenic differentiation and endochondral resorption [14, 24, 34].

Emerging evidence has also supported the role of stromal cell-derived factor (SDF-1) in skeletal repair. SDF-1 is a potent chemoattractant expressed at sites of injury to recruit MSCs from both circulating and local sources.

Kitaori demonstrated that SDF-1 expression is upregulated in periosteum at the fracture site and recruits MSCs that participated in the healing process. Additionally, blocking the function of SDF-1 significantly reduced bone formation, indicating SDF-1 has a crucial role in fracture healing [35].

*Formation of Soft Cartilaginous Callus*

By this time, the fracture hematoma has been converted to granulation tissue, containing inflammatory cytokines and growth factors that stimulate MSC differentiation, proliferation, and production of extracellular matrix. The formation of cartilaginous callus marks the initial attempts at achieving fracture union. The result is a calcified cartilaginous bridge that both provides stability and creates a template for further remodeling.

Cartilaginous callus formation is driven by growth factors, chondrocytes, fibroblasts, and mechanical stimulation across the fracture site. TGF-β and IGF-1 play primary roles in this stage of chondrogenesis and endochondral bone formation, stimulating the recruitment, proliferation, and differentiation of MSCs. BMPs also promote chondrogenesis. Several days after fracture, chondrocytes derived from MSCs proliferate and synthesize collagen. Starting from the periosteum and the fractured ends, chondrogenesis progresses by appositional replacement of adjacent granulation tissue with cartilage matrix [29]. Fibroblasts produce fibrous tissue in areas with limited cartilage production. Micromotion across the fracture stimulates callus formation, and increased callus formation provides more mechanical stability to the fracture. When sufficient callus and stability have been attained, roughly 2 weeks after fracture, chondrocytes undergo hypertrophic differentiation. Proliferation ceases. Collagen synthesis is downregulated. Hypertrophic chondrocytes release vesicular stores containing calcium, proteases, and phosphatases into the surrounding matrix. As the collagen matrix is degraded, released phosphate ions bind with calcium to promote cartilage calcification. These calcium and phosphate deposits become the nidus for hydroxyapatite crystal formation [8].

At the same time, intramembranous ossification occurs in areas of low strain, beneath the periosteum, and directly adjacent to the fractured cortices. Within 24 h following injury, MSCs from the bone marrow differentiate into osteoblastic phenotypes. Proliferation and differentiation peak at day 7–10. Woven bone is formed in these regions without a cartilage scaffold.

*Revascularization and Angiogenesis*

Fracture healing begins in a relatively hypoxic environment; injury to vessels, periosteum, and soft tissue compromises local blood supply [22]. Early cartilage callus can form in this hypoxic environment. However, as healing progresses, subsequent callus remodeling and bone formation require adequate oxygen delivery. Failure to do so leads to delayed healing. Revascularization is thus critical for progressive healing and bone formation [9, 11, 12, 36–38].

Two main molecular pathways regulate this process: an angiopoietin-dependent pathway and a VEGF-dependent pathway. Angiopoietins promote formation of larger vessels and collateral vessels off existing vessels. VEGF promotes endothelial cell differentiation, proliferation, and neoangiogenesis, and it mediates the principal vascularization pathway [11, 24].

Inflammatory cytokines from early fracture healing, particularly TNF-α, induce expression of angiopoietin, allowing for early vascular ingrowth from existing periosteal vessels [9, 33]. However, the primary vascularization process is driven by VEGF. Following calcification of cartilage callus, osteoblasts and hypertrophic chondrocytes housed in callus express high levels of VEGF, stimulating neoangiogenesis into the avascular chondral matrix [36, 38, 39]. Concurrently, matrix metalloproteinases (MMPs) degrade calcified cartilage to facilitate ingrowth of new vessels [40].

### *Hard Callus Formation*

With the onset of neoangiogenesis, the next event is characterized by the transition from soft callus to hard callus: the removal of calcified cartilage and its replacement with woven bone matrix. This process is mediated by MMPs, BMPs, osteoclasts, chondroclasts, and osteoblasts [36, 40, 41].

Osteoclasts have historically been considered the key cell type in soft callus resorption. However, more recent evidence suggests that resorption is nonspecific and mediated by multiple cell lines, including osteoclasts and chondroclasts alike, and by MMP expression [40, 41]. This has been supported by findings that impaired osteoclast function does not necessarily impair healing. In an osteoclast-deficient osteopetrosis mouse model, there was no difference in callus remodeling or union rates compared with control mice [42].

Cartilage callus is removed and subsequently replaced by woven bone. Mature osteoblasts secrete osteoid, a combination of type I collagen, osteocalcin , and chondroitin sulfate. Collagen fibrils are randomly oriented,

producing an irregular structure known as woven bone [41].

**Remodeling Phase**

While woven bone provides more biomechanical stability than fibrous tissue and soft callus, its irregular and disordered structure is mechanically inferior to native cortical bone. Further remodeling is required to restore structural integrity. The final phase of fracture healing converts irregular woven bone into structured lamellar bone. The process encompasses both catabolic and anabolic mechanisms, regulated by the coordinated relationship between osteoblasts and osteoclasts. Whereas the earlier phases take place over the course of days to weeks, this final phase spans months to years after injury [9].

Remodeling is characterized by woven bone resorption followed by lamellar bone formation. Osteoclasts are multinucleated polarized cells that attach to mineralized surfaces. At sites of attachment, osteoclasts form ruffled borders, effectively increasing surface area through which lysosomal enzymes and hydrogen ions are secreted. Enzymes degrade the organic collagen components, while the acidic milieu demineralizes the bone matrix. The erosive pits left by the osteoclasts are termed “Howship’s lacuna.” Following resorption, osteoblasts form new bone within these lacunae. This process progresses along the length of hard callus, layer upon layer, replacing woven bone with lamellar bone [43, 44].

Activation and regulation of remodeling depends on intimate coupling between osteoblasts and osteoclasts. Osteoblasts initiate remodeling by producing factors to stimulate osteoclastogenesis and osteoclast function. The principle cytokines secreted by osteoblasts are M-CSF, receptor-activated NF-κβ ligand (RANKL), and osteoprotegerin (OPG) . M-CSF and RANKL are essential for osteoclast formation. Osteoblasts express RANKL on their cell membranes, whereas mononuclear osteoclast progenitors express the complementary receptor, RANK. Upon contact, RANKL interacts with RANK to induce fusion of osteoclast progenitors and thus produce mature multinucleated osteoclasts. Alternatively, osteoblasts can also secrete OPG, which acts as a decoy by binding RANK and consequently disrupts RANKL–RANK interactions. By modulating RANKL and OPG expression, osteoblasts can tightly regulate osteoclast activation. Osteoblasts express and secrete M-CSF, which induces osteoclast precursor proliferation and

differentiation. Additionally, M-CSF upregulates the expression of RANK on osteoclast precursors [43–45].

**Metaphyseal Fracture Healing**

The principles underlying fracture healing have largely been based on diaphyseal models. By comparison, the existing literature for metaphyseal healing is limited. Metaphyseal bone differs from diaphyseal bone in anatomy and biologic activity. Periosteum is thicker around the metaphysis. Blood supply is richer to the metaphysis [12]. Additionally, metaphyseal bone has a larger active bone surface area with consequently higher bone turnover rates [46].

Diaphyseal bone healing hinges on the interrelationship between biomechanics and biology. Early in the healing process, the mechanical environment determines the biologic response, whether healing will proceed by direct or indirect means. In stable situations, healing proceeds directly to osteogenesis. In unstable conditions, healing begins with chondrogenesis. The same holds true for metaphyseal healing. Under rigidly stable conditions, newly formed bone bridges the fracture gap with minimal chondrogenic tissue, similar to direct healing. Under more flexible conditions, bone intermixed with islands of chondrogenic tissue forms across the gap, analogous secondary healing. Interestingly, both situations do not generate a significant amount of external callus [47]. Whereas progenitor cells need to be recruited in diaphyseal healing, the metaphysis houses a large reservoir of precursor cells, obviating the need for a large periosteal reaction and MSC recruitment [48].

## 2.2.2 Biomechanics of Fracture Healing

The relationship between mechanics and biology is well established in skeletal physiology. Wolff's law stipulates that bone structurally adapts to its loading conditions. Likewise, biomechanics plays a central role in skeletal repair. Following injury, the mechanical environment influences the biologic healing response. This response in turn attempts to restore skeletal integrity. Understanding how biomechanical factors affect healing is therefore fundamental to fracture treatment. The existing body of literature has identified three mechanical parameters that impact fracture healing: interfragmentary strain, gap size, and hydrostatic pressure. The degree to

which these parameters affect healing, and the timing at which they are applied, will be discussed in this section.

### *2.2.2.1 Interfragmentary Strain*

Perren's strain theory proposes that "a tissue cannot be produced under strain conditions which exceed the elongation at rupture of the given tissue element" [16]. Thus, bone can only form in low strain environments, while fibrous tissue can form in high strain environments. In stable fractures, a low strain environment allows for primary osteogenesis across the fracture gap. However, in unstable fractures, high strains preclude direct bone formation. Instead, precursor tissues must first bridge the gap, providing adequate mechanical stability for osteogenesis to ultimately occur. Such is the case with endochondral bone formation. Cartilage callus first bridges the gap and provides provisional stability across the fracture. When sufficient stability has been attained, the cartilage callus can then undergo calcification, and woven bone can replace the chondral matrix. If strain is still too high, more callus is produced, increasing its diameter and effectively increasing its strength. If strain still remains too high, bone bridging may not occur and a fibrous nonunion may develop instead.

The relationship between strain and tissue differentiation correlates with both histomorphometric and finite element analyses [15, 49, 50]. In models of indirect healing, intramembranous bone formation occurs at the periosteum and directly adjacent to the cortex, areas characterized by low strain. Cartilaginous callus developed between the fractured ends, in areas of high strain. Increasing the mechanical stress and strain, by early loading or delayed stabilization, impairs bone bridging and delayed healing across the fracture [51, 52]. Histological analysis in these animal models of delayed stabilization demonstrated higher proportions of cartilage and fibrous tissue in the fracture site compared to fractures that were stabilized early (Fig. 2.9) [53]. Similarly, Augat demonstrated in a sheep model that higher gap sizes and higher strains led to lower amounts of bone formation and higher proportions of connective tissue and fibrocartilage formation across the fracture (Fig. 2.10) [49].

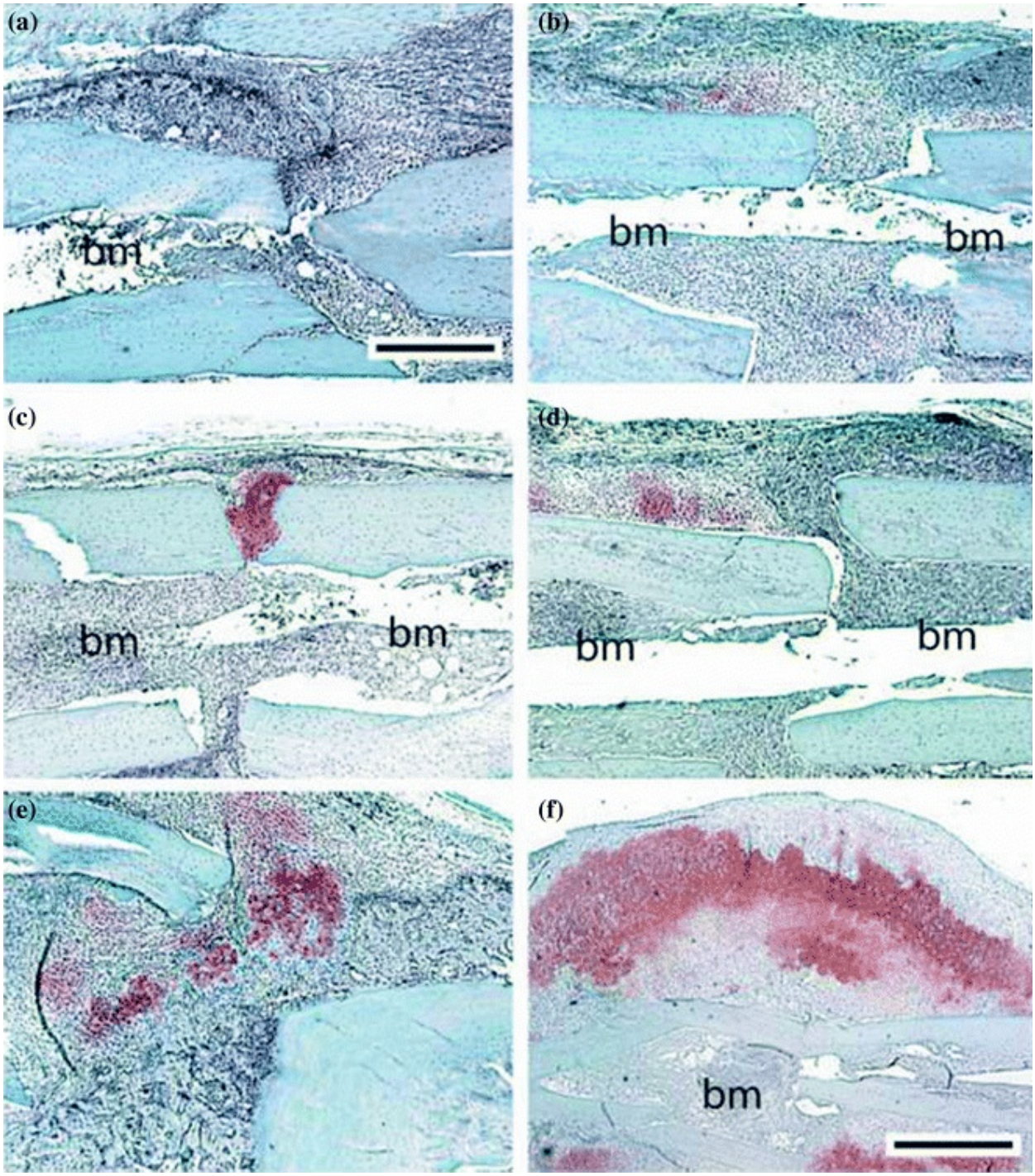



***Fig. 2.9*** Histological findings in impaired healing. Nonstabilized fractures (**e**) demonstrate increased cartilage formation compared to stabilized fractures (**d**). From Miclau et al. [53] with permission

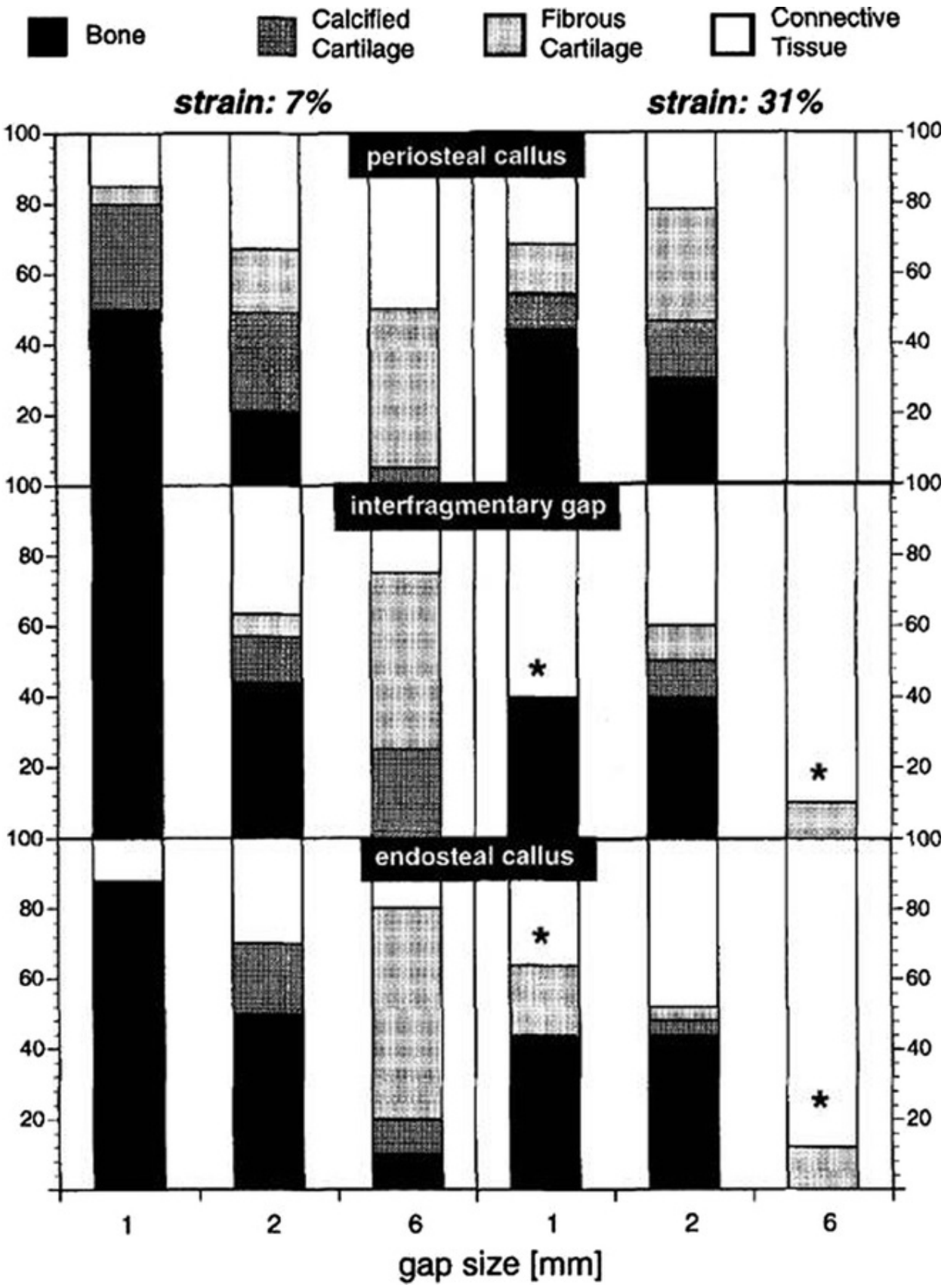

Bone
Calcified Cartilage
Fibrous Cartilage
Connective Tissue
strain: 7%
strain: 31%
periosteal callus
interfragmentary gap
endosteal callus
100
80
60
40
20
*
1
2
6
gap size [mm]

***Fig. 2.10*** Influence of fracture gap size and strain on tissue differentiation. Tissue differentiation as a function of fracture gap size and strain. With higher gaps and strains, there is an increasing proportion of connective tissue and fibrocartilage at the fracture site and within the callus. Conversely, low strains and gaps had higher amounts of bone formation. From Augat et al. [49], with permission

### *2.2.2.2 Fracture Gap*

While the strain theory accounts for some of the clinical observations seen in fracture healing, further work has shown that strain is not the only determinant of tissue differentiation. Fracture gap is as important, if not more important, than strain. Augat et al. and Claes et al. examined the effects of increasing gap size (1, 2, and 6 mm) and different strains (7 vs. 31%) on bone healing and mechanical strength. Augat demonstrated in a sheep model that higher gap sizes and higher strains led to lower amounts of bone formation and higher proportions of connective tissue and fibrocartilage formation across the fracture (Fig. 2.10) [49]. Increasing gap correlated with less bone formation. Cases in which bone failed to bridge the fracture gap were only observed for gaps >2 mm. Regardless of interfragmentary strain, gaps of 6 mm never healed. Strain played a more subtle role. While there was no difference in mechanical properties between strain groups, those that experienced higher strain (31%) had higher cartilage and fibrous tissue content, and lower bone content [49, 50]. Additionally, hydrostatic pressure and local stress play a role in tissue differentiation.

### *2.2.2.3 Timing in Fracture Healing*

Fracture healing involves a complex temporal and spatial sequence of events. The timing at which mechanical stimulation is introduced appears to affect the outcomes of skeletal repair. The initial mechanical environment is an early determinant of tissue differentiation and of healing outcome [14]. Immediate and early full weight bearing in a sheep model has been shown to delay healing, demonstrating lower bone content compared to delayed weight bearing [51]. Others have likewise shown that early or immediate mechanical loading led to decreased bone formation and inferior mechanical properties [52, 54]. These same studies also showed that delayed loading led to higher proportions of bone formation and improved biomechanical properties. Miclau et al. showed that delayed stabilization for even 24 h in mice led to higher cartilage callus formation and lower bone content compared to those who had immediate stabilization [53]. Taken together, these findings

demonstrate that timing of mechanical loading impacts fracture healing. When loading occurs prematurely or exceeds tolerable amounts, it can disrupt early healing and have deleterious effects. However, with callus providing some inherent stability across the fracture site, loading is better tolerated and may stimulate further callus formation and bony healing.

### 2.2.3 Assessment of Fracture Healing

The accurate assessment of fracture union is often a difficult undertaking, but nonetheless fundamental to clinical practice and research. Nonunions can be a source of significant disability, and its early diagnosis and treatment is paramount to improving patients' quality of life and return to function [55]. The definition of nonunion provided by the United States Food and Drug Administration (FDA) requires a minimum of at least nine months to elapse since the initial injury and no signs of healing for the final three months. Yet, there are no standardized methods of assessing fracture union, and there still remains considerable variability among clinicians and researchers alike [56, 57]. However, advances in imaging techniques, improved knowledge about the biology and biomechanics of fracture healing, and new scoring systems are refining our ability to assess fracture healing.

#### *2.2.3.1 Clinical Criteria*

Physical examination and clinical evaluation remain the cornerstone of fracture healing assessment. Weight bearing status has been shown to correlate with fracture tissue stiffness [58], though the clinicians' ability to assess stiffness is not reliable [59]. Weight bearing without pain is the most commonly endorsed factor, used in over half of all published studies to assess healing [57]. Pain at the fracture site and tenderness to palpation are also important signs in assessing healing. Conversely, the lack of weight bearing is considered the most important clinical criteria for impaired healing.

#### *2.2.3.2 Radiologic Scores*

The Radiographic Union Score for Hip (RUSH) and the Radiographic Union Score for Tibia (RUST) were developed to provide standardized, reliable radiographic measures of fracture healing [60–63]. These scoring systems evaluate healing on the basis of cortical bridging and fracture line visibility

on AP and lateral views (Table 2.2; Figs. 2.11 and 2.12). Both RUST and RUSH have high interobserver agreement, with intraclass correlation coefficients of 0.86 and 0.85, respectively. Compared to subjective assessment, these scores increase reliability and agreement among clinicians in assessing radiographic progression of fracture healing [62–65].

***Table 2.2*** Calculation of RUST and RUSH scores

| Score per cortex | Callus | Fracture line |
|---|---|---|
| 1 | Absent | Visible |
| 2 | Present | Visible |
| 3 | Present | Invisible |

The RUST and RUSH scores are based on radiographic findings on AP and lateral projections. Each cortex is scored according to the presence of callus and visibility of fracture line, with a maximum score of 12 for 4 cortices

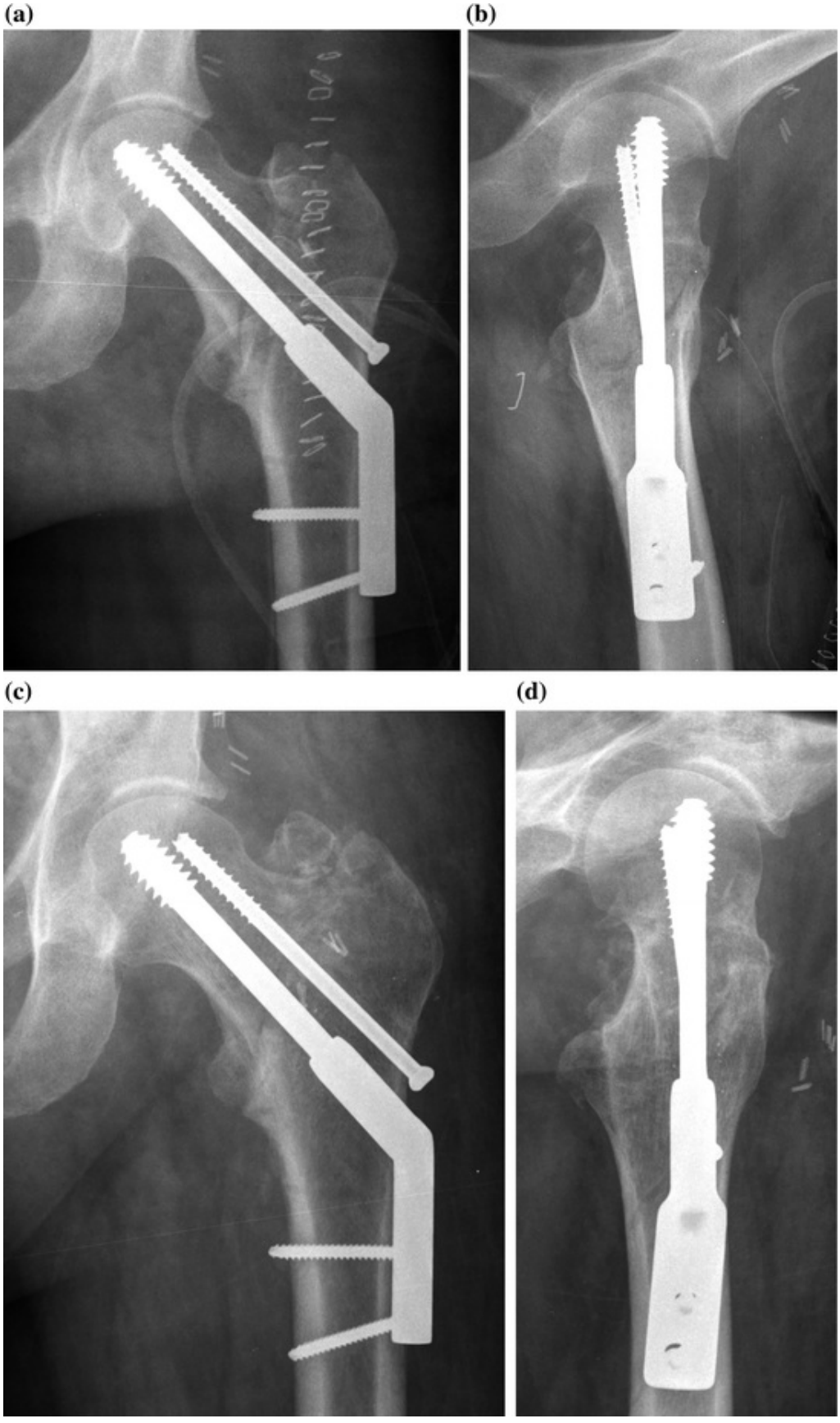

(a)
(b)
(c)
(d)

***Fig. 2.11*** Radiographic union score for hip (RUSH) fracture healing assessment, Assignment of RUSH in a patient who sustained a left intertrochanteric fracture. **a**, **b** Immediate postoperative radiographs, with a RUSH = 4. **c**, **d** 6-week follow-up radiographs, with a RUSH = 8, demonstrating callus on the anteroposterior view and lateral views, though the fracture lines are still visible

**(a)**


**(b)**

***Fig. 2.12*** Radiographic union score for tibia (RUST) fracture healing assessment. Assignment of RUST in a patient with distal tibial shaft fracture at 3 months. **a** At 4 weeks, there is healing callus along the medial, lateral, and anterior cortices, but fractures lines are visible. RUST score = 8. **b** At 10 weeks, there is bridging callus and no fracture line at the anterior and medial cortices. Fracture lines are still visible posteriorly and laterally. RUST score = 10

The lack of consensus in the orthopedic community limits the ability to establish consistent criteria to define union. Most practices use a combination of clinical and radiographic criteria to assess fracture healing. Additionally, several serologic markers of bone metabolism and cytokines, including TGF-β, have been identified as candidate biomarkers for tracking healing progression [8, 66]. Tools to measure mechanical properties in healing bone are also being developed. As our understanding of fracture healing continues to evolve, so too will our ability to gauge the healing process.

## 2.3 Modulation of Fracture Healing

### 2.3.1 Comorbidities

#### *2.3.1.1 Aging*

Aging has profound effects on bone health, modeling, and repair. Bone mass declines with advancing age, owing in part to hormonal changes, limited physical activity, and altered biologic responses. Additionally, elderly patients have a higher prevalence of comorbidities and take more medications, some of which may directly impact bone healing.

Animal studies have demonstrated decreased fracture healing capacity with increasing age [67]. Compared to adults, juveniles exhibit faster healing rates and remodeling potential [68]. In murine models, juveniles had more robust periosteal responses, higher chondrocytic and osteoblastic differentiation, and faster healing rates [67]. Additionally, juveniles mounted a larger angiogenic response, illustrated by higher VEGF, HIF-1α, and MMP expression [69]. In contrast, adults had relative delays in endochondral ossification, decreased periosteal thickness, and decreased chondrogenic potential in the periosteum [46]. Furthermore, skeletal maturity brought on a sharp drop in regenerative potential [67]. Additionally, elderly mice demonstrated decreased angiogenic potential [69]. In a murine model of senile osteoporosis, bone marrow-derived MSCs had increased adipogenic

and decreased osteogenic differentiation. Despite these abnormalities, the process of fracture healing was unchanged [70].

How aging affects fracture healing after skeletal maturity remains controversial, and the clinical evidence has thus far been limited and inconclusive. D'Ippolito et al. [71] demonstrated lower numbers of MSCs with osteogenic potential in adult human vertebrae. In contrast, Stenderup et al. [72] found no age-related decrement in the number of osteogenic stem cells from iliac crest marrow. The effects of age on fracture healing in humans, independent of other associated variables such as metabolic bone diseases, require further investigation.

### *2.3.1.2 Metabolic Bone Disease*

**Osteoporosis**

Osteoporosis is the most common metabolic bone disease, affecting over 200 million people worldwide [73]. Unlike normal age-related changes, osteoporosis is a metabolic disease characterized by decreased bone mass, decreased mineral content, increased porosity, and compromised microarchitecture. On a cellular level, the balance between anabolic and catabolic processes is unhinged to favor net bone resorption. Clinically, the weakened architecture predisposes to fragility fractures. Almost half of women with osteoporosis will sustain at least one fragility fracture in their lifetime [73].

Osteoporotic fractures are challenging to treat. Appropriate management requires an appreciation of how osteoporosis affects bone health, bone quality, and healing. As most clinical studies have focused on medical management and fracture prevention, there is limited data on how osteoporosis influences fracture healing in humans. More recently, Nikolaou et al. assessed the effect of osteoporosis on healing time in patients with femoral shaft fractures following intramedullary nailing. The elderly group of patients with radiologic evidence of osteoporosis had delayed healing compared to a younger cohort (19.4 weeks versus 16.2 weeks, respectively), though this difference is probably not clinically significant [74].

Animal studies have shown that osteoporosis impairs fracture healing. In an ovariectomized rat osteoporosis model, Namkung-Matthai et al. [75] demonstrated early failure in the repair process with a 40% reduction in callus size, and decreased bone mineral density and strength. Walsh et al.

[76] demonstrated delayed healing and decreased tensile and bending strength in estrogen-deficient rats. Lill et al. likewise demonstrated decreased bending stiffness and delayed healing in their osteoporotic sheep model. However, final strength at the end of healing was not different from healthy sheep [77].

To what degree osteoporosis impairs fracture healing remains unclear. While the healing potential is present in patients with osteoporosis, it may not be as robust. Furthermore, concomitant comorbidities such as vitamin D deficiency or other disorders of calcium homeostasis in these patients may also impair the healing response.

### *2.3.1.3 Endocrine Disorders*

Hyperparathyroidism , thyroid disorders, and hypogonadism have also been shown to impair fracture healing [78, 79]. In patients with unexplained nonunions, Brinker et al. found a high prevalence of these metabolic and endocrine disorders that had previously been unrecognized. The mechanisms by which these impede the healing process are still undetermined. However, medical management of the underlying abnormality, in conjunction with surgical fixation, successfully treats the majority of cases [78]. While routine screening is not indicated in the acute setting, impaired healing in otherwise appropriately treated fractures warrants further evaluation for metabolic abnormalities.

**Diabetes Mellitus**

Diabetes mellitus poses significant challenges to fracture management through impairment of healing, protective sensation, and host immunity. These effects are mediated by incompetent microcirculation, and in severe cases, they may also be associated with peripheral vascular disease. Delayed fracture healing in diabetic patients has been well documented. Early observations by Cozen showed significantly delayed fracture healing and nonunions in a series of diabetic patients [80]. Healing time in nondisplaced fractures was prolonged by 87% in non-neuropathic diabetic patients compared to nondiabetic patients [81].

Diabetes is a chronic inflammatory disorder; type I is an autoimmune disorder against insulin-producing islet of Langerhans beta cells, while type II is associated with obesity-related inflammation. Acute inflammation plays

a pivotal role in early fracture healing in recruiting skeletal progenitors to the site of injury. However, these events are tightly regulated; inflammatory cytokine levels are active within the first 72 h after injury, and at specific points in the healing cascade. Continued inflammation and continued cytokine expression, left unchecked, can halt the progression of bone remodeling and fracture healing [82, 83].

Recent evidence from animal studies suggests that uncontrolled diabetes may directly impact callus formation, chondrocyte survival, and osteoclast activity. Hyperglycemia upregulates the expression of proinflammatory factors, such as TNF-α and VEGF [82]. Upregulation of TNF-α stimulates chondrocyte apoptosis. Additionally, diabetes is associated with premature resorption of the cartilaginous callus and increased osteoclastogenesis. Impaired matrix synthesis, chondrocyte dysfunction, and premature resorption all decrease callus formation. These mechanisms may explain its weaker biomechanical strength in diabetic fracture healing [83–86].

Glycemic control should be the cornerstone of fracture management in diabetic patients. It has repeatedly been shown to reduce or prevent the aforementioned issues with bone healing [87]. Successful fracture healing in these patients often requires prolonged immobilization and weight bearing precautions [80]. Soft tissue management is also paramount, particularly in those with peripheral neuropathy. Surgical interventions likewise should respect soft tissue coverage; aggressive dissection and inattentiveness to soft tissue handling may further compromise the already tenuous blood supply in diabetic patients [81, 85, 88].

## 2.3.2 Habits

### *2.3.2.1 Smoking*

Smoking is well known to impair fracture healing. In multiple clinical trials, smoking has consistently been associated with nonunion, pseudarthrosis , and delayed healing. In the Lower Extremity Assessment Project (LEAP) , smokers, both former and active, were 32 and 37% more likely to develop nonunion, respectively. Smokers also required longer healing times [89, 90]. For midshaft clavicle fractures, smoking was the strongest risk factor for nonunion [91]. Among distal tibia fractures treated with two-ring hybrid external fixators, smoking delayed union by 10 weeks [92]. Additional, smoking has been associated with higher complication, reoperation, and

infection rates [89, 93].

Cigarette smoke contains hundreds of chemicals and gases, among them nicotine, carbon monoxide, and carcinogens. Carbon monoxide impairs oxygen delivery, creating a hypoxic environment for tissues. Nicotine induces vasoconstriction, likewise impairing oxygen delivery to tissues. Recent studies have found a bimodal dose-dependent effect of nicotine on osteoblasts. At high concentrations, nicotine had an inhibitory effect on osteoblast proliferation and differentiation, but at lower doses, it actually stimulated osteoblast activity [94]. While considered the addictive constituent in cigarettes, the role of nicotine in impaired fracture healing has undergone re-evaluation [95, 96]. Tobacco extract without nicotine reduced the mechanical strength in healing femoral fractures compared to nicotine alone [96]. The negative effects of smoking toward fracture healing are likely due to other constituents in cigarette smoke rather than from nicotine itself. These studies suggest that nicotine replacement may be safe and would reduce exposure to inhaled CO and other chemicals that may pose more physiologic harm.

#### *2.3.2.2 Alcohol Consumption*

Alcoholism and binge drinking are well-documented risk factors for traumatic injuries, disrupted bone metabolism, and impaired fracture healing. Not only does alcohol abuse confer higher fracture risk [97], but it also prolongs healing times. Nyquist et al. [98] showed that alcohol abusers with transverse tibia fractures required longer healing times than nonalcoholic patients. Alcoholic patients have lower bone mineral density and abnormal bone turnover markers consistent with defective bone formation and osteoblast dysfunction [99, 100]. Furthermore, alcoholism is frequently paired with smoking and malnutrition, which may further compromise bone health and bone repair [101].

Alcohol exposure predominantly affects early repair and bone formation [102–104]. In vitro osteoblast cultures demonstrate decreased proliferation and osteoid synthesis when exposed to ethanol. Additionally, rodent models have demonstrated decreased mechanical properties in fracture repair tissue following alcoholic ingestion [103]. In ethanol-fed rats, there was absence of mineralized callus on radiographs while in ethanol-free controls there was complete healing [105]. Recent evidence demonstrates that production of inflammatory cytokines, including IL-1 and TNF-α, increased oxidative

stress, and impaired Wnt signaling may mediate these effects [104].

Just as acute ingestion can lead to impaired healing, abstinence can lead reversal of its effects [102, 103]. Laitinen et al. [99] found that bone formation markers improved to near control levels after two weeks of abstinence. More recent evidence also suggests a role for antioxidant treatment with N-acetylcysteine in reversing the negative healing effects of alcohol consumption [106].

## 2.3.3 Medications

### *2.3.3.1 Nonsteroidal Anti-inflammatory Drugs*

Inflammation is critical in fracture healing. As part of the inflammatory cascade, cyclooxygenase (COX) converts arachidonic acid into prostaglandins [107, 108]. Downstream, prostaglandin $E_2$ ($PGE_2$) stimulates bone metabolism, bone formation, and maintenance [108, 109]. Deficient $PGE_2$ signaling conversely leads to osteopenia and impaired bone healing [110]. Additionally, COX-2 is essential to fracture healing, mediating repair through osteogenesis. COX-2 knockout mice fail to form mineralized matrix during endochondral ossification, where COX-1 knockout mice display no disruption in healing [111].

Nonsteroidal anti-inflammatory drugs (NSAIDs) exert their analgesic effect by interfering with prostaglandin production and COX function. NSAIDs have long been used as prophylaxis for heterotopic ossification, and human studies suggest adverse effects of NSAIDs on fracture repair. However, these clinical studies are all level III-IV data, have been retrospective, and have produced conflicting results [107, 108, 112, 113]. Giannoudis et al. correlated NSAID use > 4 weeks with higher rates of nonunion in femoral shaft fractures treated with intramedullary nailing. Even short-term use demonstrated delayed union [112]. However, this study was largely limited by its retrospective nature and lack of controls; whether true causality exists cannot be extrapolated from these results.

In animal studies, NSAIDs do appear to negatively affect skeletal repair [107, 108, 111, 114]. The earliest of these studies demonstrated that indomethacin treatment not only reduced the mechanical properties of rat femora during fracture healing, but also created fibrous tissue rather than callus between fractured ends [114]. Subsequent studies have also shown that the use of both nonselective and COX-2 selective NSAIDs decreases bone

formation and cortical bridging, prolongs healing times, and increases rates of nonunion [111, 115]. These effects do appear to be both time and dose dependent [14, 107, 108, 115, 116]. Aspirin, at doses equivalent to 325 mg, similarly delayed fracture healing, though smaller doses did not demonstrate any radiographic or mechanical differences compared with controls [116].

The importance of COX-2 and prostaglandins in fracture healing has been clearly established. While the mounting evidence in animal studies supports the effect of NSAIDs in suppressing fracture healing, translation of these effects to human subjects remains less convincing. As such, there is currently inadequate clinical evidence to prohibit their routine use in acute fracture care. NSAIDs remain an important feature in the development of a multimodal, opiate-sparing approach to postinjury and postsurgical pain regimen, and further clinical work is paramount in understanding its effects in orthopedic patients.

#### *2.3.3.2 Bisphosphonates*

Bisphosphonates are a mainstay of antiresorptive osteoporosis treatment. This class of drugs acts by inhibiting osteoclast-mediated resorption, improving bone mass and mineralization. However, there have been concerns about the hypothetical risk that bisphosphonates may impair bone healing. The reparative process relies on osteoclast-mediated remodeling of hard callus into woven bone and woven bone into mature lamellar bone.

Clinical studies have reported mixed results. In a retrospective review of humeral fractures, Solomon reported a higher nonunion rate with bisphosphonate use in the postfracture period. However, the conclusions of this study should be tempered with its limitations, including the rare occurrence of fractures (0.4%) and its retrospective design [117]. Rozental et al. explored the effect of bisphosphonate use on distal radius fracture healing time. Patients treated with bisphosphonates had slightly longer healing times (55 days versus 49 days), but this difference, while statistically significant, was not considered clinically significant [118]. More recently, Gong similarly investigated the impact of bisphosphonate treatment on healing in distal radius fractures after surgical fixation. There was no difference in time to union, or in radiographic or clinical outcomes [119]. In a randomized, double-blind, placebo-controlled trial using zoledronic acid after hip fracture, Lyles et al. did not find any evidence of delayed healing. Furthermore, if administered within 90 days after surgical fixation, zoledronic acid improved

survival and reduced the incidence of new clinical fractures [120].

Thus far, animal studies have been largely reassuring and have not demonstrated a detrimental effect of bisphosphonates on fracture healing. Rather, animals treated with bisphosphonates had increased callus formation and mineralization. Others have demonstrated some evidence of delay in callus remodeling and resorption, though there was no long-term impact on healing [121–124].

The short-term results of bisphosphonate use postfracture are encouraging. Clinical and basic science studies have not shown major differences in healing with bisphosphonate use. However, its long-term effects remain unclear. Furthermore, the emergence of atypical femur fractures associated with long-term bisphosphonate use has raised safety concerns (Fig. 2.13) [125]. These fractures have a reported prolonged healing course [126, 127]. As these fractures occur in the subtrochanteric region, an area subject to high stress and prone to malunion, it is difficult to ascertain whether these healing issues are a result of the fracture or a result of the drug effect. Additionally, while true causality has yet to be determined, the FDA has proposed offering a drug holiday for certain lower risk patients, though concrete guidelines defining appropriate candidates have not been established [128].

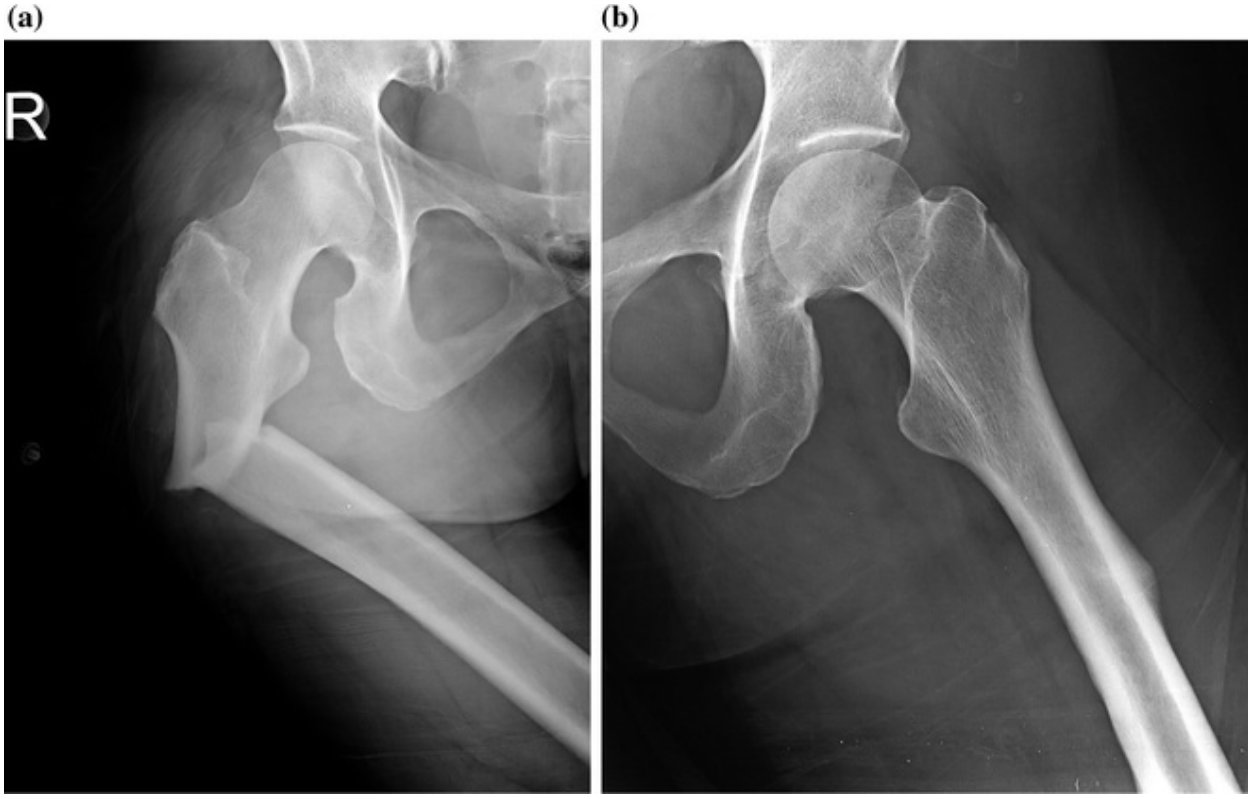



***Fig. 2.13*** Atypical femur fracture related to bisphosphonate use. The patient is a 43-year-old woman with a history of metastatic breast cancer status postlumpectomy and hormone therapy. She had a long history of bisphosphonate use. A recent positron emission tomography scan did not demonstrate any bony metastases. She sustained a low-energy right femur subtrochanteric oblique fracture after twisting that leg, consistent with an atypical femur fracture. **a** Imaging of the contralateral leg demonstrated stress reaction in the subtrochanteric region, concerning for an impending pathologic fracture **(b)**

### *2.3.3.3 Parathyroid Hormone Analogs*

Parathyroid hormone regulates serum calcium homeostasis via intestinal absorption, renal secretion, and bone metabolism. In the skeletal system, PTH binds to and stimulates osteoblasts to form new bone. Continuous PTH stimulation increases RANKL expression and decreases OPG expression, increasing osteoclast formation and catabolic function. However, intermittent PTH exposure preferentially stimulates anabolic osteoblast activity [124, 129].

Teriparatide , the biologically active 1–34 fragment of recombinant human PTH, is the first anabolic medication approved for osteoporosis [124], and its applications in fracture care are currently being investigated [130]. Animal studies in both rodent and simian models support PTH's role in enhancing fracture healing. In rodent models, PTH appears to accelerate

healing during chondrogenesis. PTH treatment elevates chondrogenic gene expression, cell recruitment, and differentiation, while osteogenic gene expression was not significantly increased. Additionally, PTH stimulates earlier chondrocyte hypertrophy and maturation of cartilage callus [131, 132]. Andreassen demonstrated increased fracture site strength and improved bone mineral content with PTH administration in a dose-dependent manner [133, 134]. Similarly, in monkeys, higher dose PTH treatment had smaller callus sizes, consistent with accelerated remodeling of callus to lamellar bone [135].

Early clinical results, while limited, have also been encouraging. In a prospective, randomized control trial, placebo, 20 μg teriparatide or 40 μg teriparatide was administered following distal radius fracture. Interestingly, median time to cortical bridging was significantly shorter in the 20 μg group (7.4 weeks) compared to both placebo (9.1 weeks) and 40 μg (8.8 weeks) groups [136]. In a prospective clinical trial of pelvic fractures using CT to evaluate fracture union, PTH treatment decreased healing time to 7.8 weeks, compared to 12.6 weeks for controls. Additionally, PTH-treated patients had better functional scores, with lower pain scores and faster “Timed Up and Go” testing compared to untreated patients [137].

## 2.4 Conclusion

In conclusion, fracture healing is a highly complex temporally and spatially coordinated process to restore mechanical integrity to bone following trauma. Appropriate management of both acute fractures and nonunions requires a comprehensive understanding of the principles that govern healing. This includes the biologic factors, the mechanical factors, and their interdependence. Previous work has concentrated on optimizing the mechanical environment for healing to occur, driving new innovations in implant design and function. More recently, the focus has shifted toward optimizing the biologic environment. The goal of fracture care is to achieve union in order to restore patients’ functionality and livelihood. To this end, our treatment strategies in fracture care will continue to evolve in stride with our growing understanding of fracture healing as well as its impact on patient-important outcomes such as health related quality of life and function.

## References

1. Spiegel DA, Gosselin RA, Coughlin RR, Joshipura M, Browner BD, Dormans JP. The burden of musculoskeletal injury in low and middle-income countries: challenges and opportunities. J Bone Joint Surg Am. 2008;90(4):915–23.
[PubMed]

2. United States Bone and Joint Initiative. Injuries. The burden of musculoskeletal diseases in the United States (BMUS), 3rd ed. Rosemont, IL; 2014. Available at http://www.boneandjointburden.org. Accessed 31 Aug 2015.

3. Burge R, Dawson-Hughes B, Solomon DH, Wong JB, King A, Tosteson A. Incidence and economic burden of osteoporosis-related fractures in the United States, 2005–2025. J Bone Miner Res. 2007;22(3):465–75.
[PubMed]

4. Tzioupis C, Giannoudis PV. Prevalence of long-bone non-unions. Injury. 2007;38(Suppl 2):S3–9. *Erratum* in Injury. 2007;38(10):1224.

5. Brinker M, O'Connor DP. The incidence of fractures and dislocations referred for orthopaedic services in a capitated population. J Bone Joint Surg. 2004;86-A(2):290–7.

6. Dahabreh Z, Dimitriou R, Giannoudis PV. Health economics: a cost analysis of treatment of persistent fracture non-unions using bone morphogenetic protein-7. Injury. 2007;38(3):371–7.
[PubMed]

7. Kanakaris NK, Giannoudis PV. The health economics of the treatment of long-bone non-unions. Injury. 2007;38(Suppl):S77–84.
[PubMed]

8. Einhorn TA. The cell and molecular biology of fracture healing. Clin Orthop Relat Res. 1998;355(Suppl):S7–21.

9. Marsell R, Einhorn TA. The biology fracture healing. Injury. 2011;42(6):551–5.
[PubMed]

10. Giannoudis PV, Einhorn TA, Marsh D. Fracture healing: the diamond concept. Injury. 2007;38(3 Suppl):S3–6.

11. Hankenson KD, Dishowitz M, Gray C, Schenker M. Angiogenesis in bone regeneration. Injury. 2011;42(6):556–61.
[PubMed]

12. Rhinelander FW. Tibial blood supply in relation to fracture healing. Clin Orthop Relat Res. 1974;105:34–81.

13. Thompson Z, Miclau T, Hu D, Helms JA. A model for intramembranous ossification during fracture healing. J Orthop Res. 2002;20(5):1091–8.
[PubMed]

14. Pape HC, Marcucio R, Humphrey C, Colnot C, Knobe M, Harvey EJ. Trauma-induced inflammation and fracture healing. J Orthop Trauma. 2010;24(9):522–5.

[PubMed]

15. Claes LE, Heigele CA. Magnitudes of local stress and strain along bony surfaces predict the course and type of fracture healing. J Biomech. 1999;32(3):255–66.
[PubMed]

16. Perren SM. Evolution of the internal fixation of long bone fractures. J Bone Joint Surg Br. 2002;84(8):1093–10.
[PubMed]

17. Rahn BA, Gallinaro P, Baltensperger A, Perren SM. Primary bone healing. An experimental study in the rabbit. J Bone Joint Surg. 1971;53(4):783–6.
[PubMed]

18. Schenck R, Willenegger H. On the histological picture of so-called primary healing of pressure osteosynthesis in experimental osteotomies in the dog. Experientia. 1963;19:593–5. [article in German].

19. Perren SM. Physical and biological aspects of fracture healing with special reference to internal fixation. Clin Orthop Relat Res. 1979;138:175–96.

20. Shapiro F. Cortical bone repair: the relationship of the lacunar-canalicular system and intercellular gap junctions to the repair process. J Bone Joint Surg. 1988;70(7):1067–81.
[PubMed]

21. Cruess RL, Dumont J. Fracture healing. Can J Surg. 1975;18(5):403–13.
[PubMed]

22. Kolar P, Schmidt-Bleed K, Schell H, Gaber T, Toben D, Schmidmaier G, et al. The early fracture hematoma and its potential role in fracture healing. Tissue Eng Part B Rev. 2010;16(4):427–34.
[PubMed]

23. Oe K, Miwa M, Sakai Y, Lee SY, Kuroda R, Kurosaka M. An in vitro study demonstrating that haematomas found at the site of human fractures contain progenitor cells with multilineage capacity. J Bone Joint Surg Br. 2007;89(1):133–8.
[PubMed]

24. Tsiridis E, Upadhyay N, Giannoudis P. Molecular aspects of fracture healing: which are the important molecules? Injury. 2007;38(Suppl 1):S11–25.
[PubMed]

25. Sarahrudi K, Thomas A, Albrecht C, Aharinejad S. Strongly enhanced levels of sclerostin during human fracture healing. J Orthop Res. 2012;30(10):1549–55.
[PubMed]

26. Sarahrudi K, Thomas A, Mousavi M, Kaiser G, Kottstorfe J, Kecht M, et al. Elevated transforming growth factor-beta 1 (TGF-ß1) levels in human fracture healing. Injury. 2011;42(8):833–7.
[PubMed]

27. Sarahrudi K, Mousavi M, Thomas A, Eipeldauer S, Vecsei C, Pietschmann P, Aharinejad S.

Elevated levels of macrophage colony-stimulating factor in human fracture healing. J Orthop Res. 2010;28(5):671–6.
[PubMed]

28. Bielby R, Jones E, McGonagle. The role of mesenchymal stem cells in maintenance and repair of bone. Injury. 38(Suppl 1):S26–32.

29. Malizos KN. Paptheodorou. The healing potential of the periosteum. Injury. 2005;36(Suppl 3):S13–9.
[PubMed]

30. Shah K, Majeed Z, Jonason J, O'Keefe RJ. The role of muscle in bone repair: the cells, signals, and tissue responses to injury. Curr Osteoporos Rep. 2013;11(2):130–5.
[PubMed]

31. Yang X, Ricciardi BG, Hernandez-Soria A, Shi Y, Pleshko Camacho N, Bostrom MP. Callus mineralization and maturation are delayed during fracture healing in interleukin-6 knockout mice. Bone. 2007;41(6):928–36.
[PubMed]

32. Glass GE, Chan JK, Freidin A, Feldmann M, Horwood NJ, Nanchahal J. TNF-alpha promotes fracture repair by augmenting the recruitment and differentiation of muscle-derived stromal cells. Proc Nat Acad Sci U S A. 2011;108(4):1585–90.

33. Lehmann W, Edgar CM, Wang K, Cho TJ, Barnes GL, Kakar S, et al. Tumor necrosis factor alpha (TNF-A) coordinately regulates the expression of specific matrix metalloproteinases (MMPs) and angiogenic factors during fracture healing. Bone. 2005;36:300–10.
[PubMed]

34. Gerstenfeld LC, Cho TJ, Kon T, Aizawa T, Tsay A, Fitch J, et al. Impaired fracture healing in the absence of TNF-alpha signaling: the role of TNF-alpha in endochondral cartilage resorption. J Bone Miner Res. 2003;18(9):1584–92.
[PubMed]

35. Kitaori T, Ito H, Schwarz EM, Tsutsumi R, Yoshitomi H, Oishi S, et al. Stromal cell-derived factor 1/CXCR4 signaling is critical for the recruitment of mesenchymal stem cells to the fracture site during skeletal repair in a mouse model. Arthritis Rheum. 2009;60(3):813–23.
[PubMed]

36. Schindeler A, McDonald MM, Bokko P, Little DG. Bone remodeling during fracture repair: the cellular picture. Semin Cell Dev Biology. 2008;19(5):459–66.

37. Lu C, Saless N, Wang X, Sinha A, Decker S, Kazakia G, et al. The role of oxygen during fracture healing. Bone. 2013;52(1):220–9.
[PubMed]

38. Keramaris NC, Calori GM, Nikolaou VS, Schemitsch EH, Giannoudis PV. Fracture vascularity and bone healing: a systematic review of the role of VEGF. Injury. 2008;39(Suppl 2):S45–57.
[PubMed]

39. Gerber HP, Vu TH, Ryan AM, Kowalski J, Werb Z, Ferrara N. VEGF couples hypertrophic cartilage remodeling, ossification and angiogenesis during endochondral bone formation. Nat

Med. 1999;5(6):623–8.
[PubMed]

40. Behonick DJ, Xing Z, Lieu S, Buckley JM, Lotz JC, Marcucio RS, et al. Role of matrix metalloproteinase 13 in both endochondral and intramembranous ossification during skeletal regeneration. PLoS ONE. 2007;2(11):e1150.
[PubMed]

41. Shapiro F. Bone development and its relation to fracture repair. The role of mesenchymal osteoblasts and surface osteoblasts. Eur Cell Mater. 2008;15:53–76.
[PubMed]

42. Flick LM, Weaver JM, Ulrich-Vinther M, Abuzzahab F, Zhang X, Dougall WC, et al. Effects of receptor activator of NFkappaB (RANK) signaling blockade on fracture healing. J Orthop Res. 2003;21(4):676–84.
[PubMed]

43. Raisz LG. Physiology and pathophysiology of bone remodeling. Clin Chem. 1999;45(8 Pt 2):1353–8.
[PubMed]

44. Sims NA, Gooi JH. Bone remodeling: multiple cellular interactions required for coupling of bone formation and resorption. Semin Cell Dev Biol. 2008;19(5):444–51.
[PubMed]

45. Teitelbaum SL. Bone resorption by osteoclasts. Science. 2000;289(5484):1504–8. (Review).

46. Fan W, Crawford R, Xiao Y. Structural and cellular differences between metaphyseal and diaphyseal periosteum in different aged rats. Bone. 2008;42(1):81–9.
[PubMed]

47. Uhthoff HK, Rahn BA. Healing patterns of metaphyseal fractures. Clin Orthop Relat Res. 1981;160:295–303.

48. Claes L, Reusch M, Göckelmann M, Ohnmacht M, Wehner T, Amling M, et al. Metaphyseal fracture healing follows similar biomechanical rules as diaphyseal healing. J Orthop Res. 2011;29(3):425–32.
[PubMed]

49. Augat P, Margevicius K, Simon J, Wolf S, Suger G, Claes L. Local tissue properties in bone healing: influence of size and stability of the osteotomy gap. J Orthop Res. 1998;16(4):475–81.
[PubMed]

50. Claes LE, Heigele CA, Neidlinger-Wilke C, Kaspar D, Seidl W, Margevicius KJ, Augat P. Effects of mechanical factors on the fracture healing process. Clin Orthop Relat Res. 1998;(355 Suppl):S132–47.

51. Augat P, Merk J, Ignatius A, Margevicius K, Bauer G, Rosenbaum D, Claes L. Early full weightbearing with flexible fixation delays fracture healing. Clin Orthop Relat Res. 1996;328:194–202.

52. Willie BM, Blakytny R, Glockelmann M, Ignatius A, Claes L. Temporal variation in fixation

stiffness affects healing by differential cartilage formation in a rat osteotomy model. Clin Orthop Relat Res. 2011;469(11):3094–101.
[PubMed]

53. Miclau T, Lu C, Thompson Z, Choi P, Puttlitz C, Marcucio R, Helms JA. Effects of delayed stabilization on fracture healing. J Orthop Res. 2007;25(12):1552–8.
[PubMed]

54. Weaver AS, Su YP, Begun DL, Miller JD, Alford AI, Goldstein SA. The effects of axial displacement on fracture callus morphology and MSC homing depend in the timing of application. Bone. 2010;47(1):41–8.
[PubMed]

55. Brinker MR, Hanus BD, Sen M, O'Connor DP. The devastating effects of tibial nonunion on health-related quality of life. J Bone Joint Surg Am. 2013;95(24):2170–6.
[PubMed]

56. Bhandari M, Fong K, Sprague S, Williams D, Petrisor B. Variability in the definition and perceived causes of delayed unions and nonunions: a cross-sectional, multinational survey of orthopaedic surgeons. J Bone Joint Surg Am. 2012;94(15):e1091–6.
[PubMed]

57. Corrales LA, Morshed S, Bhandari M, Miclau T 3rd. Variability in the assessment of fracture-healing in orthopaedic trauma studies. J Bone Joint Surg Am. 2008;90(9):1862–8.
[PubMed]

58. Joslin CC, Eastaugh-Waring SJ, Hardy JR, Cunningham JL. Weight bearing after tibial fracture as a guide to healing. Clin Biomech (Briston, Avon). 2008;23(3):329–33.

59. Webb J, Herling G, Gardner T, Kenwright J, Simpson AH. Manual assessment of fracture stiffness. Injury. 1996;27(5):319–20.
[PubMed]

60. Whelan DB, Bhandari M, Stephen D, Kreder H, McKee MD, Zdero R, Schemitsch EH. Development of the radiographic union score for tibial fractures for the assessment of tibial fracture healing after intramedullary fixation. J Trauma. 2010;68(3):629–32.
[PubMed]

61. Kooistra BW, Dijkman BG, Busse JW, Sprague S, Schemitsch EH, Bhandari M. The radiographic union scale in tibial fractures: reliability and validity. J Orthop Trauma. 2010;24(Suppl 1):S81–6.
[PubMed]

62. Bhandari M, Chiavaras M, Ayeni O, Chakraverrty R, Parasu N, Choudur H, et al. Assessment Group for Radiographic Evaluation and Evidence (AGREE) Study Group (AGREE Investigators Writing Committee). Assessment of radiographic fracture healing in patients with operatively treated femoral neck fractures. J Orthop Trauma. 2013;27(9):e213–9.
[PubMed]

63. Bhandari M, Chiavaras MM, Parasu N, Choudur H, Ayeni O, Chakravertty R, et al. Radiographic union score for hip substantially improves agreement between surgeons and radiologists. BMC Musculoskelet Disord. 2013;14:70.

[PubMed]

64. McClelland D, Thomas PB, Bancroft G, Moorcroft CI. Fracture healing assessment comparing stiffness measurements using radiographs. Clin Orthop Relat Res. 2006;457:214–9.

65. Davis BJ, Roberts PJ, Moorcroft CI, Brown MF, Thomas PB, Wade RH. Reliability of radiographs in defining union of internally fixed fractures. Injury. 2004;35(6):557–61.
[PubMed]

66. Cox G, Einhorn TA, Tzioupis C, Giannoudis PV. Bone-turnover markers in fracture healing. J Bone Joint Surg Br. 2010;92(3):329–34.
[PubMed]

67. Lu C, Miclau T, Hu D, Hansen E, Tsui K, Puttlitz C, Marcucio RS. Cellular basis for age-related changes in fracture repair. J Orthop Res. 2005;23(6):1300–7.
[PubMed]

68. Desai BJ, Meyer MH, Porter S, Kellam JF, Meyer RA Jr. The effect of age on gene expression in adult and juvenile rats following femoral fracture. J Orthop Trauma. 2003;17(10):689–98.
[PubMed]

69. Lu C, Sapozhnikova A, Hu D, Miclau T, Marcucio RS. Effect of age on vascularization during fracture repair. J Orthop Res. 2008;26(10):1384–9.
[PubMed]

70. Egermann M, Heil P, Tami A, Ito K, Janicki P, Von Rechenberg B, et al. Influence of defective bone marrow osteogenesis on fracture repair in an experimental model of senile osteoporosis. J Orthop Res. 2009;28(6):798–804.

71. D'Ippolito G, Schiller PC, Ricordi C, Roos BA, Howard GA. Age-related osteogenic potential of mesenchymal stromal stem cells from human vertebral bone marrow. J Bone Miner Res. 1999;14(7):1115–22.
[PubMed]

72. Stenderup K, Justesen J, Eriksen EF, Rattan S, Kassem M. Number and proliferative capacity of osteogenic stem cells are maintained during aging and in patients with osteoporosis. J Bone Miner Res. 2001;16(6):1120–9.
[PubMed]

73. International Osteoporosis Foundation. Epidemiology. http://www.iofbonehealth.org/epidemiology. Accessed 27 Aug 2015.

74. Nikolaou VS, Efstathopoulos N, Kontakis G, Kanakaris NK, Giannoudis PV. The influence of osteoporosis in femoral fracture healing time. Injury. 2009;40(6):663–8.
[PubMed]

75. Namkung-Matthai H, Appleyard R, Jansen J, Hao Lin J, Maastricht S, Swain M, et al. Osteoporosis influences the early period of fracture healing in a rat osteoporotic model. Bone. 2001;28(1):80–6.
[PubMed]

76. Walsh WR, Sherman P, Howlett CR, Sonnabend DH, Ehrlich MG. Fracture healing in a rat

osteopenia model. Clin Orthop Relat Res. 1997;342:218–27.

77. Lill CA, Hesseln J, Schlegel U, Eckhardt C, Goldhahn J, Schneider E. Biomechanical evaluation of healing in a non-critical defect in a large animal model of osteoporosis. J Orthop Res. 2003;21(5):836–42.
[PubMed]

78. Brinker MR, O'Connor DP, Monla YT, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trauma. 2007;21(8):557–70.
[PubMed]

79. Lancourt JE, Hochberg F. Delayed fracture healing in primary hyperparathyroidism. Clin Orthop Rel Res. 1977;124:214–8.

80. Cozen L. Does diabetes delay fracture healing? Clin Orthop Relat Res. 1972;82:134–40.
[PubMed]

81. Loder R. The influence of diabetes mellitus on the healing of closed fractures. Clin Orthop. 1988;232:210–6.

82. Albowi J, Tian C, Siqueira MF, Kayal RA, McKenzie E, Behl Y, et al. Chemokine expression is upregulated in chondrocytes in diabetic fracture healing. Bone. 2013;53(1):294–300.

83. Kayal RA, Siqueira M, Alblowi J, McLean J, Krothapalli N, Faibish D, et al. TNF-alpha mediates diabetes-enhanced chondrocyte apoptosis during fracture healing and stimulates chondrocyte apoptosis through FOXO1. J Bone Miner Res. 2010;25(7):1604–15.
[PubMed]

84. Kayak RA, Tsatsas D, Bauer MA, Allen B, Al-Sebaei MO, Kakar S, et al. Diminished bone formation during diabetic fracture healing is related to the premature resorption of cartilage associated with increased osteoclast activity. J Bone Miner Res. 22(4);560–8.

85. Kagel EM, Einhorn TA. Alterations of fracture healing in the diabetic condition. Iowa Orthop J. 16:147–52.

86. Follak N, Klöting I, Merk H. Influence of diabetic metabolic state on fracture healing in spontaneously diabetic rats. Diabetes Metab Res Rev. 2005;21(3):288–96.
[PubMed]

87. Gandhi A, Beam HA, O'Connor JP, Parsons JR, Lin SS. The effects of local insulin delivery on diabetic fracture healing. Bone. 2005;37(4):482–90.
[PubMed]

88. Chaudhary SB, Liporace FA, Gandhi A, Donley BG, Pinzur MS, Lin SS. Complications of ankle fracture in patients with diabetes. J Am Acad Orthop Surg. 2008;16(3):159–70.
[PubMed]

89. Castillo RC, Bosse MJ, MacKenzie EJ. Patterson BM; LEAP Study Group. Impact of smoking on fracture healing and risk of complications in limb-threatening open tibia fractures. J Orthop Trauma. 2005;19(3):151–7.

[PubMed]

90. Adams CI, Keating JF, Court-Brown CM. Cigarette smoking and open tibial fractures. Injury. 2001;32(1):61–5.
[PubMed]

91. Murray IR, Foster CJ, Robinson CM. Risk factors for nonunion after nonoperative treatment of displaced midshaft fractures of the clavicle. J Bone Joint Surg Am. 2013;95(13):1153–8.
[PubMed]

92. Ristiniemi J, Flinkkila T, Hyvonen P, Lakovaara M, Pakarinen H, Biancari F, Jalovaara P. Two-ring hybrid external fixation of distal tibial fractures: a review of 47 cases. J Trauma. 2007;62(1):174–83.
[PubMed]

93. Govender S, Csimma C, Genant HK, Valentin-Opran A, Amit Y, Arbel R, et al. BMP-2 Evaluation in Surgery for Tibial gtrauma (BESTT) Study Group. Recombinant human bone morphogenetic protein-2 for treatment of open tibial fractures: a prospective, controlled, randomized study of four hundred and fifty patients. J Bone Joint Surg Am. 2002;84-A(12):2123–34.

94. Shen Y, Liu HX, Ying XZ, Yang SZ, Nie PF, Cheng SW, et al. Dose-dependent effects of nicotine on proliferation and differentiation of human bone marrow stromal cells and the antagonistic action of vitamin C. J Cell Biochem. 2013;114(8):1720–8.
[PubMed]

95. Lee JJ, Patel R, Biermann S, Dougherty PJ. The musculoskeletal effects of cigarette smoking. J Bone Joint Surg Am. 2013;95(9):850–9.
[PubMed]

96. Skott M, Andreassen TT, Ulrich-Vinther M, Chen X, Keyler DE, LeSage MG, et al. Tobacco extract but not nicotine impairs the mechanical strength of fracture healing in rats. J Orthop Res. 2006;24(7):1472–9.
[PubMed]

97. Kristensson H, Lunden A, Nilsson BE. Fracture incidence and diagnostic roentgen in alcoholics. Acta Orthop Scan. 1980;51(2):205–7.

98. Nyquist F, Berglund M, Nilsson BE, Obrant KJ. Nature and healing of tibial shaft fractures in alcohol abusers. Alcohol. 1997;32(1):91–5.

99. Laitinen K, Lamberg-Allardt C, Tunninen R, Harkonen M, Valimski M. Bone mineral density and abstention-induced changes in bone and mineral metabolism in noncirrhotic male alcoholics. Am J Med. 1992;93(6):642–50.
[PubMed]

100. Diamond T, Stiel D, Lunzer M, Wilkinson M, Posen S. Ethanol reduces bone formation and may cause osteoporosis. Am J Med. 1989;86(3):282–8.
[PubMed]

101. Hillers VN, Massey LK. Interrelationships of moderate and high alcohol consumption with diet and health status. Am J Clin Nutr. 1985;41(2):356–62.

[PubMed]

102. Chakkalakal DA. Alcohol-induced bone loss and deficient bone repair. Alc Clin Exper Res. 2005;29(12):2077–90.

103. Chakkalakal DA, Novak JR, Fritz ED, Mollner TJ, McVicker DL, Garvin KL, et al. Inhibition of bone repair in a rat model for chronic and excessive alcohol consumption. Alcohol. 2005;36(3):1–14.

104. Jung MK, Callaci JJ, Lauing KL, Otis JS, Radek KA, Jones MK, Kovacs EJ. Alcohol exposure and mechanisms of tissue injury and repair. Alcohol Clin Exp Res. 2011;35(3):392–9.
[PubMed]

105. Janicke-Lorenz J, Lorenz R. Alcoholism and fracture healing. a radiological study in the rat. Arch Orthop Trauma Surg. 1984;103(4):286–9.
[PubMed]

106. Volkmer DL, Sears B, Lauing KL, Nauer RK, Roper PM, Yong S, et al. Antioxidant therapy attenuates deficient bone fracture repair associated with binge alcohol exposure. J Orthop Trauma. 2011;25(8):516–21.
[PubMed]

107. Kurmis AP, Kurmis TP, O'Brien JX, Dalén T. The effect of nonsteroidal anti-inflammatory drug administration on acute phase fracture-healing: a review. J Bone Joint Surg Am. 2012;94(9):815–23.
[PubMed]

108. Abdul-Hadi O, Parvizi J, Austin MA, Viscusi E, Einhorn T. Nonsteroidal anti-inflammatory drugs in orthopaedics. J Bone Joint Surg Am. 2009;91(8):2019–27.

109. Jee WS, Ma YF. The in vivo anabolic actions of prostaglandins in bone. Bone. 1997;21(4):297–304.
[PubMed]

110. Li M, Healy DR, Li Y, Simmons HA, Crawford DT, Ke HZ, et al. Osteopenia and impaired fracture healing in aged EP4 receptor knockout mice. Bone. 2005;37(1):46–54.
[PubMed]

111. Simon AM, Manigrasso MB, O'Connor JP. Cyclo-oxygenase 2 function is essential for bone fracture healing. J Bone Miner Res. 2002;17(6):963–76.
[PubMed]

112. Giannoudis PV, MacDonald DA, Matthews SJ, Smith RM, Furlong AJ, De Boer P. Nonunion of the femoral diaphysis: the influence of reaming and non-steroidal anti-inflammatory drugs. J Bone Joint Surg Br. 2000;82(5):655–8.
[PubMed]

113. Dahners LE, Mullis BH. Effects of nonsteroidal anti-inflammatory drugs on bone formation and soft-tissue healing. J Am Acad Orthop Surg. 2004;12(3):139–43.
[PubMed]

114.

Ro J, Sudmann E, Marton PF. Effect of indomethacin on fracture healing in rats. Acta Orthop Scand. 1976;47(6):588–99.

115. Gerstenfeld LC, Thiede M, Seibert K, Mielke C, Phippard D, Svagr B, et al. Differential inhibition of fracture healing by non-selective cyclooxygenase-2 selective non-steroidal anti-inflammatory drugs. J Orthop Res. 2003;21(4):670–5.
[PubMed]

116. Lack WD, Fredericks D, Petersen E, Donovan M, George M, Nepola J, et al. Effect of aspirin on bone healing in a rabbit ulnar osteotomy model. J Bone Joint Surg Am. 2013;95(6):488–96.
[PubMed]

117. Solomon HM, Mogun H, Schneeweiss S. The relation between bisphosphonate use and non-union of fractures of the humerus in older adults. Osteoporos Int. 2009;20(6):895–901.
[PubMed]

118. Rozental TD, Vazquez MA, Chacko AT, Ayogu N, Bouxsein ML. Comparison of radiographic fracture healing in the distal radius for patients on and off bisphosphonate therapy. J Hand Surg Am. 2009;34(4):595–602.

119. Gong HS, Song CH, Lee YH, Thee SH, Lee HJ, Baek GH. Early initiation of bisphosphonate does not affect healing and outcomes of volar plate fixation of osteoporotic distal radial fractures. J Bone Joint Surg Am. 2012;94(19):1729–36.
[PubMed]

120. Lyles KW, Colón-Emeric CS, Magaziner JS, Adachi JD, Pieper CF, Mautalen C, et al. HORIZON recurrent fracture trial. Zoledronic acid and clinical fractures and mortality after hip fracture. N Engl J Med. 2007;357(18):1799–809.
[PubMed]

121. Li J, Mori S, Kaji Y, Mashiba T, Kawanishi J, Norimatsu H. Effect of bisphosphonate (incadronate) on fracture healing of long bones in rats. J Bone Miner Res. 1999;14:969–79.
[PubMed]

122. McDonald MM, Dulai S, Godfrey C, Amanat N, Sztynda T, Little DG. Bolus or weekly zoledronic acid administration does not delay endochondral fracture repair but weekly dosing enhances delays in hard callus remodeling. Bone. 2008;43(4):653–62.

123. Saito M, Shiraishi A, Ito M, Sakai S, Kayakawa N, Mihara M, Marumo K. Comparison of effects of alfacalcidol and alendronate on mechanical properties and bone collagen cross-links of callus in the fracture repair rat model. Bone. 2010;46(4):1170–9.
[PubMed]

124. Jørgensen NR, Schwarz P. Effects of anti-osteoporosis medications on fracture healing. Curr Osteoporos Rep. 2011;9(3):149–55.
[PubMed]

125. Odvina CV, Zerwekh JE, Rao DS, Maalouf N, Gottschalk FA, Pak CY. Severely suppressed bone turnover: a potential complication of alendronate therapy. J Clini Endocrinol Metab. 2005;90(3):1294–301.

126. Egol KA, Park JH, Rosenberg ZS, Peck V, Tejwani NC. Healing delayed but generally reliable after bisphosphonate-associated complete femur fractures treated with IM nails. Clin Orthop Relat Res. 2014;472(9):2728–34.
[PubMed]

127. Weil YA, Rivkin G, Safran O, Liebergall M, Foldes AJ. The outcome of surgically treated femur fractures associated with long-term bisphosphonate use. J Trauma. 2011;71(1):186–90.
[PubMed]

128. Diab DL, Watts NB. Bisphosphonate drug holiday: who, when and how long. Ther Adv Musculoskelet Dis. 2013;5(3):107–11.
[PubMed]

129. Ellegaard M, Jorgensen NR, Schwarz P. Parathyroid hormone and bone healing. Calcif Tissue Int. 2010;87(1):1–13.
[PubMed]

130. Goldhahn J, Feron JM, Kanis J, Papapoulos A, Reginster JY, Rizzoli R, et al. Implications for fracture healing of current and new osteoporosis treatments: an ESCEO consensus paper. Calcif Tissue Int. 2012;90(5):343–53.
[PubMed]

131. Kakar S, Einhorn TA, Vora S, Miara LJ, Hon G, Wigner NA, et al. Enhanced chondrogenesis and Wnt signaling in PTH-treated fractures. J Bone Miner Res. 2007;22(12):1903–12.
[PubMed]

132. Holzer G, Majeska RJ, Lundy MW, Hartke JR, Einhorn TA. Parathyroid hormone enhances fracture healing. A preliminary report. Clin Orthop Relat Res. 1999;366:258–63.

133. Andreassen TT, Ejersted C, Oxlund H. Intermittent parathyroid hormone (1–34) treatment increases callus formation and mechanical strength of healing rat fractures. J Bone Miner Res. 1999;14(6):960–8.
[PubMed]

134. Andreassen TT, Willick GE, Morley P, Whitfield JF. Treatment with parathyroid hormone hpth(1–34), hpth(1–31), and monocyclic hpth(1–31) enhances fracture strength and callus amount after withdrawal fracture strength and callus mechanical quality continue to increase. Calcif Tissue Int. 2004;74(4):351–6.
[PubMed]

135. Manabe T, Mori S, Mashiba T, Kaji Y, Iwata K, Komatsubara S. Human parathyroid hormone (1–34) accelerates natural fracture healing process in the femoral osteotomy model of cynomolgus monkeys. Bone. 2007;40(6):1475–82.
[PubMed]

136. Aspenberg P, Genant HK, Johansson T, Nino AJ, See K, Krohn K, et al. Teriparatide for acceleration of fracture repair in humans: a prospective, randomized double-blind study of 102 postmenopausal women with distal radial fractures. J Bone Miner Res. 2010;25(2):404–14.
[PubMed]

137.

Peichl P, Holzer LA, Maier R, Holzer G. Parathyroid hormone 1-84 accelerates fracture-healing in pubic bones of elderly osteoporotic women. J Bone Joint Surg Am. 2011;93(17):1583–7. [PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_3

# 3. Clavicle Nonunions

Laura A. Schemitsch[1], Emil H. Schemitsch[1] and Michael D. McKee[1]

(1) Department of Surgery, Division of Orthopedic Surgery, University of Toronto, St. Michael's Hospital, Toronto, Canada

**Emil H. Schemitsch**
**Email:** schemitsche@smh.ca

**Keywords** Clavicle fractures – Nonunion – Treatment strategies – Review – Evidence

## 3.1 Introduction

The clavicle is a flat S-shaped bone that connects the arm to the body and is located directly above the first rib. It is the first bone to ossify in utero [1]. Fractures of the clavicle are particularly common, as they account for approximately 2–5% of all fractures [2, 3] and 44% of fractures to the shoulder girdle [4]. These injuries tend to occur most frequently in young and active individuals, particularly men [5].

Fractures of the distal third appear in roughly 20% of cases (Fig. 3.1), mainly occurring in elderly patients as a result of a low impact fall [6–11]. In rare occurrences (5%), the medial end of the clavicle bone is fractured [7, 12, 13]. In the majority of cases, the midshaft of the clavicle fractures, accounting for approximately 80–85% of all cases [7, 14–17]. Fractures of the midshaft were traditionally treated nonoperatively due to the reported low incidence of nonunion in past studies in which clavicular fractures had been treated conservatively [16, 18, 19]. Nonunion is defined as the lack of

radiographic healing at six months post-injury [18]. Callus formation indicates that a hypertrophic nonunion has occurred, whereas an atrophic nonunion results when no callus has formed. In an article published in the *American Journal of Surgery* in 1941, Ghormley et al. stated that clavicular fractures resulting in nonunion were “unusual” [20]. Neer’s evaluation of 2235 patients in 1960 reported only three patients as having un-united fractures [2]. In 1968, Rowe reported four nonunions within a series of 566 patients, thus resulting in an extremely low rate of nonunion (0.8%) [4]. Although nonunion was considered to be a serious complication in relation to clavicle fractures, the understanding that it was rare had significant implications discouraging operative intervention.

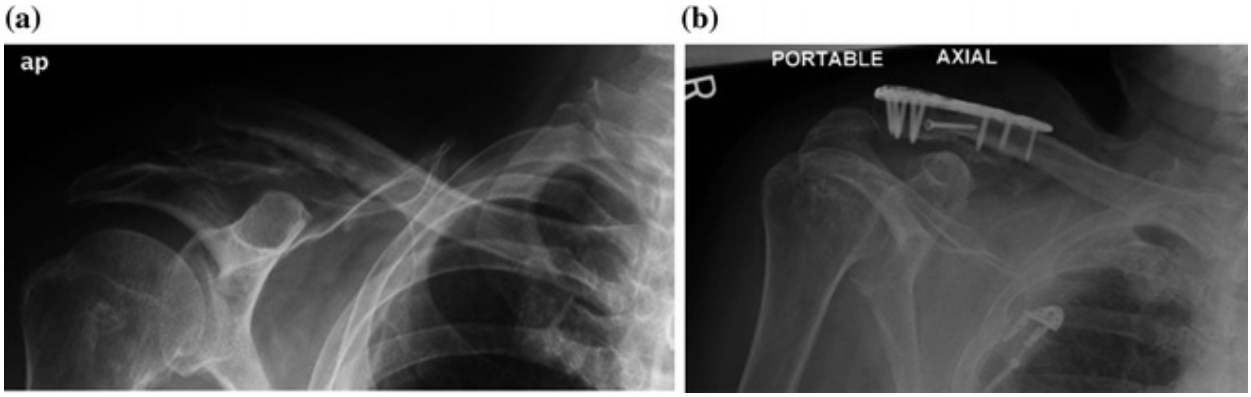



***Fig. 3.1*** **a** Preoperative radiograph of distal clavicle nonunion. **b** Post-operative radiograph of distal clavicle nonunion following open reduction and internal fixation with a precontoured distal clavicle plate and a lag screw

Recent studies of displaced midshaft clavicle fractures have shed new light on the reported incidence of nonunion. In 1997, Hill et al. reviewed 52 patients with displaced midshaft clavicle fractures treated nonoperatively [21]. They reported a nonunion rate of 15% (8 out of 52) and an unsatisfactory clinical outcome in 31% of patients. In a review of 2144 displaced midshaft fractures collected from the literature between 1975 and 2005, Zlowodzki et al. reported a nonunion rate of 15.1% (Table 3.1) [22]. This is markedly higher than the rate described by Neer and Rowe. Several other recent studies have reinforced these findings [8, 17, 23–27]. In 2007, a study conducted by the Canadian Orthopedic Trauma Society compared nonoperative treatment versus plate fixation for displaced midshaft clavicle fractures [18]. This series found that there was a significantly lower rate of nonunion in the operative group (62 patients, 2 nonunions, rate of nonunion

3%) compared with the patients treated conservatively (49 patients, 7 nonunions, rate of nonunion 14%). These findings are helpful in establishing that primary fixation may benefit young and active individuals with fully displaced midshaft clavicle fractures. An assessment of a patient's injuries as well as their functional expectations remains one of the most important considerations regarding decisions for treatment [7]. While operative treatment is typically effective for established nonunions, primary prevention of nonunion would be preferential (Figs. 3.2 and 3.3).

***Table 3.1*** Nonunion of the clavicle following various treatments. Meta-analysis of nonoperative treatment, intramedullary pinning, and plate fixation of displaced midshaft fractures of the clavicle from series published in 1975 through 2005 (From Zlowodzki et al. [22], with permission)

| | **Nonunions** | **Infections (Total)** | **Infection (Deep)[a]** | **Fixation Failures[b]** |
|---|---|---|---|---|
| Nonoperative ($n$ = 159) | 15.1 | N/A | N/A | 0 |
| Plating ($n$ = 460) | 2.2 | 4.6 | 2.4 | 2.2 |
| Intramedullary pinning ($n$ = 152) | 2 | 6.6 | 0 | 3.9 |
| Total ($N$ = 771) | 4.8 | 5.1 | 1.8 | 2.1 |
| | (3.5–6.5) | (3.6–7.1)[c] | (1–3.2)[c] | (1.3–3.3) |

N/A = not applicable
Data are percentages with 95% confidence intervals in parentheses
[a]Any infection described as deep or superficial requiring irrigation and debridement; infections of unknown significance were not included
[b]One includes refractures
[c]Infection rates only include operatively treated fractures

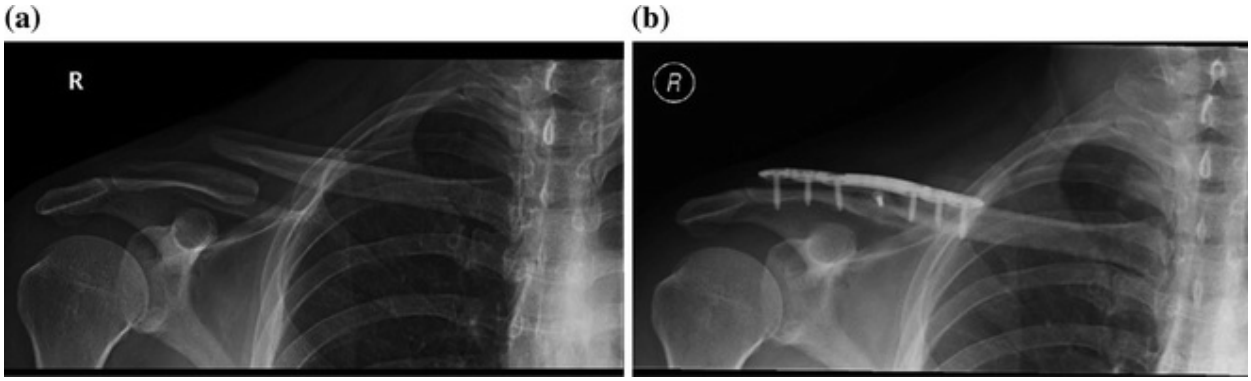



***Fig. 3.2*** **a** Preoperative radiograph of midshaft clavicle nonunion in an active 28-year-old man

fourteen months after fracture. **b** Post-operative radiograph demonstrating union of midshaft clavicle nonunion following open reduction and internal fixation with a precontoured clavicle plate. Note the solid "bridge" portion of the plate, providing extra strength at the nonunion site

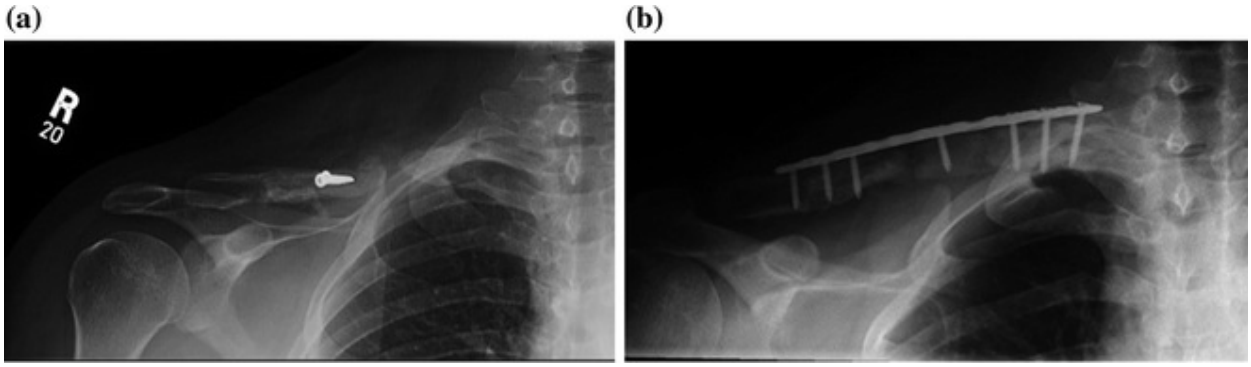



***Fig. 3.3*** **a** Preoperative radiograph of midshaft clavicle nonunion in a 39-year-old man who had failed two prior attempts at operative fixation. There is significant bony defect. **b** Post-operative radiograph of midshaft clavicle nonunion following open reduction and internal fixation with a precontoured plate and an intercalary tricortical iliac crest bone graft

Several recent studies have indicated that certain patient populations pose a greater risk of nonunion, malunion, and/or poor functional outcomes [8, 23, 25, 28]. Reported risk factors for nonunion include: a "refracture," clavicle shortening (>15–20 mm), female gender, fracture comminution, increasing fracture displacement, older age, severe initial trauma, and unstable lateral fracture (Neer type II) [2, 21, 28–32]. It is highly likely that there are other factors that increase risk of nonunion that requires further clarification, such as multiple ipsilateral rib fractures or associated scapular/glenoid fractures [14, 32–34].

## 3.2 Classification

With the Allman classification from 1967 [35], clavicle fractures can be divided into three groups; Group 1, fractures of the middle third; Group 2, fractures of the lateral third; and Group III, fractures of the medial third. These three groups can be further subdivided into nondisplaced and displaced fractures. Within Group 1, there is a further subgroup that consists of fracture comminution and one or more displaced intermediary fragments. In 1998, Robinson presented a new and more comprehensive classification for clavicle fractures [17]. This was performed following a study of 1000 consecutive patients who were treated for isolated clavicle fractures in the Orthopedic

Trauma Unit at the Royal Infirmary of Edinburgh. The age, gender, and mechanism of injury were recorded for each patient within 72 h of the injury. The anatomical site, configuration, type, and extent of comminution were also recorded for each fracture. Radiographs from twenty randomly selected fractures aided in the development of this new classification system.

## 3.3 Epidemiology

The findings of Robinson's epidemiological study estimated that in patients over 13 years of age, clavicle fractures occurred at a rate of 29.14 per 100,000 annually [9]. Prior to this, Nordqvist and Peterson had found the incidence of clavicle fractures to be 64 per 100,000 annually [26]. The mean age for clavicular fractures was 29 years in men and 45 years in women. In that series, clavicle fractures occurred most frequently in males under the age of 30 years as a result of a sports injury or a road traffic accident (RTA). The majority of fractures (89%) healed without complication when treated conservatively. Nonunion was only seen in one nondisplaced Type A fracture (Type 3A-1). The rate of delayed union and nonunion was far higher in the displaced Type B fracture cohort, with a rate of 2.7 and 4.8%, respectively. They occurred almost entirely following a displaced diaphyseal (type 2B) or displaced lateral end (type 3B) clavicular fracture. The odds ratio (OR) for delayed union or nonunion following a type 2B fracture compared with a type 2A fracture was 18 (meaning that a patient with a 2B fractures is eighteen times more likely to develop a delayed or nonunion than a patient with a 2A fracture pattern). The OR for delayed union or nonunion following a type 3B fracture compared with a type 3A fracture was seventy-five. However, due to the increased prevalence of type 2B fractures, more patients from this cohort were seen with a delayed or nonunion. High-energy injuries such as falls from a height, RTA, and direct violence indicated a greater incidence of delayed and nonunion compared with low-energy mechanisms of injury. Differences in the rate of union were not significant amongst the age or gender cohorts.

## 3.4 Rates of Nonunion in Displaced Clavicular Fractures

Nonunion is significantly more common in displaced clavicular fractures, particularly of the midshaft [36]. A direct correlation has been demonstrated between increased degrees of displacement and poor functional outcomes [18]. The study conducted by Robinson et al. [8] demonstrated that patients with a displaced, comminuted midshaft clavicular fracture had a nonunion rate of 21%.

Neer and Rowe initially reported low rates of nonunion [2, 4]. Subsequent studies have shown significantly higher rates of nonunion. In 1986, Eskola et al. reported a nonunion rate of 3% [37]. In 1997, Hill et al. reported an exponentially higher nonunion rate of 15% [21]. In 2004, Robinson et al. found an overall nonunion rate of 6.2: 8.3% of medial fractures, 4.5% of diaphyseal fractures, and 11.5% of fractures to the lateral end [8]. Similar to Hill, Zlowodzki et al. reported a nonunion rate of 15.1% in 2005 [22]. The Canadian Orthopedic Trauma Society's 2007 trial reported a rate of nonunion in the operative group of 3% compared with 14% in the nonoperative group [18]. Most recently in 2011, Kulshrestha et al. [38] conducted a study of 73 patients, aged 20–50 years, who were allocated to operative or nonoperative treatment for their displaced midshaft clavicle fractures. In the operative group, none of the 45 patients went on to nonunion. In the nonoperative group, eight patients out of twenty-eight developed nonunion which resulted in an extremely high rate of nonunion (29%) [20]. New knowledge regarding the potential rate of nonunion for this injury has come to influence management options for orthopedic surgeons who treat these fractures.

## 3.5 Possible Risk Factors for Nonunion

Murray et al. [39] sought to determine risk factors for nonunion in an adult population of patients with displaced midshaft clavicle fractures. They retrospectively reviewed 941 patients who were at least 18 years of age, who had initially received nonoperative management for their injuries from January 1994 to December 2007. No nonunions were reported in the 184 patients who were under 18 years of age, and thus, they were excluded from the study. The risk of nonunion in children was zero, and age was not a significant risk factor for nonunion in the adult population. The study population was typical of most midshaft clavicular fracture populations in that it consisted mainly of young and active men. One hundred and twenty-five of these patients went on to develop nonunion which constitutes a risk of

nonunion of 13.3% (95% confidence interval [CI], 11.3–15.6%). Using bivariate analysis, the significant factors for a higher nonunion rate were female gender, smoking, increased fracture displacement, overlap, translation, and comminution. In the multivariate analysis, the significant factors were smoking (OR, 3.76), comminution (OR, 1.75), and increased fracture displacement (OR, 1.17) (Fig. 3.4). The negative effect of smoking on fracture healing has been shown in previous studies [40–42]. This is the first time that smoking was identified as a risk factor for clavicular nonunion since earlier studies that investigated whether smoking was a risk factor showed no correlation [21, 28]. However, poorer outcomes in patients with comminution had been shown in prior studies [8, 22, 27, 40]. Murray et al. also identified comorbidities that could potentially increase the risk of nonunion which included rheumatoid disease, immunocompromise, renal failure, epilepsy, and use of drugs such as corticosteroids and those interfering with vitamin D metabolism [39]. Since many patients with a high risk of nonunion will not develop this complication, and many patients with fewer risk factors do go on to nonunion, these risk factors are best used as a guide for surgeons when making a plan for management of this injury. This knowledge would ideally improve the functional outcomes for certain patients who would benefit from surgical intervention, while simultaneously avoiding unwarranted surgery for others [39].

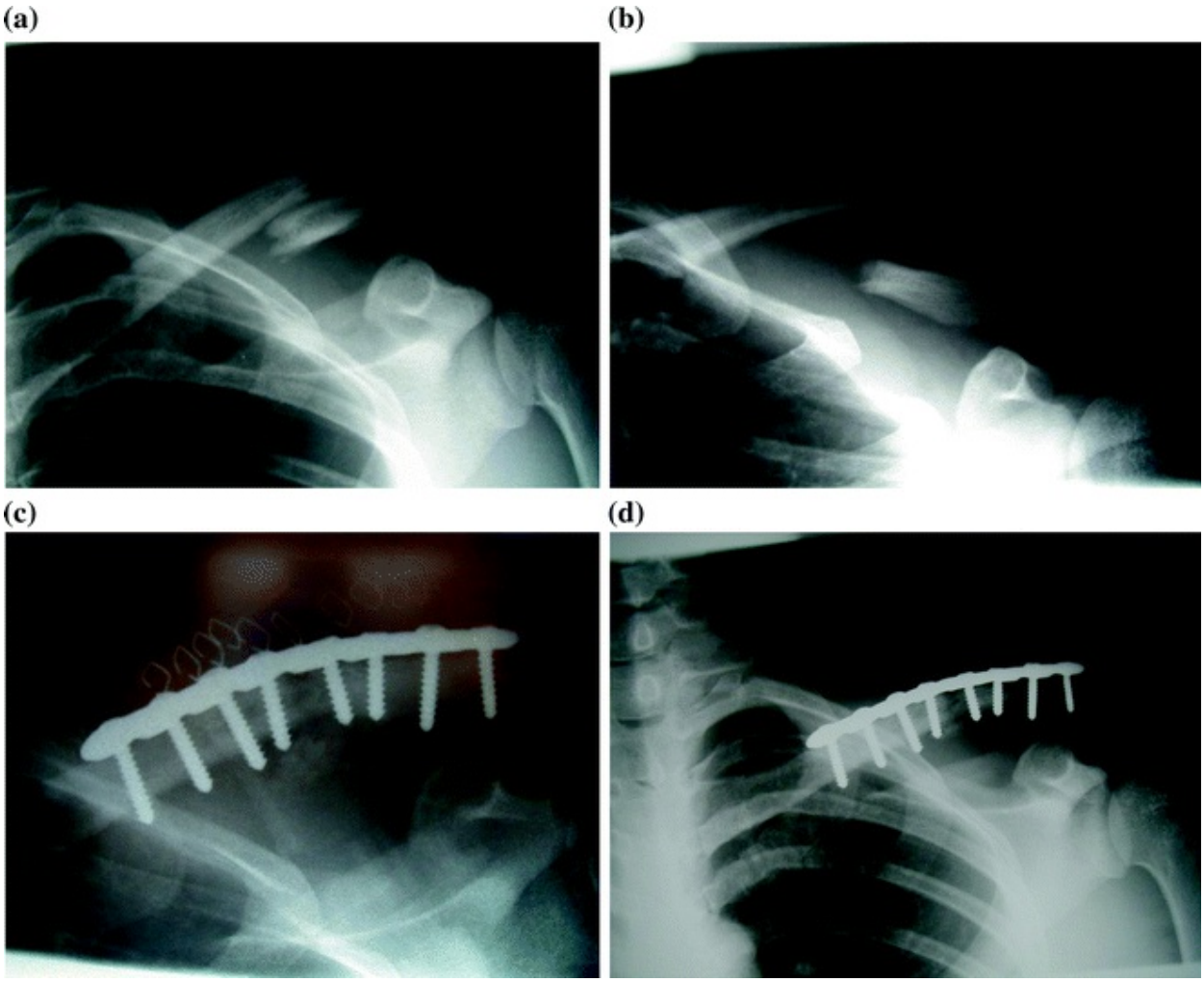



***Fig. 3.4*** **a** Preoperative radiograph of a midshaft clavicle fracture in a 25-year-old female. **b** The patient was treated nonoperatively and went on to develop a symptomatic atrophic nonunion with severe displacement. **c** Post-operative radiograph of midshaft clavicle nonunion following open reduction and internal fixation with a precontoured plate and an iliac crest bone graft. **d** Radiograph of healed midshaft clavicle fracture at final follow-up

## 3.6 Treatment Options

Nonunion is generally defined as the lack of radiographic healing at six months post-initial injury and delayed unions are typically defined when there is progression of healing, but the fracture has not achieved radiographic union at three months [7]. Symptomatic clavicular nonunion is typically painful and debilitating for active patients, causing local pain, persistent deformity, and shoulder weakness resulting in neurologic symptoms consistent with brachial plexus impingement. Treatment options vary from

nonoperative techniques that include symptomatic treatment with a sling or figure-of-eight bandage, to noninvasive techniques involving electrical stimulation or low-intensity ultrasound [5, 7, 15, 43], and surgical intervention. Asymptomatic patients with a radiographic nonunion who have full clinical function do not require surgical intervention and respond well to conservative treatment.

## 3.7 Nonoperative

Clavicle fractures have traditionally been managed nonoperatively due to studies published in the 1960s that indicated low nonunion rates [2, 4]. This treatment involves mobilizing the shoulder in a sling or figure-of-eight bandage. In a randomized, controlled trial that compared the treatment of clavicle fractures with a sling versus a figure-of-eight bandage, the functional and cosmetic outcomes were found to be identical between the two groups [44]. Recent studies have shown that the risk of nonunion following nonoperative treatment of midshaft clavicle fractures ranges from five to twenty per cent [8, 18, 21, 22]. Patients with displaced fractures are at an even greater risk of developing nonunion [8, 21, 22]. Patients who undergo primary fixation of their displaced midshaft clavicle fractures have better functional outcomes than those treated nonoperatively [18]. Additionally, patients who undergo secondary fixation due to the development of nonunion following nonoperative treatment have results that are somewhat inferior to outcome following primary fixation. [18, 45]. This would suggest that primary fixation for displaced midshaft clavicle fractures may be the preferred course of management for certain patients [18]. However, as Murray et al. [39] and McKee [7] suggest, it is important to identify a patient's risk factors for nonunion in order to assess their suitability for surgical treatment to optimize management.

## 3.8 Operative

Methods of intervention include excision [37, 46–49], intramedullary fixation [14, 50–52], and open reduction and internal fixation (ORIF) with cortical [53] or autogenous bone graft [32, 37, 40, 54–64]. Dual plating with 100% union rates has also been reported [65]. Recent studies have shown that young and active patients with symptomatic nonunion benefit from surgical

treatment. However, residual functional impairment can still occur [66]. The series presented by Robinson et al. [8] found that the highest rate of nonunion occurred in elderly female patients with diaphyseal clavicle fractures. However, due to the reduced functional demands of elderly patients, it is possible that surgery may not be required in these individuals. This is likely not the case for active and young patients who still account for many of the nonunions that occur in clavicle fracture cases. Poor function can hinder lifestyle, and for this reason, surgical intervention is usually desirable since it has been shown to significantly improve long-term shoulder function [18].

Clavicle nonunions are primarily treated with reconstructive procedures, and in rare cases, a salvage procedure may occur when there are few or no options left for a patient [67]. Salvage procedures have involved partial or total resection of the clavicle bone (claviculectomy) as well as excision of a bony prominence in order to relieve local pain caused by symptomatic nonunion.

### 3.8.1 Reconstructive Procedures

The primary surgical treatment of clavicle nonunions involves reconstructive procedures. There are a number of methods used in the treatment of clavicle nonunions, with an increasing body of literature to describe such methods and cases (Table 3.2) [14, 37, 40, 50, 54–58, 60, 62, 68, 69]. There are two main operative treatment options used to achieve clavicular union: plate fixation and intramedullary screw/pin fixation. Plates are typically fixated to the superior surface of the clavicle, but good outcomes have also been reported with compression plates fixated to the anteroinferior surface [70].

***Table 3.2*** Treatment of clavicular nonunion with various methods of operative intervention

| Study | No. of patients who achieved union | Male | Female | Age (y) (min) | Age (y) (max) | Hyper-tropic | Atrophic | Approach | Complications | H re |
|---|---|---|---|---|---|---|---|---|---|---|
| Ballmer et al. [54] (mean follow-up 8.6 y) | 35/37 (16 patients had primary operative treatment) | 22 | 15 | 14 | 54 | 13 | 24 | Decortification with plate osteosynthesis. Autogenous cancellous bone graft (24 cases), tricortical, iliac crest intercalary | 2 persistent symptomatic nonunions | 2 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | graft (9) | | |
| Bradbury et al. [55] (mean follow-up 7 y post-surgery) | 31/32 | 23 | 9 | 17 | 60 | 21 \| 10 +1 true pseudoarthrosis | | 17 AO reconstruction plate with autologous cancellous bone graft, 15 AO dynamic compression plate with autologous cancellous bone graft | none | 1 |
| Boyer and Axelrod [68] | 7/7 | 5 | 2 | 17 | 55 | 0 | 7 | Excision with 3.5-mm pelvic construction plate or dynamic compression plate, lag screw for interfragmentary compression, cancellous bone graft | none | N |
| Davids et al. [56] | 14/14 | 8 | 6 | 19 | 74 | 5 | 9 | AO reconstruction plate with iliac crest bone graft | 1 patient had refracture 1 year post-hardware removal | 6 p s |
| Ebraheim et al. [57] (mean follow-up 12.9 mo) | 15/16 | 9 | 7 | 15 | 52 | 11 | 5 | ORIF with reconstruction or dynamic plate, autogenous bone graft in 14 cases, double plating in 3 cases | 2 cases persistent mild pain, 1 hardware failure (healed post-revision), 1 hardware failure (persistent nonunion, pain subsided) | 1 |
| Endrizzi et al. [58] | 42/47 (1 patient LTF, 1 deceased) | 33 | 14 | 12 | 68 | N/A | N/A | ORIF-27 curved pelvic reconstruction plates, 16 straight pelvic reconstruction plates, 4 straight dynamic compression | 3 revision surgeries due to implant loosening | 1 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | plates<br>14 cases demineralized bone matrix, 3 autogenous iliac crest graft, 1 resected rib graft | | |
| Eskola et al. [37] | 20/22<br>+2 patients treated with resection | 16 | 8 | 22 | 79 | – | – | Twenty-one cancellous bone grafts were executed, with eighteen of these involving rigid plate fixations and one Kirschner pin fixation. In two cases, bone grafting was used solely. In one case, only plate fixation was performed | 4 cases of clavicle shortening | – |
| Kabak et al. [40] | 31/33 | 19 | 14 | 19 | 66 | 8 | 25 | ORIF with dynamic compression plate (DCP) or low-contact (LC-DCP) and autogenous corticancellous chips or sculptured graft | 1 incomplete brachial plexus palsy, 2 revision surgeries went on to union | 5<br>2<br>D |
| Laursen and Dossing [60]<br>(mean follow-up 24 mo) | 16/16 | 10 | 6 | 17 | 62 | 5 | 11 | Compression plate fixation with autologous cancellous bone graft | 1 revision due to screw loosening, 1 revision due to fracture of plate | 4 |
| Manske and Szabo [62] | 10/10 | 7 | 3 | 16 | 60 | – | – | Compression plate and iliac crest bone graft | None | |
| Olsen et al. [69] | 15/16 | 10 | 6 | 13 | 55 | 6 \|9<br>1 failed | | ORIF with 3.5-mm plate and | 1 persistent nonunion, 2 | Ir<br>w |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | osteosynthesis | | autologous iliac crest bone graft | patients “fair” outcome | p n h r d l o |
| *IM pinning* | | | | | | | | | | |
| Capicotto et al. [14] (mean follow-up 4 y) | 14/14 | – | – | 18 | 62 | – | – | Steinman pin fixation and only iliac crest bone graft | None | 1 |
| Enneking et al. [50] (mean follow-up 40 mo) | 13/14 | 10 | 4 | 19 | 83 | 3 | 11 | ORIF with Rushpin fixation and iliac crest bone graft | None (aside from 1 case of nonunion) | 3 |

*ORIF* open reduction and internal fixation
*AO* Arbeitsgemeinschaft für Osteosynthesefragen

## 3.9 Open Reduction Internal Fixation with Autogenous Bone Graft

### 3.9.1 Surgical Method

ORIF with a compression plate and iliac crest bone graft is considered the gold standard for treatment of clavicle nonunions [1, 7, 71]. Due to the clavicle’s close proximity to the subclavian vascular bundle and brachial plexus, surgical technique must be precise so that neurovascular injury is avoided [29]. This surgical process involves carefully dividing and preserving the myofascial layer and identifying the superior surface of the clavicle. Once this is accomplished, the two ends of the nonunion must be identified and mobilized. Derotation of the (usually anteriorly rotated) distal fragment typically allows the superior surface to be exposed which enables plate fixation on the flat superior surface. The proximal and distal ends of the nonunion are then reduced. If there is excess callous on the superior surface, then it should be ronguered away to create a flat superior surface that

facilitates placement of the plate on the bone. This excess callous should be saved, morcellized, and later inserted into the nonunion site. The use of a lag screw or small K-wire to hold the reduction while a plate is applied to the surface can be very beneficial [7]. Due to the complexity of the bone's structure and the multidirectional biomechanical forces that act on the nonunion, at least three screws should be placed on both sides of the fracture site to stabilize the bone [57, 72]. In a series in which clavicular nonunion were treated with short plates (4 hole semi-tubular), there was a high risk of failure reported [73]. In hypertrophic nonunions, the residual autograft from the local bone should be applied to the nonunion site followed by a standard closure. In cases of atrophic nonunion, an autograft from the iliac crest should be applied to the fracture site (see Fig. 3.2) [7]. If there is significant bone loss with shortening, then it has been proposed that an intercalary graft be used (Fig. 3.3) [7, 59]. The goal in treating these fractures is to restore the length equal to the uninjured contralateral size. Preoperative radiographic and clinical evaluations should be conducted to determine the length of the uninjured side. In the instance of clavicle shortening, it is generally accepted if the bone is shortened by ≤1 cm. If there is significant bone loss with shortening, then it has been proposed that an intercalary graft such as a tricortical autogenous iliac crest bone graft from is used from the contralateral hip. Previously, it has been suggested that low-contact dynamic compression plates are a superior choice for fixation of clavicular nonunion due to its increased ability to be contoured and preserve blood supply to the underlying bone fragments due the plate's structured undersurface [72]. There have been numerous reports of favourable outcomes and high rates of success (up to 100%) with this method of intervention [54, 58, 60, 69, 74, 75]. It is important that surgeons make certain that an implant of the correct size and length is used in order to aid in successful union of the fracture. The availability of precontoured plates designed specifically for the clavicle, which have been shown to be biomechanically equivalent to compression plates, makes them the implant of choice in this setting [7, 30].

### 3.9.2 An Innovative Method

In 1997, Boyer and Axelrod [68] reported on an innovative method for treating atrophic nonunion of the clavicle. They bevelled the clavicle using cuts at 45° to the long axis of the bone using an oscillating saw to minimize shortening and provide a flat surface of healthy viable bone. Each patient's

nonunion was fixed with a dynamic compression plate or, in one case, a pelvic reconstruction plate. A lag screw provided intra-fragmentary compression in all seven cases and the nonunion site was grafted with autogenous cancellous bone. All patients achieved union between six and 12 weeks with full pain relief and return to normal activity levels. The authors determined that each patient was very satisfied with their result and the slight narrowing (<2 cm) of the shoulder's width did not cause the patients to be dissatisfied with the cosmetic result of the intervention.

### 3.9.3 Literature Review

Olsen et al. [69] reported on sixteen consecutive cases of clavicular nonunion that were initially treated conservatively. Each patient was treated with ORIF and iliac crest bone grafting. Twelve of the patients had an excellent result with the remaining two receiving a good grade, one fair and one failure resulting in persistent nonunion. This patient had initially been treated with a resection of the medial fragment.

In a series with long follow-up from the time of surgical intervention (7 years), thirty-one out of thirty-two nonunions were treated successfully [55]. The majority of these clavicle nonunions were the result of high-energy traumas. Fifteen patients were treated with dynamic compression plates and autologous bone graft, and seventeen were treated with reconstruction plates and autologous cancellous bone graft. Six compression plates and seven reconstruction plates were removed with the authors recommending reconstruction plates to be used in treatment of this injury due to its ability to be contoured more easily to the complex shape of the clavicle bone. In a series comparing the use of dynamic compression plating (DCP) and low-contact dynamic compression plating (LC-DCP), union was obtained in all cases where a LC-DCP plate was used compared with 87.5% with the DCP plate [40]. Better functional outcomes were obtained in patients who were treated with a LC-CDP plate. In a series of eight patients with symptomatic nonunion who were treated with surgical intervention, all eight patients achieved clinical and radiographic union [61]. Patients were treated using a 3.5-mm low-contact dynamic compression plate (LC-DCP) and an autologous iliac crest graft. Despite a relatively short rate of follow-up (average 8 months), the authors stated that all the patients were highly satisfied with their treatment.

In a retrospective review of twenty-four nonunions between 1994 and

2001 [75], twenty-two out of twenty-four nonunions eventually healed and had favourable outcomes. This series was treated with operative fixation using a DCP or reconstruction plate along with autogenous bone grafting. The time from injury to operation ranged from three months to 29 months with the mean time being 10.2 months. Post-operative follow-up ranged from six to 74 months with a mean follow-up of 42.1 months. Considering the average length of follow-up, this series provides some indication of long-term success of operative treatment for this injury.

In a series of fourteen patients with delayed and nonunion of midshaft clavicle fractures who were treated with a reconstruction plate and iliac crest bone grafting, all patients achieved radiographic bony union at 10–30 weeks post-surgery with excellent results [56]. This study had the benefit of a long period of follow-up with a mean of 60 months (16–101 months). In a series of ten consecutive patients who received operative treatment for their midshaft clavicle nonunions, all ten patients achieved union at ten weeks post-operatively and had a full and painless range of motion [62].

Eskola et al. reported on an adult population (aged 22–79 years) with an average follow-up of three years. Twenty-four patients were initially treated nonoperatively, eventually resulting in nonunion, and then subsequently received surgical intervention [37]. Twenty-one cancellous bone grafts were performed, with eighteen of these involving rigid plate fixation and one Kirschner pin fixation. In two cases, bone grafting was used solely. In one case, only plate fixation was performed. In two cases, the medial end of the clavicle was resected. For the twenty-two cases that were treated with fixation and bone grafting, twenty achieved bony union of the clavicle fracture. The study also found that the clavicle was shortened in four cases which resulted in some weakness and decreased range of motion. This is consistent with other studies have shown decreasing functional outcomes with increasing deformity.

It is important to remember these studies were performed before the routine availability of precontoured plates: Their improved biomechanical strength (compared to reconstruction plates) and superior fit (compared to straight compression plates) make them the implant of choice for the treatment of clavicular nonunion.

### 3.9.4 Multicenter, Randomized Controlled Trials

There have been two large multicenter, randomized controlled trials aimed at

comparing the results of open reduction and plate fixation with nonoperative treatment for displaced midshaft clavicle fractures (Table 3.3). In 2007, the Canadian Orthopedic Trauma Society [18] conducted a series involving 132 patients with this injury. Final follow-up was available for sixty-two patients allocated to operative treatment and forty-nine patients allocated to nonoperative treatment. Outcomes were measured using standard clinical follow-up, the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, the Constant questionnaire, and plain radiographs. DASH and Constant scores were significantly better in the operative group at each time point. The rate of nonunion in the operative group was 3%, and in the nonoperative group, it was 14%. At one-year follow-up, the patients in the operative group were more likely to be satisfied with the appearance of the shoulder ($P = 0.001$) and the shoulder in general ($P = 0.002$). Hardware removal was the most common intervention for the surgical group.

***Table 3.3*** Multicenter randomized, controlled trials comparing open reduction and internal fixation with nonoperative treatment of displaced midshaft clavicle fractures

| Study | Total number of patients | Operative group (n) | Nonoperative group (n) | Mean age (years) | Surgical approach | Primary outcome measures | Rate of nonunion (%) | Follow up (% |
|---|---|---|---|---|---|---|---|---|
| Robinson et al. [76] | 200 | 95 | 105 | 32 | ORIF using locking clavicle plate | Union confirmed by 3D computer tomography (CT). Functional assessment measured through Short Form (SF-12), the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, the constant questionnaire, and shoulder motion | ORIF: 1.2<br>Nonoperative: 17<br>(13 nonunions in nonoperative group underwent surgical intervention) | 89 |
| Canadian Orthopedic | 131 | 62 | 49 | 33.5 | ORIF using | Standard clinical | ORIF: 3<br>Nonoperative: | 84 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Trauma Society [18] | | | | | small fragment plate | follow-up, the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, the constant questionnaire and plain radiographs | 14.3 (1 patient allocated to the operative group declined surgery and nonunion persisted at 1 y follow-up but was still included in the operative group due to the “intention to treat” principle) | |

*ORIF* open reduction and internal fixation

In 2013, Robinson et al. [76] reported on 200 patients who had an isolated displaced fracture of the middle third of the clavicle. At two weeks post-injury, 95 patients were allocated to surgery and 105 to nonoperative treatment. Union was confirmed by 3D computer tomography (CT). Functional assessment was measured through Short Form (SF-12), the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, the Constant questionnaire, and shoulder motion. There was a nonunion rate of 1% in the operative group and a significantly higher rate of 17% in the nonoperative group. Thirteen patients with nonunion in the nonoperative group underwent surgical intervention and eight patients experienced delayed union, with union occurring between 6 and 12 months post-injury. The ORIF group had better DASH scores at 3- and 12-month follow-up. The SF-12 physical and mental scores did not differ between groups. The mean patient age was 33.5 and 32, respectively, in these studies, and follow-up rates were 84 and 89%. Both of these studies indicated that primary operative intervention for displaced midshaft clavicle fractures decreases the rate of nonunion.

## 3.10 ORIF Without Bone Grafting

Other studies have suggested that autogenous bone grafting may not be

necessary in the treatment of clavicular nonunion. Baker and Mullett [77] reported on a series of fifteen patients who achieved clinical and radiographic union following fixation with precontoured locking plates. One patient underwent hardware removal due to local discomfort but the remaining patients experienced favourable outcomes. Ramoutar et al. [78] reported a series of eleven patients who also achieved union without bone graft. In this series, the patients were fixated with fragment decortication and compression plating.

## 3.11 ORIF with Allograft Bone Substitute

Iliac crest bone grafting has been shown to be very effective for nonunion healing, although various studies have shown that significant complications can occur. These complications can include temporary or prolonged pain at the harvest site, pelvic fracture, infection, persistent drainage, and sensory disturbances [79–86]. In an effort to reduce these complications, certain alternatives to autogenous bone grafting have been proposed. Demineralized bone matrix (DBM) has been shown to be effective in the treatment of humeral and tibial nonunions [82, 84]. However, the literature regarding its use and efficacy for treatment of clavicle nonunions is limited. Endrizzi et al. [58] reported on a series of forty-seven patients who were treated for clavicular nonunion. Fourteen of these patients received DBM, and although they reported a high rate of union (93%), the study did not focus solely on this treatment type. In a recent study, conducted by Riggenbach et al. in 2011 [49], nineteen patients were treated with ORIF and allograft bone substitute. Twelve patients received platelet-rich plasma (PRP) with DBM, and the other seven received allograft corticocancellous chips. Sixteen of the patients achieved bony union, while three required revision due to failure, including two “catastrophic failures.” Two of the three persistent nonunions went on to heal fully. A final clinical healing rate of 95% in this study indicates that DBM may be a viable treatment option for clavicle nonunion. It is interesting to note that the three patients who required revision were smokers and that nonunion was more common in smokers ($P = 0.08$) compared with patients who did not smoke. This could have potential implications for surgeons who are treating smokers with clavicular nonunion. Iliac crest bone graft may be a better option for these patients.

## 3.12 Vascularized Bone Graft Reconstruction

Momberger et al. [87] presented three patients who had each experienced a completely displaced midshaft clavicle fracture. After an average of 3.7 procedures, each clavicle had failed to unite and was characterized by segmental bone loss. Each patient was treated with vascularized fibula transfer. At an average follow-up of 2.8 years, each clavicle had achieved bony union as well as decreased pain and improved shoulder function. This method of intervention could be considered following repeated failure of clavicle fractures to unite using traditional surgical methods, especially when segmental bone loss is present.

---

## 3.13 Intramedullary Pin Fixation

Intramedullary (IM) pinning with autogenous bone grafting has several potential advantages as a treatment option for patients with clavicle nonunion. A smaller initial incision, less tissue dissection, and less periosteal stripping are necessary [50]. A small incision for removal of the pin under local anaesthesia (if necessary) results in improved cosmetic appearance and a simpler and less invasive method of hardware removal [50, 88]. It is also likely that IM pinning will cause decreased hardware irritation [7]. Boehme et al. [88] reported on twenty-one patients (from a series of fifty) who were treated with ORIF using a modified Hagie intramedullary pin and autogenous bone grafting. Twenty of the patients achieved union. Enneking et al. [50] used Rush pin fixation along with autogenous bone grafting in a series of fourteen patients, with thirteen of these nonunions going on to heal with good results. Three patients required hardware removal. Capicotto et al. [14] reviewed fourteen patients with clavicle nonunions caused mainly by high-velocity injuries. Three of these patients were treated with plating that failed, and in one case, the fracture was pathological through irradiated bone. These nonunions were treated with intramedullary Steinman pin fixation along with iliac crest bone graft. All fourteen nonunions went on to achieve bony union and had their hardware removed between 7 and 24 weeks post-surgery. Two refractures occurred through osteopenic bone, but this was the only complication of significance.

Despite the increased ease of hardware removal with this technique, IM fixation is weaker biomechanically [7] and there have been reported

complications such as pin migration and breakage that should be taken into consideration [89–91]. McKee [7] recommended that a randomized, prospective trial be conducted that compares plate and IM fixation for the treatment of displaced midshaft clavicle fractures in order to better assess their use in relation to this injury: two such studies are currently underway. Wu et al. [63] conducted a retrospective review comparing plate with IM fixation in thirty-three patients with midshaft asceptic clavicle nonunion. They found that nine of eleven patients treated with plate fixation healed, while sixteen of eighteen patients treated with IM fixation healed. However, a more comprehensive study with a larger patient population is required.

### 3.13.1 Resection

Resection of the clavicle is a salvage technique used to alleviate pain and discomfort caused by severe or recalcitrant nonunion in low demand patients. Although absolute indications for claviculectomy are rare, the primary indication is multiple failed conventional surgeries and some relative indications have included clavicular tumour, vascular injury, and associated unipolar or bipolar dislocation [48, 92–98].

Historically, Abbot and Lucas stated that as long as the medial and distal thirds of the clavicle were left intact, the middle third of the clavicle could be removed. In 1968, Rowe specified that for patients who had had several unsuccessful attempts at bone grafts and were “sufficiently disabled,” resection of the clavicle could result in “surprisingly good function and cosmetic appearance” [4]. In 1986, Wood [99] reported on five cases of total claviculectomy and determined that the procedure resulted in excellent functional outcomes. More recently, Krishnan et al. [100] conducted a retrospective chart review of six cases of unilateral claviculectomy with a mean follow-up of 5.7 years. They found that in a relatively young cohort of patients, there was good restoration of range of motion, minimal strength deficits, and improved pain relief. They report that in situations where restoration of clavicular anatomy is impossible, total claviculectomy can result in good functional outcomes. However, it is important to note that three of the six patients had a major complication as well as four patients experiencing infection (two deep and two superficial).

Other studies have raised questions about resection procedures used to treat clavicle nonunion. Connolly and Dehne [46] reported that resection can result in delayed problems such as thoracic outlet syndrome and should

therefore be avoided. In 1992, Rockwood and Wirth argued for the preservation of the clavicle due to low patient satisfaction following total claviculectomy [53]. In 2007, Wessel and Schapp [101] reported on six cases of total claviculectomy. Good results were seen in one chronic osteitis case and two malignancy cases. Three post-traumatic cases had poor results due to persistent pain. All six patients regained full range of motion of the shoulder. No final conclusions were drawn due to the small sample size and conflicting outcomes. Overall, the literature on this method of intervention for clavicle nonunion is sparse, with small reported sample populations. Due to limited knowledge on the effectiveness of this procedure, the importance of the clavicle for upper extremity function and stability, and the availability of modern methods of intervention, total claviculectomy should be regarded solely as a salvage procedure [7].

## 3.14 Conclusion

Nonunion is a complication of clavicular fractures that typically results in pain and loss of shoulder function and mobility. Nonoperative treatment, especially in displaced midshaft clavicle fractures, can result in nonunion that causes increased deficits in shoulder function and pain. Due to the significant rate of nonunion in displaced fractures in young and active adults, operative intervention, which has been shown to have high success rates for pain relief and bony union, should be explored as an option in cases where surgeons believe the patient would benefit. If nonunion does occur, plate fixation with autogenous bone grafting is a reliable operation with a high success rate and low complication rate.

## References


1. Post M. Current concepts in the treatment of fractures of the clavicle. Clin Orthop Relat Res. 1989;245:89–101.

2. Neer CS 2nd. Nonunion of the clavicle. JAMA. 1960;172:1006–11. [Crossref]

3. Postacchini F, Gumina S, De Santis P, Albo F. Epidemiology of clavicle fractures. J Shoulder Elbow Surg. 2002;11(5):452–6. [Crossref][PubMed]

4. Rowe CR. An atlas of anatomy and treatment of midclavicular fractures. Clin Orthop Relat Res. 1968;58:29–42.
[Crossref][PubMed]

5. Crenshaw AH. Campbell's operative orthopaedics. St Louis: Mosby; 1996.

6. Goldberg JA, Bruce WJ, Sonnabend DH, Walsh WR. Type 2 fractures of the distal clavicle: a new surgical technique. J Shoulder Elbow Surg. 1997;6(4):380–2.
[Crossref][PubMed]

7. McKee MD. Fractures of the clavicle. In: Bucholz RU, Heckman JD, Court-Brown CM, Tornetta P, editors. Rockwood and Green's fractures in adults. 7th ed. Philadelphia: Lippincott Williams and Wilkins; 2010. p. 1106–43.

8. Robinson CM, Court-Brown CM, McQueen MM, Wakefield AE. Estimating the risk of nonunion following nonoperative treatment of a clavicular fracture. J Bone Joint Surg Am. 2004;86-a(7):1359–1365.

9. Rockwood CA. Fractures of the outer clavicle in children and adults. J Bone Joint Surg Br. 1982;64B:642.

10. Webber MC, Haines JF. The treatment of lateral clavicle fractures. Injury. 2000;31(3):175–9.
[Crossref][PubMed]

11. Robinson CM, Cairns DA. Primary nonoperative treatment of displaced lateral fractures of the clavicle. J Bone Joint Surg Am. 2004;86-a(4):778–82.

12. Seo GS, Aoki J, Karakida O, Sone S. Nonunion of a medial clavicular fracture following radical neck dissection: MRI diagnosis. Orthopedics. 1999;22(10):985–6.
[PubMed]

13. Throckmorton T, Kuhn JE. Fractures of the medial end of the clavicle. J Shoulder Elbow Surg. 2007;16(1):49–54.
[Crossref][PubMed]

14. Capicotto PN, Heiple KG, Wilbur JH. Midshaft clavicle nonunions treated with intramedullary Steinman pin fixation and onlay bone graft. J Orthop Trauma. 1994;8(2):88–93.
[Crossref][PubMed]

15. Craig EV. Fractures of the clavicle. In: Rockwood CA, Green DP, Bucholz RW, Heckman JD, editors. Rockwood and Green's fractures in adults. Philadelphia: Lippincott-Raven; 1996. p. 1109–61.

16. Neer CS. Fractures of the clavicle. In: Rockwood CA, Green DP, editors. Fractures in adults. 2nd ed. Philadelphia: Lippincott; 1984. p. 707–13.

17. Robinson CM. Fractures of the clavicle in the adult. Epidemiology and classification. J Bone Joint Surg Br. 1998;80(3):476–84.
[Crossref][PubMed]

18. Canadian Orthopaedic Trauma Society. Nonoperative treatment compared with plate fixation of

displaced midshaft clavicular fractures. A multicenter, randomized clinical trial. J Bone Joint Surg Am. 2007;89(1):1–10.
[Crossref]

19. Neviaser JS. The treatment of fractures of the clavicle. Surg Clin N Am. 1963;43:1555–63.
[Crossref][PubMed]

20. Ghormley RK, Black JR, Cherry JH. Ununited fractures of the clavicle. Am J Surg. 51(2):343–349.

21. Hill JM, McGuire MH, Crosby LA. Closed treatment of displaced middle-third fractures of the clavicle gives poor results. J Bone Joint Surg Br. 1997;79(4):537–9.
[Crossref][PubMed]

22. Zlowodzki M, Zelle BA, Cole PA, Jeray K, McKee MD. Treatment of acute midshaft clavicle fractures: systematic review of 2144 fractures: on behalf of the evidence-based orthopaedic Trauma working group. J Orthop Trauma. 2005;19(7):504–7.
[Crossref][PubMed]

23. Brinker MR, Edwards TB, O'Connor DP. Estimating the risk of nonunion following nonoperative treatment of a clavicular fracture. J Bone Joint Surg Am. 2005;87(3):676–7; author reply 7.

24. Edwards DJ, Kavanagh TG, Flannery MC. Fractures of the distal clavicle: a case for fixation. Injury. 1992;23(1):44–6.
[Crossref][PubMed]

25. McKee MD, Pedersen EM, Jones C, Stephen DJ, Kreder HJ, Schemitsch EH, et al. Deficits following nonoperative treatment of displaced midshaft clavicular fractures. J Bone Joint Surg Am. 2006;88(1):35–40.
[PubMed]

26. Nordqvist A, Petersson CJ, Redlund-Johnell I. Mid-clavicle fractures in adults: end result study after conservative treatment. J Orthop Trauma. 1998;12(8):572–6.
[Crossref][PubMed]

27. McKee MD, Schemitsch EH, Stephen DJ, Kreder HJ, Yoo D, Harrington J. Functional outcome following clavicle fractures in polytrauma patients. J Trauma Acute Care Surg. 1999;47(3):616.

28. Nowak J, Holgersson M, Larsson S. Can we predict long-term sequelae after fractures of the clavicle based on initial findings? A prospective study with nine to ten years of follow-up. J Shoulder Elbow Surg. 2004;13(5):479–86.
[Crossref][PubMed]

29. Ebraheim NA, Xu R, Ahmed M, Frogament AD. The anatomic relation of the clavicle to the subclavian neurovascular bundle. Orthop Int Ed. 1987;5:279–81.

30. Martetschlager F, Gaskill TR, Millett PJ. Management of clavicle nonunion and malunion. J Shoulder Elbow Surg. 2013;22(6):862–8.
[Crossref][PubMed]

31. McKee MD, Wild LM, Schemitsch EH. Midshaft malunions of the clavicle. J Bone Joint Surg

Am. 2003;85-a(5):790–797.

32. Wilkins RM, Johnston RM. Ununited fractures of the clavicle. J Bone Joint Surg Am. 1983;65(6):773–8.
[Crossref][PubMed]

33. Stanley D, Norris SH. Recovery following fractures of the clavicle treated conservatively. Injury. 1988;19(3):162–4.
[Crossref][PubMed]

34. White RR, Anson PS, Kristiansen T, Healy W. Adult clavicle fractures: relationship between mechanism of injury and healing. Orthop Trans. 1989;13:514–5.

35. Allman FL Jr. Fractures and ligamentous injuries of the clavicle and its articulation. J Bone Joint Surg Am. 1967;49(4):774–84.
[Crossref][PubMed]

36. Wick M, Muller EJ, Kollig E, Muhr G. Midshaft fractures of the clavicle with a shortening of more than 2 cm predispose to nonunion. Arch Orthop Trauma Surg. 2001;121(4):207–11.
[Crossref][PubMed]

37. Eskola A, Vainionpää S, Myllynen P, Pätiälä H, Rokkanen P. Outcome of clavicular fracture in 89 patients. Arch Orthop Trauma Surg. 1986;105(6):337–8.
[Crossref][PubMed]

38. Kulshrestha V, Roy T, Audige L. Operative versus nonoperative management of displaced midshaft clavicle fractures: a prospective cohort study. J Orthop Trauma. 2011;25(1):31–8.
[Crossref][PubMed]

39. Murray IR, Foster CJ, Eros A, Robinson CM. Risk factors for nonunion after nonoperative treatment of displaced midshaft fractures of the clavicle. J Bone Joint Surg Am. 2013;95(13):1153–8.
[Crossref][PubMed]

40. Kabak S, Halici M, Tuncel M, Avsarogullari L, Karaoglu S. Treatment of midclavicular nonunion: comparison of dynamic compression plating and low-contact dynamic compression plating techniques. J Shoulder Elbow Surg. 2004;13(4):396–403.
[Crossref][PubMed]

41. Moghaddam A, Zimmermann G, Hammer K, Bruckner T, Grutzner PA, von Recum J. Cigarette smoking influences the clinical and occupational outcome of patients with tibial shaft fractures. Injury. 2011;42(12):1435–42.
[Crossref][PubMed]

42. Gullihorn L, Karpman R, Lippiello L. Differential effects of nicotine and smoke condensate on bone cell metabolic activity. J Orthop Trauma. 2005;19(1):17–22.
[Crossref][PubMed]

43. Brighton CT, Pollack SR. Treatment of recalcitrant non-union with a capacitively coupled electrical field. A preliminary report. J Bone Joint Surg Am. 1985;67(4):577–85.
[Crossref][PubMed]

44. Andersen K, Jensen PO, Lauritzen J. Treatment of clavicular fractures. figure-of-eight bandage versus a simple sling. Acta Orthop Scand. 1987;58(1):71–4.
[Crossref][PubMed]

45. Potter JM, Jones C, Wild LM, Schemitsch EH, McKee MD. Does delay matter? The restoration of objectively measured shoulder strength and patient-oriented outcome after immediate fixation versus delayed reconstruction of displaced midshaft fractures of the clavicle. J Shoulder Elbow Surg. 2007;16(5):514–8.
[Crossref][PubMed]

46. Connolly JF, Dehne R. Nonunion of the clavicle and thoracic outlet syndrome. J Trauma. 1989;29(8):1127–1132; discussion 32-3.

47. Edvardsen P, Odegard O. Treatment of posttraumatic clavicular pseudarthrosis. Acta Orthop Scand. 1977;48(5):456–7.
[Crossref][PubMed]

48. Middleton SB, Foley SJ, Foy MA. Partial excision of the clavicle for nonunion in National Hunt Jockeys. J Bone Joint Surg Br. 1995;77(5):778–80.
[PubMed]

49. Riggenbach MD, Jones GL, Bishop JY. Open reduction and internal fixation of clavicular nonunions with allograft bone substitute. Int J Shoulder Surg. 2011;5(3):61–7.
[Crossref][PubMed][PubMedCentral]

50. Enneking TJ, Hartlief MT, Fontijne WP. Rushpin fixation for midshaft clavicular nonunions: good results in 13/14 cases. Acta Orthop Scand. 1999;70(5):514–6.
[Crossref][PubMed]

51. Sakellarides H. Pseudoarthrosis of the clavicle: a report of 20 cases. J Bone Joint Surg Am. 1961;43A:130–8.
[Crossref]

52. Taylor AR. Nonunion of fractures of the clavicle: a review of 31 cases. J Bone Joint Surg Br. 1969;51-B:568–9.

53. Rockwood CA, Wirth MA. Don't throw away the clavicle. Orthop Trans. 1992;16:763.

54. Ballmer FT, Lambert SM, Hertel R. Decortication and plate osteosynthesis for nonunion of the clavicle. J Shoulder Elbow Surg. 1998;7(6):581–5.
[Crossref][PubMed]

55. Bradbury N, Hutchinson J, Hahn D, Colton CL. Clavicular nonunion. 31/32 healed after plate fixation and bone grafting. Acta Orthop Scand. 1996;67(4):367–70.
[Crossref][PubMed]

56. Davids PH, Luitse JS, Strating RP, van der Hart CP. Operative treatment for delayed union and nonunion of midshaft clavicular fractures: AO reconstruction plate fixation and early mobilization. J Trauma. 1996;40(6):985–6.
[Crossref][PubMed]

57. Ebraheim NA, Mekhail AO, Darwich M. Open reduction and internal fixation with bone grafting

of clavicular nonunion. J Trauma. 1997;42(4):701–4.
[Crossref][PubMed]

58. Endrizzi DP, White RR, Babikian GM, Old AB. Nonunion of the clavicle treated with plate fixation: a review of forty-seven consecutive cases. J Shoulder Elbow Surg. 2008;17(6):951–3.
[Crossref][PubMed]

59. Jupiter JB, Leffert RD. Non-union of the clavicle. Associated complications and surgical management. J Bone Joint Surg Am. 1987;69(5):753–60.
[Crossref][PubMed]

60. Laursen MB, Dossing KV. Clavicular nonunions treated with compression plate fixation and cancellous bone grafting: the functional outcome. J Shoulder Elbow Surg. 1999;8(5):410–3.
[Crossref][PubMed]

61. Leupin S, Jupiter JB. LC-DC plating with bone graft in posttraumatic nonunions in the middle third of the clavicle. Swiss Surg. 1998;4(2):89–94.
[PubMed]

62. Manske DJ, Szabo RM. The operative treatment of mid-shaft clavicular non-unions. J Bone Joint Surg Am. 1985;67(9):1367–71.
[Crossref][PubMed]

63. Wu CC, Shih CH, Chen WJ, Tai CL. Treatment of clavicular aseptic nonunion: comparison of plating and intramedullary nailing techniques. J Trauma. 1998;45(3):512–6.
[Crossref][PubMed]

64. Jupiter JB, Ring D. Fractures of the clavicle. In: Iannotti JP, Williams GR, editors. Disorders of the shoulder: diagnosis and management. Philadelphia: Lippincott Williams & Wilkins; 1999.

65. Sadiq S, Waseem M, Peravalli B, Doyle J, Dunningham T, Muddu BN. Single or double plating for nonunion of the clavicle. Acta Orthop Belg. 2001;67(4):354–60.
[PubMed]

66. Rosenberg N, Neumann L, Wallace AW. Functional outcome of surgical treatment of symptomatic nonunion and malunion of midshaft clavicle fractures. J Shoulder Elbow Surg. 2007;16(5):510–3.
[Crossref][PubMed]

67. Simpson NS, Jupiter JB. Clavicular nonunion and malunion: evaluation and surgical management. J Amer Acad Orthop Surg. 1996;4(1):1–8.
[Crossref]

68. Boyer MI, Axelrod TS. Atrophic nonunion of the clavicle: treatment by compression plate, lag-screw fixation and bone graft. J Bone Joint Surg Br. 1997;79(2):301–3.
[Crossref][PubMed]

69. Olsen BS, Vaesel MT, Sojbjerg JO. Treatment of midshaft clavicular nonunion with plate fixation and autologous bone grafting. J Shoulder Elbow Surg. 1995;4(5):337–44.
[Crossref][PubMed]

70.

Stufkens SA, Kloen P. Treatment of midshaft clavicular delayed and non-unions with anteroinferior locking compression plating. Arch Orthop Trauma Surg. 2010;130(2):159–64. [Crossref][PubMed]

71. Pyper JB. Non-union of fractures of the clavicle. Injury. 1978;9(4):268–70. [Crossref][PubMed]

72. Mullaji AB, Jupiter JB. Low-contact dynamic compression plating of the clavicle. Injury. 1994;25(1):41–5. [Crossref][PubMed]

73. Pedersen M, Poulsen KA, Thomsen F, Kristiansen B. Operative treatment of clavicular nonunion. Acta Orthop Belg. 1994;60(3):303–6. [PubMed]

74. Khan SA, Shamshery P, Gupta V, Trikha V, Varshney MK, Kumar A. Locking compression plate in long standing clavicular nonunions with poor bone stock. J Trauma. 2008;64(2):439–41. [Crossref][PubMed]

75. O'Connor D, Kutty S, McCabe JP. Long-term functional outcome assessment of plate fixation and autogenous bone grafting for clavicular non-union. Injury. 2004;35(6):575–9. [Crossref][PubMed]

76. Robinson CM, Goudie EB, Murray IR, Jenkins PJ, Ahktar MA, Read EO, et al. Open reduction and plate fixation versus nonoperative treatment for displaced midshaft clavicular fractures: a multicenter, randomized, controlled trial. J Bone Joint Surg Am. 2013;95(17):1576–84. [Crossref][PubMed]

77. Baker JF, Mullett H. Clavicle non-union: autologous bone graft is not a necessary augment to internal fixation. Acta Orthop Belg. 2010;76(6):725–9. [PubMed]

78. Ramoutar DN, Rodrigues J, Quah C, Boulton C, Moran CG. Judet decortication and compression plate fixation of long bone non-union: is bone graft necessary? Injury. 2011;42(12):1430–4. [Crossref][PubMed]

79. Ahlmann E, Patzakis M, Roidis N, Shepherd L, Holtom P. Comparison of anterior and posterior iliac crest bone grafts in terms of harvest-site morbidity and functional outcomes. J Bone Joint Surg Am. 2002;84-a(5):716–720.

80. Arrington ED, Smith WJ, Chambers HG, Bucknell AL, Davino NA. Complications of iliac crest bone graft harvesting. Clin Orthop Relat Res. 1996;329:300–9. [Crossref]

81. Fowler BL, Dall BE, Rowe DE. Complications associated with harvesting autogenous iliac bone graft. Am J Orthop (Belle Mead, NJ). 1995;24(12):895–903.

82. Friedlaender GE, Perry CR, Cole JD, Cook SD, Cierny G, Muschler GF, et al. Osteogenic protein-1 (bone morphogenetic protein-7) in the treatment of tibial nonunions. J Bone Joint Surg Am. 2001;83-A Suppl 1(Pt 2):S151–8.

83. Goulet JA, Senunas LE, DeSilva GL, Greenfield ML. Autogenous iliac crest bone graft.

Complications and functional assessment. Clin Orthop Relat Res. 1997;339:76–81.
[Crossref]

84. Hierholzer C, Sama D, Toro JB, Peterson M, Helfet DL. Plate fixation of ununited humeral shaft fractures: effect of type of bone graft on healing. J Bone Joint Surg Am. 2006;88(7):1442–7.
[PubMed]

85. Keller EE, Triplett WW. Iliac bone grafting: review of 160 consecutive cases. J Oral Maxillofac Surg. 1987;45(1):11–4.
[Crossref][PubMed]

86. Kurz LT, Garfin SR, Booth RE Jr. Harvesting autogenous iliac bone grafts. A review of complications and techniques. Spine. 1989;14(12):1324–31.
[Crossref][PubMed]

87. Momberger NG, Smith J, Coleman DA. Vascularized fibular grafts for salvage reconstruction of clavicle nonunion. J Shoulder Elbow Surg. 2000;9(5):389–94.
[Crossref][PubMed]

88. Boehme D, Curtis RJ Jr, DeHaan JT, Kay SP, Young DC, Rockwood CA Jr. Non-union of fractures of the mid-shaft of the clavicle. Treatment with a modified Hagie intramedullary pin and autogenous bone-grafting. J Bone Joint Surg Am. 1991;73(8):1219–26.
[Crossref][PubMed]

89. Lyons FA, Rockwood CA Jr. Migration of pins used in operations on the shoulder. J Bone Joint Surg Am. 1990;72(8):1262–7.
[Crossref][PubMed]

90. Naidoo P. Migration of a Kirschner wire from the clavicle into the abdominal aorta. Arch Emerg Med. 1991;8(4):292–5.
[Crossref][PubMed][PubMedCentral]

91. Nordback I, Markkula H. Migration of Kirschner pin from clavicle into ascending aorta. Acta Chir Scand. 1985;151(2):177–9.
[PubMed]

92. Abbott LC, Lucas DB. The function of the clavicle; its surgical significance. Ann Surg. 1954;140(4):583–99.
[Crossref][PubMed][PubMedCentral]

93. Codman EA. Rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa. The shoulder. Boston: Thomas Todd; 1934.

94. Elkin DC, Cooper FW Jr. Resection of the clavicle in vascular surgery. J Bone Joint Surg Am. 1946;28:117–9.
[PubMed]

95. Green RM, Waldman D, Ouriel K, Riggs P, Deweese JA. Claviculectomy for subclavian venous repair: long-term functional results. J Vasc Surg. 2000;32(2):315–21.
[Crossref][PubMed]

96. Gurd FB. The treatment of complete dislocation of the outer end of the clavicle: an hitherto undescribed operation. Ann Surg. 1941;113(6):1094–8.
[Crossref][PubMed][PubMedCentral]

97. Sonnabend D. The origin of the shoulder: a fairytale based on fact. 9th International Congress on Surgery of the Shoulder Meeting; 2004 May 2–5; Washington, DC.

98. Spar I. Total claviculectomy for pathological fractures. Clin Orthop Relat Res. 1977;129:236–7.
[Crossref]

99. Wood VE. The results of total claviculectomy. Clin Orthop Relat Res. 1986;207:186–90.

100. Krishnan SG, Schiffern SC, Pennington SD, Rimlawi M, Burkhead WZ Jr. Functional outcomes after total claviculectomy as a salvage procedure. A series of six cases. J Bone Joint Surg Am. 2007;89(6):1215–9.
[Crossref][PubMed]

101. Wessel RN, Schaap GR. Outcome of total claviculectomy in six cases. J Shoulder Elbow Surg. 2007;16(3):312–5.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_4

# 4. Proximal Humerus Nonunions

Ethan S. Lea[1] and Philip R. Wolinsky[1]

(1) Department of Orthopedic Surgery, University of California at Davis Medical Center, 4860 Y St, Suite 3800, Sacramento, CA 95608, USA

**Philip R. Wolinsky**
**Email:** philipwolinsky@hotmail.com

**Keywords** Proximal humerus nonunions – Locking plates – Intramedullary nails – Hemiarthroplasty – Reversed total shoulder arthroplasty – Bone morphogenic protein – BMP

## 4.1 Introduction

Fractures of the proximal humerus are common injuries, accounting for about 5% of all fractures of the appendicular skeleton [1, 2]. The incidence increases as patients age [3] since this injury occurs more frequently in individuals who have decreased bone mineral density [4, 5]. As the population ages, the prevalence of proximal humerus fractures will rise with a projected 3-fold increase over the next three decades [6]. The majority of these fractures (approximately 85% [7]) occurs as the result of a low energy injury and are minimally displaced, have a stable configurations, and are amenable to non-operative treatment.

Some of these fractures will go on to develop a nonunion. Cadet et al. stated that the biological factors that caused or contributed to the development of the nonunion needed to be treated in order for treatment of the nonunion to succeed [8]. As the number of these fractures grows, it seems logical that the subset that fails to unite will increase as well.

## 4.2 Epidemiology

The incidence of nonunions of the surgical neck of the proximal humerus has been cited to be as high as 20% [9]. The challenge with these figures is that they are generated from tertiary care centers where patients have been referred for treatment of their nonunions rather than from centers that provide the initial care for all of these injuries, which inflates the perceived incidence of nonunions.

A large single-center clinical study found the rate of nonunion of fractures of the proximal humerus to be only 1.1% [10]. Court-Brown and McQueen prospectively followed 1027 consecutive proximal humerus fractures treated at their institution, which provides care for all the proximal humerus fractures in a population of approximately 650,000 individuals. The patients were treated at the discretion of the admitting surgeons: 89.1% were treated non-operatively, 7.1% were treated with internal fixation, and 3.8% with a hemiarthroplasty. Only 11 of the 995 patients who were followed for one year developed a nonunion. The fractures were classified using the AO/OTA and Neer classifications and comminuted bifocal fractures (AO/OTA B2.3) had the highest incidence of nonunion with two of six patients failing to unite. The authors stressed that while the nonunion rate of 33.3% for this specific subset of patients was high, these fractures were quite rare and rate calculations based on such small patient numbers may be unreliable. Displaced articular fractures (AO/OTA C2.3) and Neer 4-part fractures had nonunion rates of 8.3 and 5.9% respectively, but the treatment of these groups were heterogeneous and included some treated non-operatively and some that underwent hemiarthroplasty. Therefore, the true rates of nonunion for these subsets are unknown.

Iyengar et al. performed a meta-analysis of 12 studies with a total of 650 patients who underwent non-operative treatment of their proximal humerus fractures and found a 2% incidence of nonunion (range 0–7%). These studies found that the incidence of nonunion varied little when fractures were categorized using the Neer classification: four studies (233 patients) found that Neer 1- and 2-part fractures had a 100% union rate, four studies (66 patients) found that Neer 3- and 4-part fractures had a 98% union rate, and four studies (351 patients) that included all fracture types had a 96% union rate [11]. The same group conducted a similar systematic review of 12 studies of 514 patients with Neer 2-, 3-, or 4-part fractures surgical stabilized

with locked plating constructs and found that 16 patients failed to unite for a nonunion rate of 3.1% [12].

## 4.3 Risk Factors

Risk factors for developing a nonunion include factors related to patient demographics and fracture characteristics. Advancing age with its corresponding loss of bone mineral density has been implicated as increasing the likelihood of nonunion [13], but the largest prospective cohort study that compared patients who developed nonunions with those who did not, found no significant difference in the average ages between the two groups [10]. Nutritional deficiencies and metabolic bone disease are recognized as contributors to delayed unions and nonunions and should be screened for with appropriate laboratory markers either at the time of initial fracture treatment or at the earliest signs of delayed fracture healing [13]. Smoking increases the odds ratio of developing a nonunion to 5.5 times that of non-smokers for proximal humerus fractures deemed appropriate for non-operative management [14]. Medical comorbidities including heart disease, hypertension, and chronic obstructive pulmonary disease have high rates of prevalence among patients with proximal humerus nonunions, which suggests an association between chronic medical comorbidities and nonunions, but the study designs demonstrating this association could not infer causality. Factors such as alcohol abuse, other substance abuse, or psychiatric illnesses that affect patients' ability to appropriately participate in their care have been described as a barrier to achieving union [13].

Fracture characteristics including translation and metaphyseal comminution have been suggested as potentially increasing the risk of nonunion. Several studies have identified 2-part fractures of the surgical neck as having a higher risk of developing nonunions [13, 15, 16]. Court-Brown and McQueen noted that translated surgical neck fractures (AO/OTA A3.2) had a nonunion rate of 3.8% that increased to 4.3% when adolescents and patients who had been lost to follow-up were excluded [10]. A subset analysis was performed based on the degree of translation on initial lateral radiographs: the rate of nonunion for fractures translated less than 33% was 2.6%, increasing to 10% for those translated 33–66%, and 8.1% for those translated 66–100%. This suggests an association between translational displacement and likelihood of nonunion, but there does not seem to be a

simple linear relationship. This is emphasized by the fact that all the fractures that were translated 100% achieved union, which is difficult to explain if increasing translation leads directly to higher risk of nonunions. It should be noted that a small percentage of patients within these groups were treated surgically but they represent a minority within each of the subsets described. Another subset analysis found that increasing fracture site angulation did not correlate with the risk of nonunion.

The effect of metaphyseal comminution on union was evaluated by combining the AO/OTA subgroups A3.3 and B2.3. This group comprised only 52 of the 1027 fractures (5.1%), yet had a nonunion rate of 7.7% compared to 0.7% for all the other fractures [10]. It is unclear if greater amounts of translation and comminution increase the risk of nonunion due to decreased cortical contact or if they serve as markers for disruption of the medial soft tissues and damage to the blood supply [8]. The authors of the largest prospective review of proximal humerus nonunions remained unable to define predictive criteria for the development of nonunions because the incidence of nonunions is so low that an adequate prospective study would require the inclusion of an overwhelmingly and unrealistically large number of patients [10].

The anatomy and biomechanics of the glenohumeral joint may contribute to the likelihood of developing a nonunion [8, 13]. Muscle forces produce deformities, which may be larger for more unstable fracture patterns. The insertion of the subscapularis muscle can translate the lesser tuberosity medially when it exists as a separate fragment, or may internally rotate the head fragment depending on the remaining muscular forces acting on the proximal piece. The rotator cuff muscles attached to the greater tuberosity will abduct and externally rotate the proximal fragment when the greater tuberosity remains attached to the articular segment, or will pull the greater tuberosity superiorly and posteriorly when it is displaced. The humeral diaphysis is displaced anteriorly and medially by the insertion of the pectoralis major and pulled cranially by the deltoid muscle. The peri-articular location of these fractures may contribute to the risk of nonunion if intra-capsular extension allows synovial fluid to bathe the fracture fragments and limit the formation and organization of hematoma that leads to callus formation [17, 18].

Interposed soft tissues between fracture fragments may also be a predisposing factor for the development of nonunions. Soft tissues structures

around the proximal humerus include: the tendon of the long head of the biceps, deltoid muscle fibers, and the rotator cuff [13]. Nayak et al. [19] reviewed their experience with the operative treatment of proximal humerus nonunions and found that interposed structures blocked healing in 8 of 17 (47%) cases. The long head of the biceps was interposed in six nonunions and the deltoid was the offending structure in the remaining two. Duralde et al. [17] reviewed 20 patients who underwent surgical intervention for proximal humerus nonunions and found soft tissue interposition in 8 of 12 patients (67%) who had initially been treated non-operatively. The contribution of soft tissue interposition as a risk factor for nonunion is controversial. Court-Brown and McQueen felt it was rare, especially for low energy fractures [10].

Another potential risk factor for the development of a nonunion is the presence of glenohumeral joint arthrosis . Rooney and Cockshott presented a case series of nonunion patients who had glenohumeral stiffness from rheumatoid arthritis or a surgical arthrodesis [20]. These patients presumably had increased torsional and bending forces transferred to the fracture site due to the immobility of the adjacent joint, which overcame the process of establishing bridging callus. This pathophysiology may be generalized to other restrictive phenomenon affecting glenohumeral motion such as osteoarthritis or adhesive capsulitis, as well.

Inadequate initial immobilization of the fracture may permit increased motion that exceeds the ability to achieve union. This may result from the lack of use of a sling, cuff and collar, or other forms of shoulder immobilization. Alternatively, overly aggressive or premature rehabilitation has been implicated as a potential cause of proximal humerus nonunions. Unstable fractures treated non-operatively must be given the opportunity to consolidate prior to beginning therapy in order to prevent excessive micro-motion or frank displacement at the fracture site [13]. Inadequate immobilization may also occur after surgical interventions that do not adequately stabilize the osseous fragments or malunions reduce the fracture into an alignment that risks further displacement postoperatively, such as varus angulation of the surgical neck.

Distraction across a proximal humerus fracture has also been identified as a risk factor for nonunion. Neer described the weight of the arm itself as a sufficient distracting force to prevent the healing processes needed to achieve union. He reviewed a subset of nine patients with 3- and 4-part fractures that

had failed to achieve union after a trial of non-operative management. Six of the nine had been treated in a hanging arm cast and two with overhead traction. He hypothesized that the distracting forces were the major cause of the patients' nonunions [21].

## 4.4 Classification

Checchia et al. recognized the lack of a descriptive classification system for nonunions of the proximal humerus and published the following four-group schema based on their review of 21 cases. High 2-part nonunions describe nonunions of the anatomic neck with a small articular segment and include 3-part fractures in which the tuberosity segment consolidated with less than 5 mm of displacement. Low 2-part nonunions are differentiated by a primary nonunion of the surgical neck between the level of the lesser tuberosity and the insertion of the pectoralis major tendon and also include 3-part fractures where the tuberosity united with less than 5 mm of displacement. Complex nonunions include 3- and 4-part fractures or head-splitting fractures in which the surgical neck nonunion is accompanied by a tuberosity nonunion that is displaced greater than 5 mm. Lost fragment nonunions describe those with a large degree of bone loss associated with previous open injury or osteomyelitis [22]. This classification scheme, though helpful in communicating nonunion characteristics, has not widely been utilized in subsequent studies. Therefore, evaluation of treatment algorithms based on it has not been performed and prognostic implications of this classification are unknown.

## 4.5 Patient Evaluation

### 4.5.1 Clinical Examination

Patients with proximal humerus nonunions frequently present with a chief complaint of inability to perform simple activities of daily living due to pain at the nonunion site. The pain may be moderate at rest with the arm in a dependent position but commonly increases with attempted motion. Patients are often unable to lift even light loads. Depending on the chronicity of the nonunion, atrophy of the deltoid and para-scapular muscles may be noted. Evaluation of axillary nerve function must be performed if surgical

intervention is being considered, as it will influence the success of certain procedures, and may require electromyography (EMG) if the physical examination is equivocal. Integrity of the rotator cuff musculature should also be evaluated, but disuse and pain may make this difficult and MRI may be needed for an adequate evaluation.

Patients with proximal humerus nonunions may have soft tissue contractures that restrict shoulder range of motion and the magnitude of this limitation is related to the length of time from the initial injury. Court-Brown and McQueen measured the shoulder range of motion of patients following proximal humerus fractures and compared the motion of patients who developed nonunions at 6, 13, 26, and 52 weeks post injury to the motion of patients whose fractures united. At all time points, flexion, extension, abduction, internal and external rotation was significantly reduced in the individuals with nonunions. At the final follow-up at 52 weeks, patients with union had regained 72% of their flexion, while those with nonunions flexed to only 26% of their uninjured side. Abduction showed an even more striking variance—58% of motion was regained with united fractures, but just 6% with a nonunion. Patients with nonunions recovered only 61% of extension (vs. 90% for patients with union), 62% of external rotation (vs. 79%), and 42% of internal rotation (vs. 72%) at 52 weeks. Most significantly, they found that as early as six weeks after injury, patients who were developing nonunions had less motion, and that any functional gains after 13 weeks post injury were minimal. Motion increased in a relatively linear pattern for patients who achieved union, but patients with nonunions had not only less mobility, but lost motion in all directions except external rotation after week 26. This was attributed to contractures of the soft tissues surrounding the shoulder combined with transfer of motion to the fracture site [10].

Based on these findings, patients who are developing nonunions will have smaller arcs of motion than expected as early as 6 weeks post injury. Established nonunions will likely begin to lose mobility after 26 weeks, as opposed to patients with healed fractures, who will continue to improve their ranges. Patients with proximal humerus fractures who are failing to achieve expected mobility gains should be worked up for nonunions as early as 13 weeks after injury.

Other published case series have reviewed patients who underwent surgical interventions for their symptomatic proximal humerus nonunions and measured the preoperative range of motion, finding shoulder flexion

consistently averaged between 35° and 46° [16, 19, 23, 24], shoulder abduction averaged between 35° and 41° [15, 24], external rotation averaged 15°–26° [15, 16, 23, 24], and internal rotation averaged 25° [24] or the ability to reach the sacrum with the involved extremity [15, 19]. These studies have a selection bias since patients chose surgical intervention for their disabling nonunions and may have been more restricted than the average non-united shoulder range of motion.

Court-Brown and McQueen did not publish dedicated pain scores but used the Neer and Constant scores that contain pain assessment. They used the time it took to resume specific activities of daily living as a surrogate for recovery from the pain and disability caused by proximal humerus fractures. Patients with nonunions took slightly longer on average than those who achieved union to regain the ability to perform personal hygiene (5.4 vs. 5.1 weeks), approximately one week longer to be able to dress independently (5.7 vs. 4.6 weeks), and greater than twice as long to resume the ability to perform housework and shop (16.8 weeks vs. 7.5 weeks and 17.4 weeks vs. 8.2 weeks, respectively) [10]. Therefore, the degree of disability of proximal humerus nonunions, as well as the length of time it takes for that disability to resolve should be assessed, and suspicion for a nonunion should be high among patients who are slow to regain function after a proximal humerus fracture.

Studies that used scales to quantify the pain of nonunions of proximal humerus fractures are rare. Several small case series of patients who underwent surgical intervention for symptomatic nonunions used different pain assessments tools. Antuña et al. evaluated pain scores preoperatively in 25 patients using a 5-point scale described by Cofield [25], where a score of four indicated moderate pain and five meant severe pain. Their average score of 4.6 reflects the debilitating pain experienced by these individuals [15]. Duralde et al. [17] screened 20 patients who went on to pursue surgery for their nonunions and found that 16 (80%) classified their pain as totally disabling, requiring medications and interrupting sleep. These series illustrate the severe, disabling pain that proximal humerus nonunions can cause, but only include patients that chose intervention for their symptomatic nonunions and therefore have a selection bias. Several authors note that there are patients with proximal humerus nonunions that are less symptomatic and may not require further treatment [8, 26].

### 4.5.2 Radiographic Imaging

Radiographic evaluation of a proximal humerus nonunion begins with a standard shoulder series: a true AP in the scapular plane (Grashey view), scapular Y, and axillary views. Internal and external rotational AP views may be of use but have largely been supplanted by computed tomography (CT). Historically, in cases when standard roentgenographs were equivocal, stress radiographs were used to detect instability [27], but CT has also replaced these. Comparison views of the contralateral shoulder may be of benefit for evaluating loss of length or associated malunions involving the tuberosities.

Nonunions may be classified as hypertrophic or atrophic. Hypertrophic nonunion s have abundant callus that failed to bridge the fracture site, and have been described as having the appearance of an “elephant’s foot”. These nonunions possess preserved vascularity along with the biologic constituents to permit bone healing. Atrophic nonunion s appear osteopenic at the fracture margins with minimal callus formation. They are biologically inert with vascular and biologic compromise and will require biologic augmentation. Lytic or mixed lytic and blastic lesions can be signs of underlying pathologic or metastatic processes. Signs of a sequestrum or involucrum are pathognomonic for infection. Increased density and sclerosis throughout the humerus may indicate Paget’s disease or osteopetrosis.

Radiographs should be examined for other characteristics in addition to the nonunion. Evidence of avascular necrosis of the humeral head with subchondral sclerosis or humeral head collapse should be evaluated. The glenohumeral joint should also be inspected for signs of degeneration including: joint space narrowing, subchondral sclerosis, osteophytes, and bone loss involving either the humeral head or the glenoid. The presence of avascular necrosis or advanced osteoarthritis may shift treatment of a nonunion towards an arthroplasty.

Advanced imaging modalities may provide additional information to assist in diagnosis and surgical planning. Computed tomography offers several benefits. The multiplanar imaging provided by computed tomography with 2- and 3-dimensional reconstructions allows evaluation of tuberosity malunions, head cavitation, intra-articular extension, and glenohumeral arthritic changes. Although CT scanning offers high sensitivity in evaluating for the presence of a nonunion, the specificity has been found to be somewhat lower due to the high number of false positives. Magnetic resonance imaging can demonstrate the presence of osteonecrosis and quantify the area involved.

MRI may also address questions regarding the continuity and health of the rotator cuff that would influence arthroplasty options, though some centers are moving towards ultrasound for diagnosing rotator cuff pathology. The latter remains highly dependent on the capabilities of experienced technologists, which may not be available in all facilities.

Nuclear imaging modalities may offer additional information in the evaluation of proximal humerus nonunions by evaluating callus vascularity and metabolic activity, identifying the presence of synovial pseudarthroses, or supporting the diagnosis of acute or chronic infection at nonunion sites. Brinker and O'Connor [28] outlined the use of various nuclear imaging studies to assess nonunions. Technetium-99m-pyrophosphate studies will have increased uptake at the site of viable nonunions as the complexes are incorporated into hydroxyapatite crystals, a process which requires and indicates active osteoblastic activity and adequate vascularity. When a synovial pseudoarthrosis, or a fluid-filled pseudocapsule forming a mobile false joint is present at a nonunion site, a technetium-99m-pyrophosphate scan will show a cold cleft between metabolically active, hot ends of the nonunion. This corresponds with a lack of vascular ingrowth and osteogenesis within the pseudocapsule. Radiolabeled white blood cell scans (such as indium-111 or technetium-99m-hexamethylpropylene amine oxime) can help support the diagnosis of an acute infection since increased activity at the nonunion site occurs when labeled polymorphomuclear leukocytes accumulate. Alternatively, gallium scans use gallium-67 citrate (often with a technetium-99m sulfa colloid) that localizes to the site of a chronically infected nonunion.

### 4.5.3 Laboratory Tests

Laboratory analysis in patients with proximal humerus nonunions can help determine the cause of failure to unite and identify factors that should be corrected to allow healing. If the possibility of an infection at the nonunion site exists, preoperative laboratory evaluation should include an erythrocyte sedimentation rate and C-reactive protein level. These are non-specific markers of systemic inflammatory response and can be elevated by other processes. A white blood cell count may show leukocytosis, which further raises the suspicion of infection and the differential may demonstrate a left shift, with increased percentages of polymorphonuclear cells. Ultimately, the gold standard for diagnosing infection is cultures taken from the nonunion

site at the time of surgery.

Other laboratory values may provide evidence of nutritional or metabolic derangements that may be preventing or slowing union. Nutritional deficiencies may be assessed with an albumin level or total lymphocyte count. Values ≥ 3.0 g/dL and >1500 cells/mm$^3$ are preferred to allow appropriate healing [28]. Other endocrine markers have been found to be associated with nonunions that were felt to not be the result of either fracture-related or technical factors. Brinker et al. identified 37 patients with nonunions that were not related to infection or technical factors and referred those patients to an endocrinologist. 68% were found to be vitamin D deficient, 35% had abnormal calcium excretion, and 24% had functional thyroid abnormalities. Less frequent endocrinopathies were found by measuring levels of reproductive hormones, alkaline phosphatase, parathyroid hormone, prolactin, and growth hormone. All 31 patients diagnosed with metabolic or endocrine abnormalities underwent treatment of the abnormality and 30 achieved union. Four patients united without any surgical intervention [29]. This study was not limited to patients with nonunions of the proximal humerus but demonstrates that metabolic and endocrine abnormalities may be associated with nonunions. Preoperative assessment of a patient's thyroid function, vitamin D and calcium levels may detect modifiable factors that contributed to the development of the nonunion. If these tests are normal but the suspicion for metabolic abnormalities remains high due to lack of other obvious cause for nonunion, a referral to an endocrinologist may be advisable.

## 4.6 Surgical Timing

The time course of healing of a proximal humerus fracture is variable and can be affected by fracture characteristics, systemic diseases, and metabolic abnormalities. Bridging callus is typically seen on radiographs at a mean of 6 weeks [30]. The median time to union is 13–14 weeks [10, 14], and Norris et al. consider fractures non-united if not clinically healed by 3 months [18, 31]. Court-Brown and McQueen noted that the range of motion, Neer, and Constant scores improved for all patients over 6 months following injury but, in patients who developed nonunions, all of these were less favorable as early as 6 weeks. Nonunion patients improved from 6 weeks to 3 months, but showed only minimal gains between 3 and 6 months, which suggests that

nonunion patients can be identified early. After 6 months, nonunion patients had a decline in motion of their glenohumeral joint and the Neer and Constant scores [10]. This was attributed to the development of soft tissue contractures at the shoulder after 6 months.

Based on these findings, proximal humerus nonunions may be identifiable as early as 3 months after injury, with a lack of callus formation on imaging, poorer glenohumeral range of motion on physical exam than expected, and lower shoulder outcome scores than expected. Once diagnosed, a proximal humerus nonunion should be addressed prior to 6 months after injury, before soft tissue contractures at the glenohumeral joint develop and present a barrier to regaining optimal function.

Beredjiklian et al. reviewed the results of 39 patients who underwent late surgical intervention for proximal humerus malunions. Intra-operatively they found that 31 patients (79%) had concomitant soft tissue pathology consisting of 25 capsular contractures, 15 torn rotator cuffs, and two cases of subacromial impingement. These were treated via a circumferential capsular release, subscapularis tendon lengthening, rotator cuff repair, and acromioplasty. The authors noted a significant difference in outcomes among patients who underwent surgical intervention within one year of initial injury (84% satisfactory results) versus those who were treated after one year (55% satisfactory results) [32]. This was attributed to more soft tissue scarring and disuse atrophy in those patients for whom operative intervention was delayed and emphasizes the benefit of identifying nonunions and intervening early—prior to the development of contractures. This study also stresses the need to address soft tissue contractures during the surgical procedure after they have developed to optimize the outcome.

Studies that analyzed the results from small series of patients with proximal humerus nonunions treated many months after their initial injury emphasize the need to address the bony nonunion as well as the soft tissues contractures during surgery in order to maximize functional improvement. Preoperative assessments demonstrated significant reductions in glenohumeral motion in these patients associated with their prolonged disuse of the shoulder and highlight the need to address these contractures during surgery to prevent a united fracture with a stiff shoulder joint [13].

## 4.7 Treatment Options

### 4.7.1 Non-operative

The treatment of proximal humerus nonunions must overcome numerous challenges including the biologic insults from the initial injury and any previous surgeries, bone loss, humeral head cavitation, osteopenia, soft tissue contractures, and infection. Patients are commonly elderly with medical comorbidities. Surgery may be extensive and the postoperative course requires compliance and assistance from family and friends. For some patients, medical co-morbidities may make the risks of surgical intervention unacceptably high. Alternatively, patients may be unable or unwilling to comply with postoperative protocols. Some authors consider a nonfunctional deltoid muscle to be a contraindication for operative treatment [22]. In such cases, efforts to increase the patient's healing potential by addressing nutritional and metabolic disorders should be undertaken and the focus of treatment should be on patient comfort (Fig. 4.1a–d).

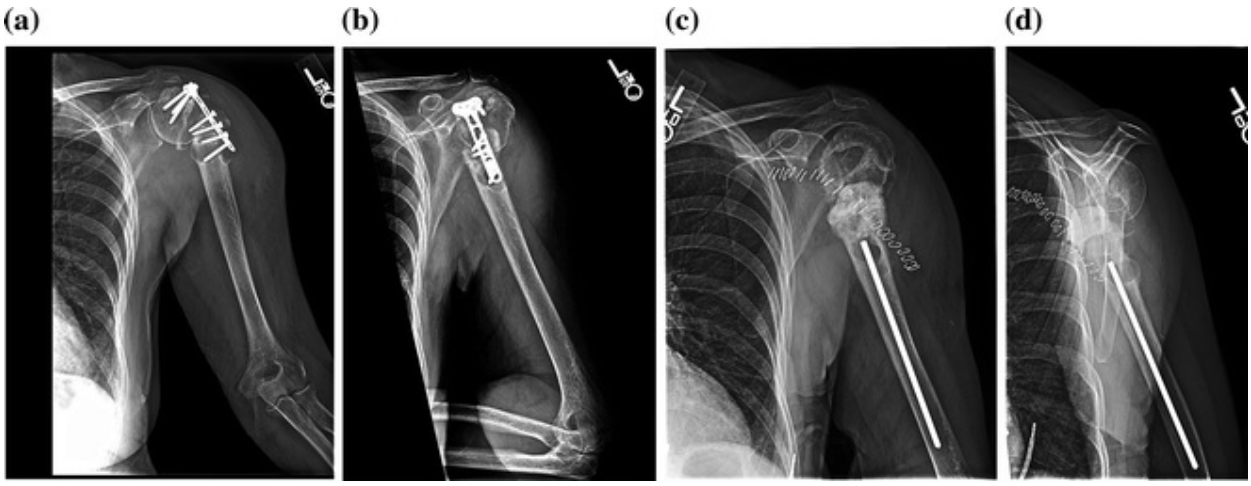



***Fig. 4.1*** An 82-year-old female two years status-post open reduction and internal fixation with standard T-plate with wound drainage, hardware failure, and surgical neck nonunion on anteroposterior (AP) (**a**) and lateral (**b**) views. Underwent stage one of a planned two-stage procedure with removal of hardware, irrigation, debridement, and placement of antibiotic cement spacer, seen one month postoperatively on AP (**c**) and lateral (**d**) views. Patient is now six months post-surgery, comfortable and able to perform activities of daily living with motion up to shoulder-level. She has declined her second stage in favor of no further surgery

The surgical indications for nonunions of the proximal humerus include debilitating pain and functional deficits. Some smaller studies found that up to 50% of patients with proximal humerus nonunions are minimally symptomatic and quite functional [19, 33]. A case report study that reviewed

the complications of locking plates used for the treatment of proximal humerus fractures found that although the Constant scores were significantly lower for patients who developed nonunions (45 vs. 68), only two of the four patients with a nonunion opted for revision surgeries [34]. A thorough conversation should be undertaken with patients in order to assess their pain and functional deficits and educate them about the risks of revision surgery so the patient may make an informed decision.

### 4.7.2 Surgical Treatment

Many techniques have been described for the repair of proximal humeral surgical neck nonunions. Plating techniques using conventional T-plates, fixed angle devices such as blade plates or anatomically designed locking plates have been described. Intramedullary implants have also been utilized, ranging from older implants such as Rush rods or Enders nails in conjunction with tension banding, to modern rigid interlocked intramedullary nails. In the setting of bone loss or atrophic nonunion s augmentation with allograft or autograft bone is necessary and structural reinforcement with fibular strut grafts has been found to be biomechanically advantageous. A successful nonunion repair requires adequate bone quality and a proximal fragment that is large enough to obtain purchase in. Although fixed angle devices and locking screw technology improve biomechanical stability in the setting of osteoporotic bone, the size and quality of the fragments should be carefully assessed before osteosynthesis is performed. Significant medial calcar comminution has also been described as a harbinger of difficulty establishing a stable configuration that must be addressed when undertaking fixation. Several techniques have been described, including impaling the head onto the shaft, reconstructing the calcar, using calcar screws to substitute for the medial cortex, or using an intramedullary fibular strut graft [8].

### 4.7.3 Avascular Necrosis

Humeral head viability is a consideration when deciding on the operative treatment of acute fractures since osteonecrosis and the resultant humeral head collapse may lead to poor results. However, the incidence of avascular necrosis and its functional implications are not fully understood. A review of proximal humerus fractures treated non-operatively found a 2% rate of AVN (12 studies and 650 patients) [11]. Similar reviews of fractures treated using

locked plating constructs found rates of AVN ranging from 7.9 to 10.8% [12, 35], although only four of the 51 patients who developed AVN chose to undergo further surgical treatment [35]. Patients who develop AVN after treatment of acute proximal humerus fractures have lower Constant scores (average 46) [36] and a decreased likelihood of achieving good or excellent results when compared to those who do not develop AVN [37].

Gerber et al. performed a subgroup analysis of patients with AVN. They found even with AVN and collapse, Constant scores of patients with an anatomical reduction were similar to patients who had undergone a primary hemiarthroplasty for a proximal humerus fracture. This suggests that patients treated with osteosynthesis after an appropriate reduction who develop AVN may function as well as patients who undergo shoulder hemiarthroplasty [36]. Although the development of AVN adversely affects Constant scores, the functional limitations that result do not drive patients to undergo further surgical intervention at high rates, and they function at levels similar to patients whose fractures were treated using a hemiarthroplasty acutely. These studies are of patients with acute proximal humerus fractures, rather than nonunions. Unfortunately, the current nonunion literature does not allow calculation of the rate of AVN or estimation of its clinical significance.

## 4.7.4 Osteosynthesis

### *4.7.4.1 ORIF with Standard Plates*

Several studies reported good results when osteosynthesis was used for the treatment of surgical neck nonunions of the proximal humerus. Healy et al. retrospectively reviewed their experience and found better functional results after open reduction and internal fixation than after hemiarthroplasty , unreamed intramedullary implants, or non-operative management. Fixation techniques varied- the majority of their patients were treated with a 4.5 mm T-plate and a tension band through the rotator cuff, but others were stabilized using semitubular plates, dynamic compression plates, or Cobra plates. 12 of 14 (86%) nonunions achieved union at an average of 4.8 months postoperatively. Both nonunions that failed to unite were performed on the same patient who had significant medical comorbidities that may have contributed to her lack of healing. Autogenous bone grafting was performed in 12 of 14 cases. 11 of 12 grafted cases and 1 of 2 procedures performed

without grafting achieved union. There were nine good results, four fair results, and one poor result. Shoulder range of motion averaged 110° of elevation, 33° of external rotation, and internal rotation to the thoracolumbar junction postoperatively [38].

### *4.7.4.2 ORIF with Blade Plates*

Osteosynthesis using a blade plate has been shown to be successful for achieving union for nonunions of the surgical neck (Fig. 4.2a–i). Ring et al. reviewed 25 patients who underwent blade plating with autogenous iliac crest bone grafting, 23 of which (92%) united their fractures. Eighty percent of the patients obtained good or excellent functional results [39]. Allende and Allende presented their results of 7 patients with atrophic proximal humerus nonunions who underwent surgical treatment using a locking blade plate and all seven cases achieved union. It took an average of 5.9 months for patients to have radiographic evidence of union and patients who had allograft bone grafting took longer to achieve union compared with those who received autogenous bone graft (average of 7 vs. 5 months). Postoperative scores using the Disabilities of the Arm, Shoulder, and Hand (DASH) averaged 25 points and Constant scores averaged 72.7 [40]. Tauber et al. used a blade plate to treat 45 of 55 patients with proximal humerus nonunions who had sufficient bone stock in the humeral head. A blade plate was fashioned by bending a one third tubular plate and patients were not bone grafted. 41 of 45 patients (91%) achieved union. The four failures were attributed to varus collapse with screw pullout and underwent revision with a blade plate made from a 4.5 mm DC plate. Patients treated with blade plates in this study had an increase in Constant scores from 30.5 to 85.5 and had improvements in active shoulder flexion and abduction. Complications included two patients (4.4%) with surgical site infections and two patients (4.4%) who developed AVN [41]. Galatz and Iannotti treated 13 patients with nonunions of the proximal humerus: ten with blade plates and three with T-plates, combined with auto- or allograft. Eleven patients achieved excellent results and only one patient failed to achieve union. The persistent nonunion occurred in a wheelchair-dependent patient who returned to functional use of the involved upper extremity early and ultimately broke her T-plate but went on to successful union and excellent results following a revision surgery. Pain scores improved from 4.2 to 1.2 on a 5-point scale and the average shoulder flexion improved from 23.8° prior to surgery to 143.8°. All patients in this study

achieved overhead elevation and were able to perform activities of daily living independently [13].

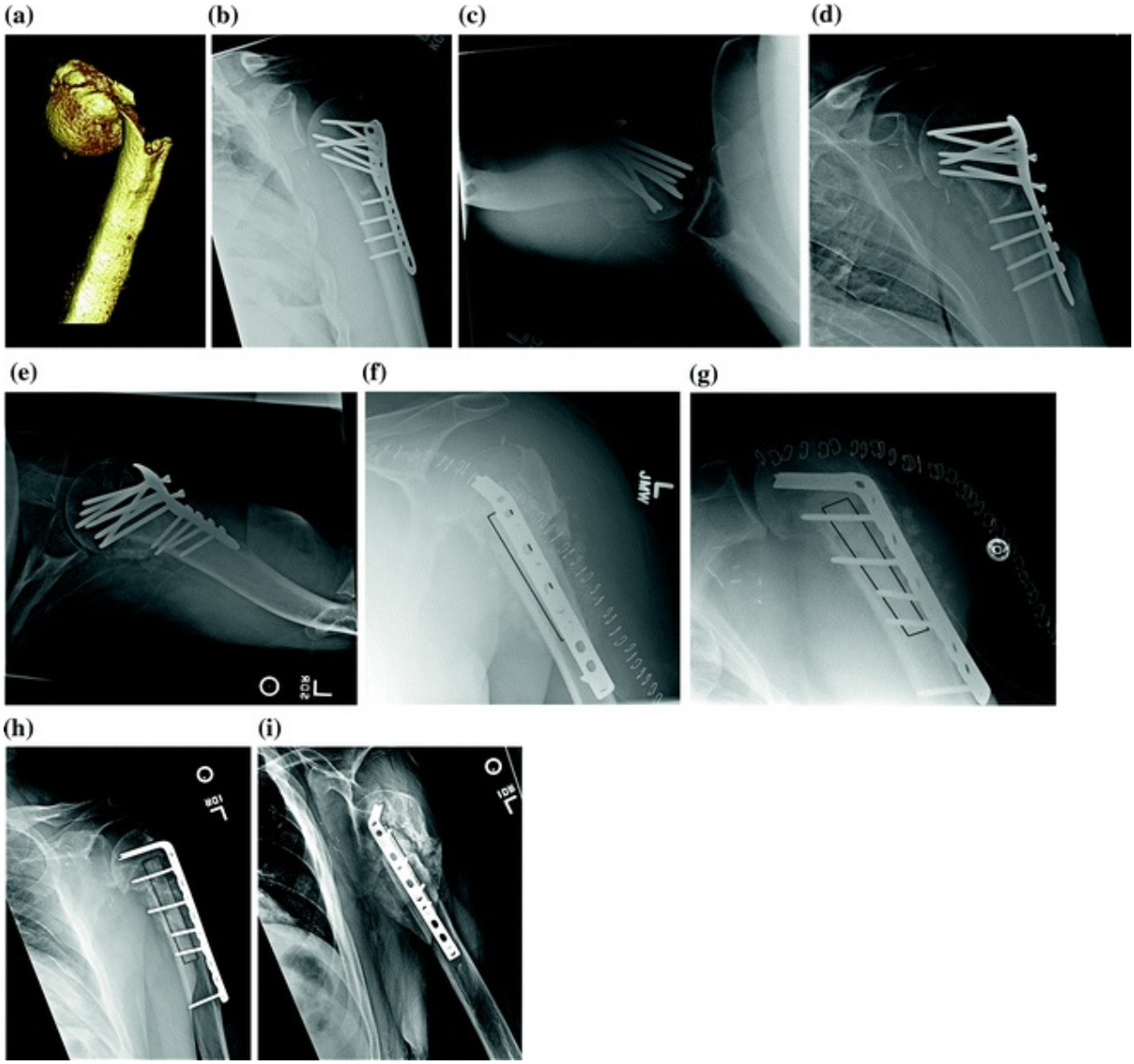



***Fig. 4.2*** A 37-year-old male sustained 3-part proximal humerus fracture (surgical neck and lesser tuberosity) during an motor vehicle accident. **a** Underwent open reduction and internal fixation with locked plating and allograft, seen on anteroposterior (AP) (**b**) and axillary (**c**) views. Went on to surgical neck nonunion with varies collapse and screw back out on AP (**d**) and axillary (**e**) views. Patient underwent hardware removal and revision fixation using a blade plate, fibular strut allograft (*outlined*), and bone morphogenic protein, seen immediately post-operatively in AP with internal rotation (**f**) and external rotation (**g**) views. Union was achieved at seven months on AP (**h**) and lateral (**i**) views

## 4.7.5 Use of Augmentation

### *4.7.5.1 Structural Graft Augmentation*

The use of an intramedullary peg graft was first described by Walch et al. who treated 20 patients with proximal humerus nonunions using cortico-cancellous auto-graft harvested from the iliac crest, tibial crest, or fibula, in addition to osteosynthesis using a T-plate. Nineteen of 20 patients went on to union at an average of 4 months and demonstrated significant pain relief and improvement in shoulder flexion from 60° to 131°. The patients had six excellent and six good results, but 3 patients sustained tibial fractures following graft harvest and authors recommended against future use of this graft site [42].

The technique of using a fibular strut allograft as an intramedullary implant provides some inherent stability when the graft is impacted into the humeral head, and improves proximal screw purchase as described by Badman and Mighell, but without the graft donor site morbidity noted above. Badman and Mighell published the results of 18 patients who underwent intramedullary fibular strut allografting and stabilization of proximal humerus nonunions using locked plating. 17 patients (94%) achieved union at an average of 5.4 months. The remaining patient was a heavy smoker who had failed two previous attempts at surgical stabilization and ultimately required a hemiarthroplasty. Postoperative assessment of shoulder motion showed average flexion to 115°, external rotation to 37°, and internal rotation to the 10th thoracic vertebra. ASES scores improved from 40 preoperatively to 81 and analog pain scores improved from 6.7 to 1.5. Complications involved two posterior cord brachial plexus palsies that improved within 3 months and two cases of adhesive capsulitis, both requiring arthroscopic capsular release [26].

### *4.7.5.2 Biologic Augmentation*

Augmentation with the recombinant human bone morphogenic protein rhBMP-2 can be used for nonunions when biological activity is felt to be lacking. The current literature supports the use of BMP for acute open tibia fractures [43], tibial nonunions [44], and recalcitrant long bone nonunions [45], but no studies have reported on its use for nonunions of the proximal humerus. A Cochrane review of BMP use for fracture healing in adults

concluded that there is a paucity of data currently available and its role in treating nonunions remains unclear. Furthermore, they highlighted the high risk of bias in these studies due to industry involvement [46]. The U.S. Food and Drug Administration has granted rhBMP-7 (OP-1) a humanitarian device exemption (HDE) for treating recalcitrant long bone nonunions when autograft is unfeasible and alternative treatments have failed [47], but rhBMP-7 cannot be used “off label.” The FDA has approved rhBMP-2 for use in acute open tibia fractures, but its use for proximal humerus nonunions must be recognized as “off label.” Therefore, the use of biologic augments such as rhBMP-2 should be carefully weighed in light of the additional cost and unsubstantiated efficacy for treating proximal humerus nonunions compared to local or iliac crest autograft [8].

### 4.7.6 Unreamed Intramedullary Rods

The use of unreamed intramedullary implants (Rush rods) for the treatment of proximal humerus nonunions led to less favorable results. Five patients in a series presented by Healy et al. were treated using intramedullary implants: two with Rush rods alone, one using Rush rods with a tension band, one with an Ender nail, and one with a Lottes nail. Only one patient united their nonunion and all had poor functional results. The average shoulder motion was 40° of flexion and 10° of external rotation [38]. Duralde et al. presented a retrospective review of 20 patients with nonunions of the surgical neck, including 10 treated with open reduction and internal fixation and 10 with hemiarthroplasty based on an intraoperative assessment of the suitability of the fracture fragments for fixation. The fixation construct utilized Enders rods with a nonabsorbable suture or wire tension band and iliac crest bone grafting of the nonunion site with an onlay of cortical bone graft surrounding the nonunion site. Five of the 10 patients achieved union at an average of seven months. Of the remaining five, two were converted to a hemiarthroplasty, one underwent revision ORIF with a free fibular graft, one developed a deep infection and underwent removal of hardware and humeral head resection, and the final patient refused further surgical intervention. Two of the five that achieved union had an excellent result and three had a satisfactory result. Three later underwent reoperation for removal of painful prominent hardware. All five that failed to achieve union had unsatisfactory results [17].

Nayak et al. reviewed 17 patients who underwent operative intervention for nonunions of the surgical neck: 10 were treated with open reduction and

internal fixation using Rush rods, tension banding, and bone graft, and seven had a hemiarthroplasty. Two of the 10 patients who had Rush rods had persistent lucencies on imaging indicating a failure to unite. The Rush rod group had a better average postoperative range of motion than the hemiarthroplasty group with more elevation (140° vs. 110°), but complications were common in this group. Intraoperative complications included one case of circumflex artery laceration, one permanent axillary nerve injury, and one cortical perforation with a Rush rod. Postoperatively, two patients had radiographic evidence of avascular necrosis of the humeral head and all had symptoms consistent with impingement of the Rush rods. Eight patients had hardware removal after achieving union [19]. No study has been able to reproduce the union rate of 92% obtained by Neer, who treated 13 patients using unlocked intramedullary implants with rotator cuff tension banding. All patients required a second surgery to remove their prominent, symptomatic hardware [21].

### 4.7.7 Interlocking Intramedullary Nails

The availability of more modern interlocked intramedullary nails has increased the union rates and decreased the rate of symptomatic hardware when intramedullary implants are used for the treatment of nonunions (Fig. 4.3a–h). Yamane et al. reviewed 13 patients who underwent surgical stabilization using an interlocking intramedullary nail with bone grafting. The intraoperative technique involved locking the nail with two proximal and two distal screws and emphasized seating the nail below the subchondral surface of the humeral head to avoid impingement. Patients with large bone voids underwent cancellous iliac crest autografting to fill the void, while smaller defects were filled with tricalcium phosphate cement. All patients achieved union without evidence of malunion or avascular necrosis. Japanese Orthopedic Association shoulder scores averaged 85 points postoperatively, with four excellent, seven good, and two fair results. Postoperative shoulder range of motion demonstrated flexion to 122° and external rotation to 35°. The only complication was the backing out of proximal interlocking screws, which required removal in 2 patients. It should be noted that 11 of 13 patients treated in this study had not previously undergone surgical treatment and the two who had been treated operatively underwent percutaneous pinning or intramedullary nailing, so it is unclear if this protocol would achieve similar results in patients who had previously undergone fixation with plating

constructs [16].

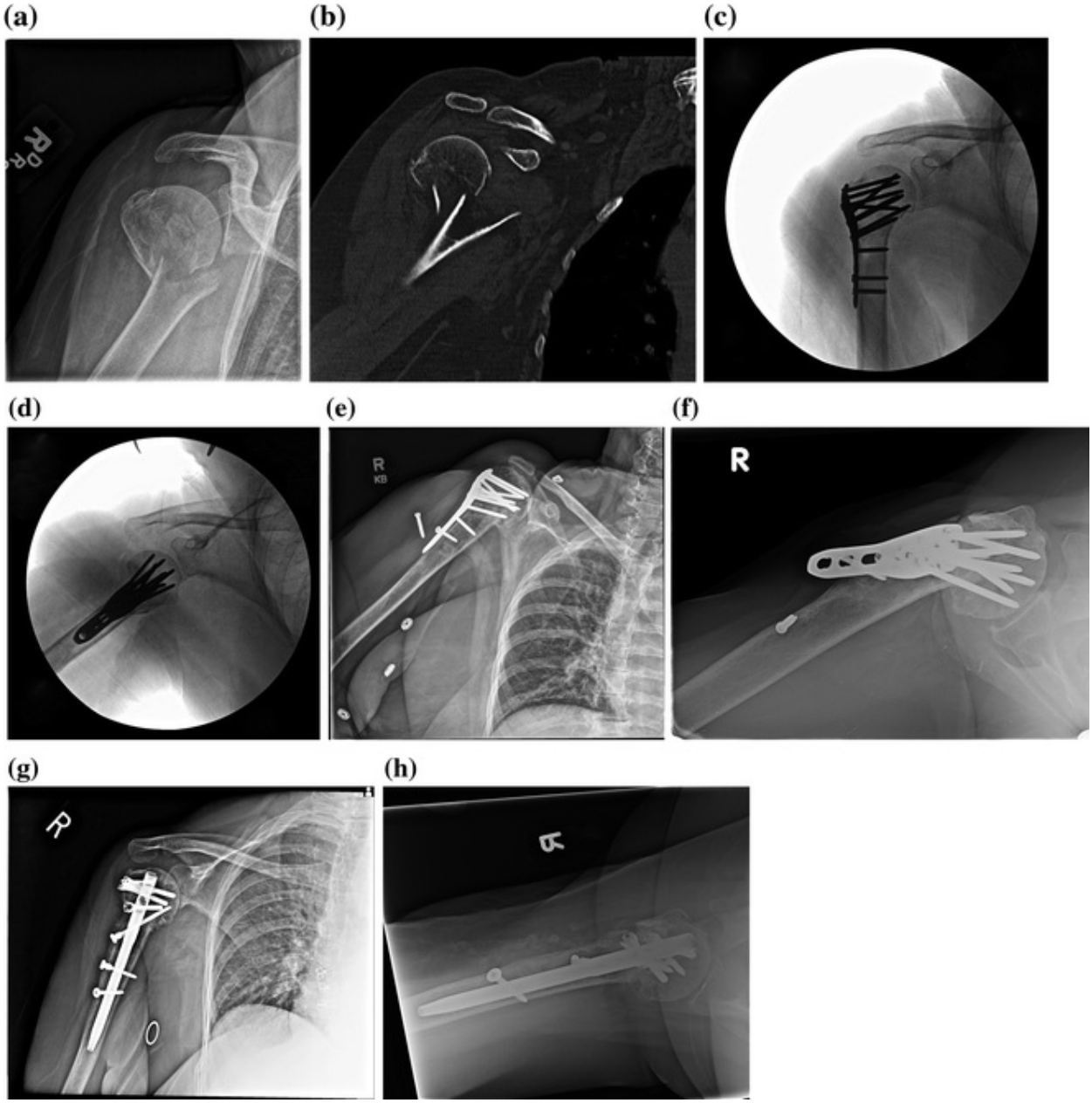



***Fig. 4.3*** A 60-year-old female sustained a surgical neck fracture after a ground-level fall, seen on anteroposterior (AP) (**a**) and coronal computed tomography (**b**). Underwent open reduction and internal fixation with locked plating and allograft on AP (**c**) and lateral (**d**) views. Presented two years postoperatively with continued pain and immobility with varus collapse and hardware pullout on AP (**e**) and axillary (**f**) views. Patient underwent hardware removal and revision fixation, using an interlocked intramedullary nail, allograft, and bone morphogenic protein, with union at three months on AP (**g**) and axillary (**h**) views

### 4.7.8 Summary of Internal Fixation Devices

Technological advances in implant design offer the potential for improved union rates and decreased postoperative hardware prominence for nonunion patients treated with osteosynthesis. The need for adequate bone stock and a viable humeral head without significant glenohumeral arthritis has not been obviated by the availability of modern implants. Regardless of the implant used, the preparation of the nonunion site with resection of fibrous tissue and avascular bone is critical. Bone loss at the nonunion site and humeral head cavitation are commonly encountered challenges and autograft bone grafting is widely utilized to address these issues, although some authors have achieved union of proximal humerus nonunions using allograft, tricalcium phosphate cements, or without augmentation. Locking plate technology and intramedullary fibular strut allografts have lessened, but certainly not eliminated the difficulties associated with the osteopenia commonly encountered in surgical neck nonunions. A careful balance should be struck between the improved stability achieved through compression at the fracture site and the deltoid weakness associated with over shortening between the articular surface and the deltoid insertion. Varus alignment is often a progressiDeepika Uve deformity and should be carefully avoided. Newer interlocking intramedullary nails increase the stability at the nonunion site compared to Rush and Ender's rods and may be inserted with decreased soft tissue stripping compared to plating constructs. These advances have served to increase the number of proximal humerus nonunions that can be treated with internal fixation rather than an arthroplasty.

---

## 4.8 Arthroplasty

Proximal humeral nonunions with bone stock that is insufficient to achieve adequate internal fixation or in which the humeral head is avascular are felt to be more amenable to arthroplasty than surgical stabilization. There are fracture characteristics that dictate which arthroplasty option is most suitable. A hemiarthroplasty requires tuberosity integrity and a functional rotator cuff to achieve optimal results. When significant glenoid arthritis is present patients may be better treated with a total shoulder arthroplasty. Patients who need an arthroplasty but do not have functional or repairable rotator cuffs or tuberosities may be candidates for a reverse total shoulder arthroplasty, but a

competent deltoid and axillary nerve is a requirement.

### 4.8.1 Unconstrained Arthroplasty

Several older series reviewed the results of patients who underwent treatment for proximal humerus nonunions using a variety of modalities, including hemiarthroplasty. These studies included patients who underwent an assortment of fixation strategies in addition to hemiarthroplasty and were not randomized. Healy et al. presented a series of 25 patients that included six patients who underwent a hemiarthroplasty with rotator cuff repair if necessary due to either inadequate bone stock for internal fixation or significant articular involvement. These patients had good pain relief but postoperative shoulder motion only averaged 72° of flexion and 30° of external rotation (compared to averages of 110° of flexion and 33° of external rotation among patients undergoing fixation with plating constructs). Despite these limitations in motion, 50% of these patients were rated as having good functional results [38].

Duralde et al. presented a series that included 10 patients who underwent hemiarthroplasty because of head cavitation, severe osteoporosis, or glenohumeral arthritis. Comparison of these patients with 10 patients who underwent ORIF of their proximal humerus nonunions showed no difference in motion, function, or pain relief. Overall functional results showed three excellent, three satisfactory, and four unsatisfactory results, but among the patients who achieved tuberosity union, only one patient had an unsatisfactory outcome. Hemiarthroplasty patients had a 70% rate of tuberosity union and two patients needed further surgery to address tuberosity nonunions. Other complications within the hemiarthroplasty group included two dislocations and one patient with asymmetric glenoid wear due to a humeral component that was cemented too proudly [17].

Nayak et al. presented seven patients treated with hemiarthroplasties for nonunions of the surgical neck and compared them to 10 patients undergoing ORIF using Rush rods, tension banding, and bone grafting. Patients treated with hemiarthroplasties had one grade more effective pain relief but their shoulder flexion averaged 110°, compared to 140° for the internal fixation patients. Functional improvement and UCLA rating scores were similar between the two groups. All patients were able to perform activities of daily living independently but no patient in either group returned to their pre-injury level of function. Complications included one axillary nerve palsy, two

asymptomatic inferior subluxations, and two cases of impingement syndrome that improved with corticosteroid injections. Tuberosity union was not quantified but the authors attributed several patients with the inability to elevate beyond 90° to suspected tuberosity nonunion [17]. These studies demonstrate the effectiveness of a hemiarthroplasty for pain relief but the amount of shoulder range of motion that is regained is unpredictable and less than that regained following successful osteosynthesis.

One study reviewed the results of unconstrained shoulder arthroplasties (either hemiarthroplasty or total shoulder arthroplasty) for the sequelae of previous proximal humerus fractures. Surgical neck nonunions represented a small cohort (six patients) of the 71 patients in this study who presented with AVN and head collapse, chronic dislocations, or tuberosity malunions. All the nonunions were treated with hemiarthroplasties used greater tuberosity osteotomies. These patients had unsatisfactory results with Constant score pain ratings of 7 (out of 15) and disappointing improvements in shoulder flexion from 50° preoperatively to 63° postoperatively. Combining these patients with others involved in the study, the most significant predictor of a poor outcome was the need for a greater tuberosity osteotomy, which was ubiquitous among nonunions, and caused the authors to recommend osteosynthesis rather than arthroplasty in treating these injuries [48]. The same authors later published a larger review including 22 surgical neck nonunions showing similarly disappointing results with Constant scores improving only from 21 to 36. They reiterated that they considered a surgical neck nonunion a relative contraindication for an unconstrained prosthesis. They cited the need for a tuberosity osteotomy, the difficulties achieving tuberosity union, and the poor functional results that followed as reasons to avoid arthroplasty. Alternatively, they recommended intramedullary peg grafting and osteosynthesis if sufficient humeral head bone stock existed or utilizing a low-profile prosthesis with large amounts of autografting to improve the likelihood of tuberosity union if osteosynthesis was not possible [49].

Two more recent studies reviewed the use of unconstrained shoulder arthroplasty for the treatment of nonunions of the proximal humerus and have focused on identifying the risk factors for unsatisfactory outcomes. Antuña et al. reviewed 25 patients treated with 21 hemiarthroplasties and four total shoulder arthroplasties for nonunions of the proximal humerus. An unconstrained arthroplasty was selected for each of these patients due to:

failure of previous fixation constructs leaving insufficient bone stock, head cavitation, significant osteoporosis, or advanced glenohumeral arthritis. Pain consistently improved for all patients treated with a hemiarthroplasty or a TSA. Range of motion averaged 88° of abduction, 38° of external rotation, and internal rotation to L3 post operatively. Twenty of 25 patients considered themselves either much better or better than preoperatively. Neer functional results were: one excellent, 11 satisfactory, and 13 unsatisfactory results, for an unsatisfactory rate greater than 50%. No subgroup analysis was performed to compare hemiarthroplasty to total shoulder arthroplasty patients, but, two of the patients with unsatisfactory results who felt they were worse after surgery were found to have advanced glenoid arthritis and yet were treated with hemiarthroplasties. This points out the importance of glenoid inspection when choosing hemiarthroplasty over TSA. The authors found that issues with tuberosity healing were the most common complications: nine patients had tuberosity resorption, two had nonunions, and one had a malunion, for an overall tuberosity complication rate of 48%. The authors emphasized using heavy nonabsorbable sutures, bone graft to fill gaps between the tuberosities and the shaft, and restricting shoulder range of motion post operatively in an effort to minimize the risk of this complication. Additional complications included one periprosthetic fracture and one dislocation, both of which required surgical intervention. There was also a significant correlation between increasing number of fracture parts using the Neer classification and less pain relief, as well as less subjective satisfaction with the procedure [15].

Duquin and colleagues reviewed the Mayo Clinic experience treating 67 proximal humerus nonunions with unconstrained arthroplasties. Their results were similar to those published by Antuña – pain was reliably decreased but motion was less predictable, with average elevation of 104° and external rotation of 50°. Neer functional ratings showed 11 excellent and 21 satisfactory results, but greater than 50% unsatisfactory results. There were no differences identified between hemiarthroplasties and TSA or press-fit and cemented humeral prostheses. Tuberosity healing was reviewed: 17 (25%) healed anatomically, 18 (27%) malunited with ≥5 mm displacement, 18 (27%) developed a nonunion, and 14 (21%) were resorbed or resected. Active elevation was significantly decreased for patients with tuberosity nonunions but the same patients did not have more pain or worse Neer functional scores. Bone grafting did not prevent tuberosity nonunions. Other complications included 11 severe subluxations or dislocations, two deep infections, and one

late periprosthetic fracture [24].

### 4.8.2 Reverse Total Shoulder Arthroplasty

The pain relieving benefit of unconstrained arthroplasty when treating proximal humerus nonunions has been tempered by the unpredictable results of glenohumeral motion. The strong association between postoperative range of motion and tuberosity healing has lead some to suggest reverse total shoulder arthroplasty as a viable alternative to hemiarthroplasty and standard total shoulder arthroplasty, due to its decreased reliance on greater tuberosity healing. Martinez et al. reviewed 18 patients who underwent reverse total shoulder arthroplasty for the treatment of proximal humerus nonunions. Cancellous autograft from the resected humeral head or, in cases of significant bone loss, humeral cortical allograft was used to augment resorbed bone. Constant scores and subjective shoulder scores increased postoperatively to 55 and 50% of the contralateral uninvolved side, respectively. Range of motion averaged 90° of flexion, 85° of abduction, 30° of external rotation, and 55° of internal rotation. Patient satisfaction was reported with eight very satisfied, six satisfied, and four unsatisfied patients. Complications included one transient axillary nerve palsy, two deep infections, and two dislocations. The authors stressed that in the setting of a proximal humerus nonunion in an elderly patient, the rotator cuff is often functionally insufficient due to the combination of trauma and chronic disuse, making a reverse TSA an excellent option because it relies on the deltoid rather than a competent rotator cuff to achieve elevation and abduction. Conversely, a functioning deltoid is a prerequisite for reverse TSA and in the same patient population disuse can make assessment difficult, so the authors recommend electromyography if concerns related to deltoid function exist [23].

### 4.8.3 Summary of Arthroplasty Options

Arthroplasty options serve as a last resort for patients whose proximal humerus nonunions have left them with advanced glenohumeral arthritis, severe osteopenia, and insufficient bone stock. These procedures offer reliable pain relief, but are associated with high complication rates and, for unconstrained arthroplasties, less predictable shoulder range of motion that is tied, at least in part, to tuberosity healing. Reverse total shoulder

arthroplasties avoid reliance on tuberosity healing but require both axillary nerve and deltoid function and have shorter longevity, allowing them only to be recommended for physiologically elderly patients.

## 4.9 Conclusion

Fractures of the proximal humerus are common and the majority of them unite uneventfully, many without surgical intervention. A small percentage develop into nonunions but the cited rates are often inflated by statistics yielded from tertiary referral centers. The small study sizes available in the literature also cause difficulty in determining the true rate of nonunion due to the infrequency of its occurrence. Nonunions of the proximal humerus present unique challenges due to biologic insults from the initial injury and previous surgeries, bone loss, humeral head cavitation, osteopenia, soft tissue contractures, and infection. Risk factors for developing a nonunion include fracture characteristics, such as increased fracture translation and metaphyseal comminution, but the relationship between these parameters and the rate of nonunion is not simple. Patient-related risk factors include nutritional or metabolic deficiencies, smoking, and medical comorbidities.

Patients developing nonunions can generally be identified as early as three months after injury based on their restricted range of motion, lower Constant scores, and greater difficulty with returning to performing activities of daily living, when compared to their healing counterparts. Once a nonunion has been identified, every effort should be made to address the problem before six months after the initial injury and prior to glenohumeral soft tissue contractures develop which may further limit the patient's function. If this window is missed, any proposed surgical plan should include an intraoperative assessment of soft tissue constraints to glenohumeral motion, which should be addressed.

Treatment options vary widely and range from nonsurgical management for minimally symptomatic patients to surgical options including osteosynthesis with standard, fixed angle, or locked plates, unreamed or interlocked intramedullary implants, and arthroplasty using hemiarthroplasties, total shoulders, or reverse total shoulders. Surgery may include augments such as cancellous autograft and allograft or structural grafts. From a functional perspective, postoperative Constant scores and range of motion are higher if successful union can be achieved with

osteosynthesis compared to arthroplasty options. Therefore, fracture characteristics that lend themselves to fixation are felt to be better prognostic indicators, and include simpler patterns, better bone stock, and maintained vascularity. Patient factors that allow more aggressive rehabilitation protocols postoperatively, such as younger age and less medical comorbidity, may also predict better functional outcomes, but the small studies available have not examined these variables specifically. Technological advances in locking plate and interlocking nail design have expanded the nonunions that may be amenable to osteosynthesis and every effort should be made to stabilize them. When these options are not appropriate, arthroplasty offers favorable results for pain control but less predictable range of motion, which seems to be at least partially dependent on achieving tuberosity union. Reverse total shoulder arthroplasties offer the theoretical advantage of decreased dependence on tuberosity union but only one small study has reviewed results from their use with proximal humeral nonunions and more research is needed to better elucidate the role for reverse TSA in treating the challenges of proximal humeral nonunions.

---

# References


1. Court-Brown CM, Caesar B. Epidemiology of adult fractures: a review. Injury. 2006;37(8):691–7. [Crossref][PubMed]

2. Lind T, Krøner K, Jensen J. The epidemiology of fractures of the proximal humerus. Arch Orthop Trauma Surg. 1989;108(5):285–7. [Crossref][PubMed]

3. Court-Brown CM, Garg A, McQueen MM. The epidemiology of proximal humerus fractures. Acta Orthop Scand. 2001;72(4):365–71. [Crossref][PubMed]

4. Lee CK, Hansen HR. Post-traumatic avascular necrosis of the humeral head in displaced proximal humerus fractures. J Trauma. 1981;21(9):788–91. [Crossref][PubMed]

5. Jensen GF, Christiansen C, Boesen J, Hegedüs V, Transbøl I. Relationship between bone mineral content and frequency of postmenopausal fractures. Acta Med Scand. 1983;213(1):61–3. [Crossref][PubMed]

6. Palvenen M, Kannus P, Niemi S, Parkkari J. Update in the epidemiology of proximal humeral fractures. Clin Orthop Relat Res. 2006;442:87–92. [Crossref]

7. Robinson CM. Proximal humerus fractures. In: Buchholz RW, Court-Brown CM, Heckman JD, Tornetta III P, editors. Rockwood and Green's fractures in adults. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2010. p. 1039–105.

8. Cadet ER, Yin B, Schulz B, Ahmad CS, Rosenwasser MP. Proximal humerus and humeral shaft nonunions. J Am Acad Orthop Surg. 2013;21(9):538–47.
[PubMed]

9. Warner JP, Costouros JG, Gerber C. Fractures of the proximal humerus. In: Buchholz RW, Heckman JD, Court-Brown C, editors. Rockwood and Green's fractures in adults. 6th ed. Philadelphia: Lippincott, Williams & Wilkins; 2006. p. 1039–105.

10. Court-Brown CM, McQueen MM. Nonunions of the proximal humerus: Their prevalence and functional outcome. J Trauma. 2008;64(6):1517–21.
[Crossref][PubMed]

11. Iyengar JJ, Devcic Z, Sproul RC, Feeley BT. Nonoperative treatment of proximal humerus fractures: a systematic review. J Orthop Trauma. 2011;25(10):612–7.
[Crossref][PubMed]

12. Sproul RC, Iyengar JJ, Devcic Z, Feeley BT. A systemic review of locking plate fixation of proximal humerus fractures. Injury. 2011;42(4):408–13.
[Crossref][PubMed]

13. Galatz LM, Iannotti JP. Management of surgical neck nonunions. Orthop Clin North Am. 2000;31(1):51–61.
[Crossref][PubMed]

14. Hanson B, Neidenbach P, de Boer P, Stengel D. Functional outcomes after nonoperative management of fractures of the proximal humerus. J Shoulder Elbow Surg. 2009;18(4):612–21.
[Crossref][PubMed]

15. Antuña SA, Sperling JW, Sánchez-Sotelo J, Cofield RH. Shoulder arthroplasty for proximal humerus nonunions. J Shoulder Elbow Surg. 2002;11:114–21.
[Crossref][PubMed]

16. Yamane S, Suenaga N, Oizumi N, Minami A. Interlocking intramedullary nailing for nonunion of the proximal humerus with Straight Nail System. J Shoulder Elbow Surg. 2008;17(5):755–9.
[Crossref][PubMed]

17. Duralde XA, Flatow EL, Pollock RG, Nicholson GP, Self EB, Bigliani LU. Operative treatment of nonunions of the surgical neck of the humerus. J Shoulder Elbow Surg. 1996;5(3):169–80.
[Crossref][PubMed]

18. Cheung EV, Sperling JW. Management of proximal humeral nonunions and malunions. Orthop Clin N Am. 2008;39(4):475–82.
[Crossref]

19. Nayak NK, Schickendantz MS, Regan WD, Hawkins RJ. Operative treatment of nonunion of surgical neck fractures of the humerus. Clin Orthop. 1995;313:200–5.

20. Rooney PJ, Cockshott WP. Pseudathrosis following proximal humeral fractures: a possible mechanism. Skeletal Radiol. 1986;15(1):21–4.
[Crossref][PubMed]

21. Neer CS II. Nonunion of the surgical neck of the humerus. Orthop Trans. 1983;7:389.

22. Checchia SL, Doneux P, Miyazaki AN, Spir IA, Bringel R, Ramos CH. Classification of non-unions of the proximal humerus. Int Orthop. 2000;24(4):217–20.
[Crossref][PubMed][PubMedCentral]

23. Martinez AA, Bejarano C, Carbonel I, Iglesias D, Gil-Albarova J, Herrera A. The treatment of proximal humerus nonunions in older patients with reverse shoulder arthroplasty. Injury. 2012;43(Suppl 2):S3–6.
[Crossref][PubMed]

24. Duquin TR, Jacobson JA, Sanchez-Sotelo J, Sperling JW, Cofield RH. Unconstrained shoulder arhtroplasty for treatment of proximal humeral nonunions. J Bone Joint Surg Am. 2012;94(17):1610–7.
[Crossref][PubMed]

25. Cofield RH. Total shoulder arthroplasty with the Neer prosthesis. J Bone Joint Surg Am. 1984;66(6):899–906.
[Crossref][PubMed]

26. Badman B, Mighell M, Drake G. Proximal humeral nonunions: surgical technique with fibular strut allograft and fixed-angle locked plating. Tech Shoulder Elbow Surg. 2006;7(2):95–101.
[Crossref]

27. Scheck M. Surgical treatment of nonunions of the surgical neck of the humerus. Clin Orthop. 1982;167:255–9.

28. Brinker MR, O'Connor DP. Nonunions: evaluation and treatment. In: Browner BD, Jupiter JB, Levine AM, Trafton PG, Krettek C, editors. Skeletal trauma: basic science, management, and reconstruction. 4th ed. Philadelphia: Saunders Elsevier; 2009. p. 615–708.
[Crossref]

29. Brinker MR, O'Connor DP, Monla YT, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trama. 2007;21(8):557–70.
[Crossref]

30. Kazakos K, Lyras DN, Galanis V, Verettas D, Psillakis I, Chatzipappas C, Xarchas K. Internal fixation of proximal humerus fractures using the polarus intramedullary nail. Arch Orthop Trauma Surg. 2007;127(7):503–8.
[Crossref][PubMed]

31. Norris TR, Turner JA, Bovil D. Nonunion of the upper humerus: an analysis of the etiology and treatment in 28 cases. In: Post M, Morrey BF, Hawkins RJ, editors. Surgery of the shoulder. Chicago: Mosby Year Book; 1990. p. 63–7.

32. Beredjiklian PK, Iannotti JP, Norris TR, Williams GR. Operative treatment of malunion of a fracture of the proximal aspect of the humerus. J Bone Joint Surg Am. 1998;80(10):1484–97.

[Crossref][PubMed]

33. Rockwood CA, Pearce JC. Management of proximal humerus nonunions. Orthop Trans. 1989;13:644.

34. Clavert P, Adam P, Bevort A, Bonnomet F, Kempf JF. Pitfalls and complication with locking plate for proximal humerus fracture. J Shoulder Elbow Surg. 2010;19(4):489–94. [Crossref][PubMed]

35. Thanasas C, Kontakis G, Angoules A, Limb D, Giannoudis P. Treatment of proximal humerus fractures with locking plates: a systematic review. J Shoulder Elbow Surg. 2009;18(6):837–44. [Crossref][PubMed]

36. Gerber C, Hersche O, Berberat C. The clinical relevance of posttraumatic avascular necrosis of the humeral head. J Shoulder Elbow Surg. 1998;7(6):586–90. [Crossref][PubMed]

37. Wijgman AJ, Roolker W, Patt TW, Raaymakers El, Marti RK. Open reduction and internal fixation of three and four-part fractures of the proximal part of the humerus. J Bone Joint Surg Am. 2002;84-A(11):1919–25.

38. Healy WL, Jupiter JB, Kristiansen TK, White RR. Nonunion of the proximal humerus. J Orthop Trauma. 1990;4(4):424–31. [Crossref][PubMed]

39. Ring D, McKee MD, Perey BH, Jupiter JB. The use of a blade plate and autogenous cancellous bone graft in the treatment of ununited fractures of the proximal humerus. J Shoulder Elbow Surg. 2001;10(6):501–7. [Crossref][PubMed]

40. Allende C, Allende BT. The use of a new locking 90 degree blade plate in the treatment of atrophic proximal humerus nonunions. Int Orthop. 2009;33(6):1649–54. [Crossref][PubMed]

41. Tauber M, Brugger A, Povacz P, Resch H. Reconstructive surgical treatment without bone grafting in nonunions of humeral surgical neck fractures. J Orthop Trauma. 2011;25(7):392–8. [Crossref][PubMed]

42. Walch G, Badet R, Nove-Josserand L, Levigne C. Nonunions of the surgical neck of the humerus: surgical treatment with an intramedullary bone peg, internal fixation, and cancellous bone grafting. J Shoulder Elbow Surg. 1996;5(3):161–8. [Crossref][PubMed]

43. Govender S, Csimma C, Genant HK, Valentin-Opran A, Amit Y, Arbel R, et al. BMP-1 evaluation in surgery for tibial trauma (BESTT) study group. Recombinant human bone morphogenic protein-2 for treatment of open tibial fractures: a prospective, controlled, randomized study of four hundred and fifty patients. J Bone Joint Surg Am. 2002;84(12):2123–34. [Crossref][PubMed]

44. Friedlander GE, Perry CR, Cole JD, Cook SD, Cierny G, Muchler GF, et al. Osteogenic protein-1 (bone morphogenic protein-7) in the treatment of tibial nonunions. J Bone Joint Surg Am. 2001;83-

A Suppl 1(Pt 2):S151–8.

45. Calori GM, Tagliabue L, Gala L, d'Imporzano M, Peretti G, Albisetti W. Application of rhBMP-7 and platelet-rich plasma in the treatment of long bone non-unions: a prospective randomized clinical study on 120 patients. Injury. 2008;39(12):1391–402.

46. Garrison KR, Shemilt I, Donell S, Ryder JJ, Mugford M, Harvey I, et al. Bone morphogenic protein (BMP) for fracture healing in adults. Cochrane Database Syst Rev. 2010;16(6):CD006950.

47. Schultz DG, U.S. Food and Drug Administration. Public health notifications (medical devices). FDA public health notification: life-threatening complications associated with recombinant human bone morphogenetic protein in cervical spine fusion. 1 Jul 2008. Accessed 30 Sept 2015.

48. Boileau P, Trojani C, Walch G, Krishnan SG, Romeo A, Sinnerton R. Shoulder arthroplasty for the treatment of the sequelae of fractures of the proximal humerus. J Shoulder Elbow Surg. 2001;10(4):299–308.
[Crossref][PubMed]

49. Boileau P, Chuinard C, Le Huec JC, Walch G, Trojani C. Proximal humerus fracture sequelae: impact of a new radiographic classification on arthroplasty. Clin Orthop Relat Res. 2006;442:121–30.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_5

# 5. Supracondylar Humeral Nonunions

Joseph Borrelli Jr.[1]

(1) Orthopedic Surgery, BayCare Medical Group, St. Joseph's Hospital-North, 4211 Van Dyke Rd, Ste 200, Lutz, FL 33558, USA

**Joseph Borrelli Jr.**
**Email:** joseph.borrelli@BayCare.org

**Keywords** Nonunion – Supracondylar – Assessment – Treatment – Humerus

## 5.1 Introduction

Nonunions of the supracondylar area of the humerus following fracture are relatively uncommon but present the surgeon and the patient with a very challenging problem. Nonunions of the supracondylar area of the humerus often result in limited use of the upper extremity as well as pain and instability at the site and elbow stiffness [1, 2]. Risk factors for their development include, s oft tissue interposition at the time of the initial fracture, infection, mechanical instability, and/or poor blood supply at the fracture site, often resulting from excess soft tissue stripping at the time of surgery. Additionally, systemic vitamin D deficiency and overall poor nutrition and smoking have been associated with the development of long bone nonunions [3, 4].

## 5.2 Assessment

The assessment of patients with a supracondylar nonunion begins with a detailed history and physical examination . All patients, but particularly those

who have previously undergone surgery, should be assessed for the presence of infection as a cause of their nonunion. Screening laboratory tests to assess the potential presence of an infection includes a complete blood count, erythrocyte sedimentation rate, and C-reactive protein . The use of a combined white blood cell/sulfur colloid scan has been found to be the least predictive method of revealing infection and is not cost effective [5]. Of course, care must be taken in interpreting each of these tests, as false positives are possible in the setting of hypertrophic nonunions, particularly those with loose or broken metallic implants.

In general, a thorough history and physical examination combined with orthogonal plain radiographs centered on the supracondylar and elbow area in question is usually sufficient to make the diagnosis of a nonunion. Reviewing serial radiographs over time will often show widening of the fracture gap, loosening or breakage of the implants, and the development of irritation-type callus, all of which are consistent with the development of a nonunion. Recently, computer tomography (CT) scanning technology has improved to the point where, when the diagnosis of a nonunion is still in doubt, CT scans with sagittal and coronal reconstructions, and even 3D reconstructions, may be helpful in confirming the presence of a nonunion. Although studies of the use of CT scans for the diagnosis of distal humeral nonunions have not been published, there are studies supporting their use in the assessment of nonunions of the feet and following spinal fusions to assess healing [6, 7]. Magnetic resonance imaging (MRI) has not shown to be helpful in the diagnosis of long bone nonunions and should therefore be used sparingly in this setting.

## 5.3 Surgical Tactic

Once the diagnosis of a supracondylar humerus nonunion has been made, the most predictable method to restore stability and elbow function is generally to remove the previously placed implants and to perform revision open reduction and internal fixation (ORIF) [1, 2, 8–11]. When treating nonunions, efforts should be made to obtain good fragment apposition and interfragmentary compression between the fracture fragments while restoring absolute stability. This is often difficult because of the commonly small size of the distal fragment, which is further compromised by the presence of disuse osteopenia. In oligotrophic and atrophic nonunions, efforts should be

made not only to obtain good apposition and interfragmentary compression between the fracture fragments but also in the effort to improve the local biological environment. The addition of autologous bone grafts, allograft demineralized bone matrix (DBX), or other biologic stimulants to the nonunion site during revision ORIF has been shown to be helpful in obtaining union [11–13].

Generally, too distal to be effectively treated with intramedullary nails, nonunions of the supracondylar area of the humerus are commonly stabilized with a combination of plate(s) and screws [2, 11]. In the author's experience, nonunions of the distal humeral shaft (upper regions of the supracondylar area of the humerus) can be effectively treated with a single large fragment compression plate, as long as the plate can be placed distal enough to allow at least two to three bicortical screws (4–6 cortices) to be placed within the distal fragment without compromise to the olecranon fossa. Nonunions of the *true* supracondylar region of the distal humerus are generally treated with double plating after restoring articular congruity, if necessary, of the distal humerus. Anatomically specific plates are available with locking screw capability and are positioned either 90° to each other (posterolateral and medial) or parallel with both plates placed posteriorly, or one plate placed medially and one plate placed laterally. For nonunions of the supracondylar or intracondylar areas deemed non-reconstructible, particularly in the low-demand elderly patients, cemented total elbow arthroplasty offers a good solution to this difficult problem [14–16]. Occasionally, supracondylar nonunions with loss of distal bone stock require the use of a customized prosthesis, commonly used in the setting of a tumor resection [17].

The use of thin wire external fixation in the treatment of infected distal humeral nonunions has also been reported [18, 19]. However, the use of these complex external fixation devices should be kept in the hands of the most experienced surgeons who perform these procedures routinely and who fully understand the potential and limitations of these devices.

In the setting of an infected supracondylar humeral nonunion, a thorough irrigation and debridement to include the removal of all avascular and infected bone, all metallic implants, and any devitalized soft tissues must be undertaken. Temporary stability of the fracture fragments is advantageous during the antibiotic phase and can sometimes (ideally) be provided with the application of an external fixator inserted into the proximal and distal fragments, or spanning the elbow. The use of circular frames utilizing thin

wires also has a role here, although great care must be taken to avoid neurovascular structures when placing the transfixation wires in each of the fragments [18, 19]. The surrounding soft tissue envelope must also be addressed, as inadequate soft tissue coverage of the bone fragments and elbow joint will compromise the chances for infection eradication and ultimate healing of the nonunion. In addition to all of these considerations, the patient will also need a minimum of six weeks of bacterial-specific systemic antibiotics. Generally, administration of these powerful antibiotics should be performed in close consultation with an expert in infectious disease. Additionally, to bring additional antibiotics into the infected nonunion site, a polymethylmethacrylate spacer impregnated with a broad-spectrum antibiotic can be placed in the nonunion space and removed during the definitive fixation of the nonunion.

## 5.4 Surgical Approaches

In most cases where primary or revision ORIF of a supracondylar nonunion is being undertaken, the distal humerus is approached via a posterior incision , with the patient positioned either in the lateral decubitus or prone position. To gain access to the distal humerus, the exposure can be performed by developing planes medial and lateral to the distal triceps and its tendonitis insertion. Of course, this should only be done once the ulnar nerve has been identified and isolated so that it can be protected throughout the procedure. The distal humerus can also be approached through a triceps splitting approach or a triceps tendon turndown. If the exposure is needed proximal to the distal flare of the humerus, care must be taken to locate and protect the radial nerve and the profunda brachial artery as they pass from proximal medial to distal lateral and around the lateral aspect of the distal humerus [20]. If there is an intra-articular component of the distal humeral nonunion, then consideration must be given to performing an olecranon osteotomy to allow exposure and anatomic reduction of the articular component. If a total elbow arthroplasty is to be performed, then the elbow can generally be replaced through intervals medial and lateral to the distal triceps without elevating the triceps off the olecranon and proximal ulna and without an olecranon osteotomy [14–16]. Again, in each case, the ulnar nerve must be identified and carefully mobilized from around the medial epicondyle and protected. During each of these approaches for treatment of distal humeral

nonunions, transposition of the ulnar nerve is not necessarily transposed and, in fact, transposition is discouraged; a recent study has shown that transposition of the ulnar nerve following ORIF of acute distal humerus fractures has been associated with a fourfold increase in postoperative ulnar neuritis [21]. If the patient had signs and symptoms of ulnar nerve compression at the elbow preoperatively, or if there is obvious impingement of the ulnar nerve after revision of the nonunion or replacement of the elbow, ulnar nerve transposition may certainly be warranted.

At times, augmentation of the nonunion fixation construct may be desirable to support healing. In these cases, nonunion augmentation could include autologous bone graft , DBX or bone-stimulating proteins, or a combination of these [12]. If an autologous iliac crest bone graft is to be utilized, the iliac crest can be easily exposed with the patient in the lateral or prone position. Preferentially, the outer iliac table and the intra-table trabecular bone are harvested with an acetabular reamer, as previously described [22–25]. This technique provides finely minced pieces of cortical and cancellus bone to facilitate nonunion healing. Corticocancellous strips and cancellous bone fragments can also be harvested from the iliac crest, and at times from the proximal ulna, and placed at the nonunion site. Demineralized bone matrix (DBX) is available in several different forms and has been shown to augment healing of humeral nonunions [12]. Bone morphogenic proteins (BMP) have also been used to augment healing in certain nonunions, but in general their use in the treatment of humeral nonunions has not been approved [26–29]. However, BMP-7 has been approved for use in the setting of a persistent fracture nonunion. Use of these proteins in patients of childbearing age must be made with great caution.

## 5.5 Postoperative Rehabilitation

Generally, once the surgical incision has healed and the sutures have been removed, rehabilitation of the elbow, shoulder, and forearm is begun in earnest. Rehabilitation should include active and active assisted flexion and extension of the elbow, pronation and supination of the forearm, and range of motion of the shoulder. Particular attention should be paid to elbow extension and forearm supination, as these are often the most difficult motions to recover. Occasionally, dynamic and passive splinting devices in conjunction with therapist-supervised rehabilitation are necessary to help the patient

regain forearm and elbow range of motion [30, 31]. These techniques can usually be started within the first six to eight weeks once there is radiographic evidence of healing at the nonunion site. Also, at approximately 6 weeks postoperatively, physical therapist directed range of motion exercises and muscle-strengthening exercises can be initiated. The goal of the postoperative rehabilitation should be to achieve full flexion and extension of the elbow, full pronation and supination of the forearm, and the restoration of normal shoulder motion.

## 5.6 Complications

As with any surgical intervention the risk of complications is substantial and is increased as the number of surgeries performed on the distal humerus increases. These risks include infection, nerve damage, vascular injury, persistent nonunion, loss of fixation, and implant failure. In an effort to avoid infection, preoperative antibiotics should be administered immediately before tourniquet inflation and incision and for 24 h postoperatively. Also, meticulous soft tissue handling techniques and timely surgery while maintaining soft tissue attachments to the nonunion fragments should be adhered to in order to help decrease the incidence of deep infection. To avoid nerve injury the ulnar nerve and, on occasion, the radial nerve should be identified and isolated sufficiently to allow gentle retraction out of harm's way during reduction and fixation of the nonunion. Knowing preoperatively whether or not the ulnar nerve was transposed will assist greatly in its identification and protection during the procedure. Intra-operative use of a tourniquet or meticulous intra-operative hemostasis will minimize the risk of intra-operative blood loss and postoperative hematoma formation, which can delay soft tissue healing and put the patient at increased risk of infection. Persistence of the nonunion is not uncommon and is most likely the result of poor vascularity of the nonunion fragments, scarring, and hypovascular surrounding soft tissues and/or residual instability at the nonunion site. Therefore, all efforts must be made to obtain fragment apposition and interfragmentary compression at the time of revision surgery. Atrophic nonunions will benefit by the addition of biologically active materials (DBX, BMPs, iliac crest bone graft), while oligotrophic and hypertrophic nonunions can often be treated with reduction and interfragmentary compression alone. Of course, seldom is the outcome after revision ORIF for a nonunion

typically as good as after primary healing following the initial procedure.

## 5.7 Conclusion

Open reduction and internal fixation of distal humeral nonunions can be quite challenging. It is important to try and determine the cause of the nonunion so that these factors can be addressed and reversed. Typically, nonunions in this area are thought to occur as a result of soft tissue interposition (non-operatively treated fractures), fracture fragment instability, underlying infection, poor local vascular environment, or underlying systemic metabolic conditions. Before undertaking revision surgery, the surgeon should examine the patient thoroughly, the patient's elbow and forearm range of motion should be documented, and necessary images, to best define the nonunion, should be obtained. The presence of infection needs to be determined and a thorough preoperative plan to address each of the contributing factors developed. Prior to surgery, the surgeon must discuss the potential risks and benefits, as well as the limitations, of the proposed surgery with the patient. Surgery should be carried out with the goals to restore length, alignment, and rotation of the extremity, contracture release of the elbow, and to obtain fragment reduction and interfragmentary compression and stable fixation to allow early elbow and forearm range of motion and osseous healing. Bone graft or bone graft substitutes should be used liberally in the setting of oligotrophic or atrophic nonunions. Release and mobilization of the elbow and proximal radius and ulnar joints are essential to facilitate healing of the nonunion and recovery of forearm range of motion. Patients must be made aware that a fair percentage of these nonunions persist and additional surgery may be necessary.

## 5.8 Case Discussions

### Case 1

A 45-year-old male who sustained a gunshot wound of his distal arm and humerus resulting in a supracondylar humerus fracture. The patient was initially treated with irrigation and debridement and ORIF of his distal humerus through a posterior approach with parallel plates.

This fracture was repaired with parallel plates (Fig. 5.1), including a one-

third tubular plate laterally, and a locking specialty plate medially. A nonunion developed as indicated by the loose and failing fixation, the appearance of a gap at the previous fracture site, and considerable irritation callus along the medial and lateral humerus.


***Fig. 5.1*** Anteroposterior (**a**) and lateral (**b**) radiographs of a supracondylar humerus nonunion. Loss of fixation and alignment of the fracture fragments can be seen, as well as a gap at the previous fracture site

The nonunion (Fig. 5.2) was first assessed for the presence of infection and then treated with removal of the implants, debridement of the nonunion site, revision ORIF with 90–90 specialty plates. An olecranon osteotomy was performed to maximize exposure of the distal fragment and to aid in the mobilization of the elbow joint. The nonunion site was also augmented with demineralized bone matrix and allograft cancellous bone chips and the nonunion went onto heal uneventfully.

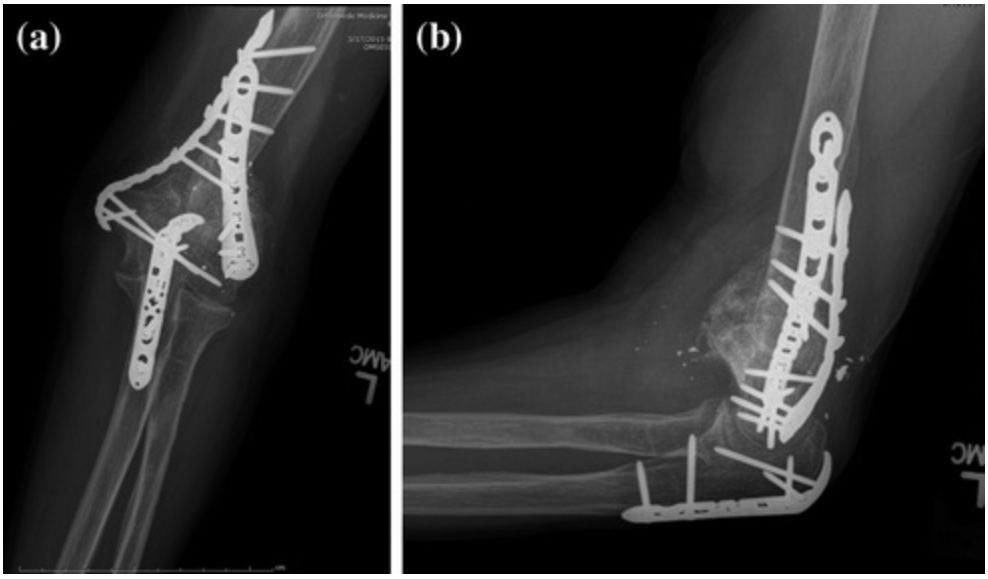



***Fig. 5.2*** Anteroposterior (**a**) and lateral (**b**) radiographs of the nonunion site, which has been reduced and stabilized with a medial and lateral plate and bone grafted with an iliac crest bone graft assisted by an olecranon osteotomy

## Case 2

A 53-year-old male fell at home sustaining a closed supracondylar intercondylar humeral fracture. He was originally treated with a closed reduction and splinting of the elbow and was subsequently treated with ORIF with an olecranon osteotomy. Early motion was begun at approximately 10 days postoperatively, and his incision healed uneventfully. Although his pain decreased during the first several weeks, at six weeks post-op, he felt a snap in the elbow and experienced increased pain and swelling of the elbow (Figs. 5.3, 5.4, 5.5 and 5.6).

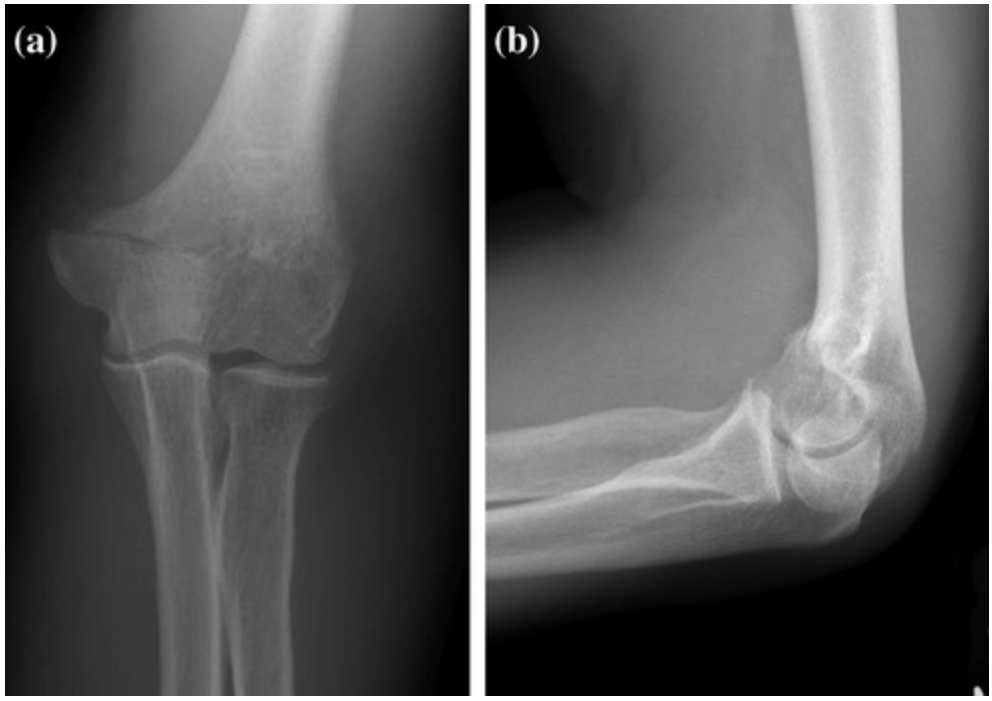



***Fig. 5.3*** Anteroposterior (**a**) and lateral (**b**) radiographs demonstrating a displaced supracondylar intercondylar humerus fracture


***Fig. 5.4*** Anteroposterior (**a**) and lateral (**b**) radiographs following open reduction and internal fixation of the displaced supracondylar intercondylar humerus fracture shown in Fig. 5.3

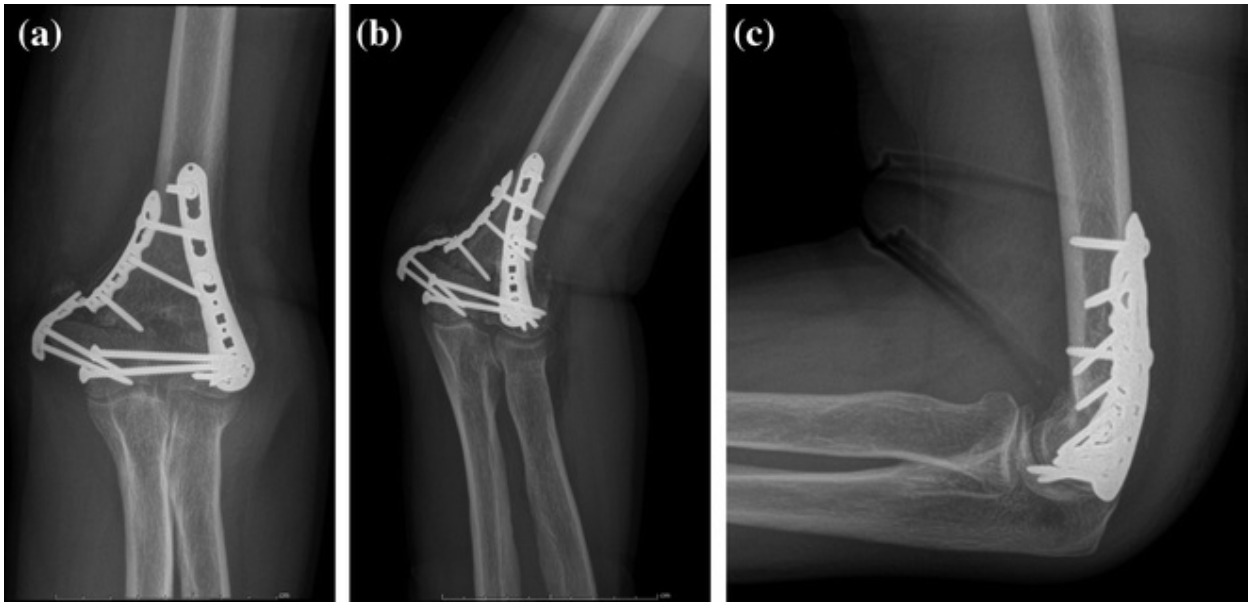



***Fig. 5.5*** Anteroposterior (**a**), oblique (**b**), and lateral (**c**) radiographs show failure of medial plate, an indication that a nonunion is developing

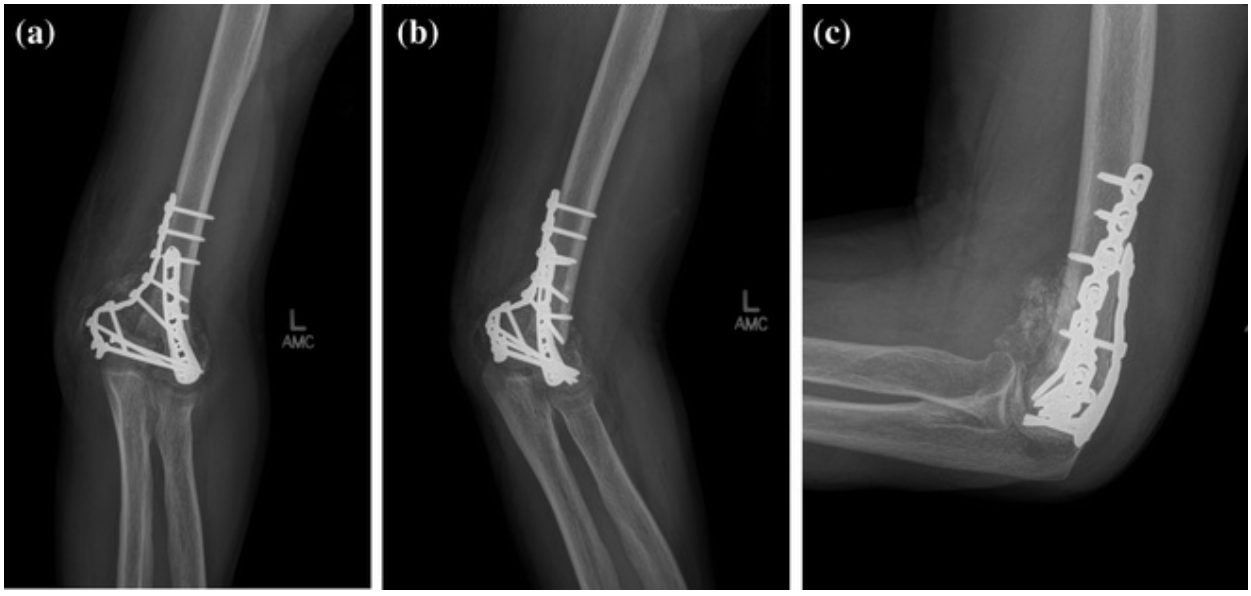



***Fig. 5.6*** Anteroposterior (**a**), oblique (**b**), and lateral (**c**) radiographs after removal of medial implants, reduction of the medial condyle, and revision open reduction and internal fixation with an iliac crest bone graft

# References

1. Ring D, Gullotta L, Jupiter JB. Unstable nonunions of the distal part of the humerus. J Bone Joint

Surg Am. 2003;85-A(6):1040–5.

2. Jupiter JB. The management of nonunion and malunion of distal humerus—30 year experience. J Ortho Trauma. 2008;22(10):742–50.
[Crossref]

3. Hak DJ, Fitzpatrick D, Bishop JA, Marsh JL, Tilp S, Schnettler R, et al. Delayed union and nonunions: epidemiology, clinical issues, and financial aspects. Injury. 2014;45(Suppl 2):S3–7.
[Crossref][PubMed]

4. Brinker MA, O'Connor DP, Monla YT. Metabolic and endocrine abnormalities in patients with nonunions. J Ortho Trauma. 2007;21(8):557–70.
[Crossref]

5. Stucken C, Olszewski DC, Creevy WR, Murakami AM, Tornetta P. Preoperative diagnosis of infection in patients with nonunions. J Bone Joint Surg Am. 2013;95(15):1409–12.

6. Carreon LY, Djurasovic M, Glassman SD, Sailer P. Diagnostic accuracy and reliability of fine-cut CT scans with reconstructions to determine the status of an instrumented posterolateral fusion with surgical exploration as reference standard. Spine. 2007;32(8):892–5.
[Crossref][PubMed]

7. Jones CP, Coughlin MJ, Shurnas PS. Prospective CT scan evaluation of hindfoot nonunions treated with revision surgery and low-intensity ultrasound stimulation. Foot Ankle Int. 2006;27(4):229–35.
[Crossref][PubMed]

8. Allende C, Allende BT. Post-traumatic distal humerus non-union: Open reduction and internal fixation: long-term results. Int Ortho. 2009;33(5):1289–94.
[Crossref]

9. Niu Y, Bai Y, Xu S, Wu D, Liu X, Wang P, et al. Treatment of bone nonunion and bone defects associated with unsuccessful humeral condylar fracture repair with autologous iliac bone reconstruction. J Shoulder Elbow Surg. 2012;21(8):985–91.
[Crossref][PubMed]

10. Helfet DL, Kloen P, Anand N, Rosen HS. Open reduction and internal fixation of delayed unions and nonunions of fractures of the distal part of the humerus. J Bone Joint Surg Am. 2003;85-A(1):33–40.

11. Helfet DL, Kloen A, Anand N, Rosen HS. ORIF of delayed unions and nonunions of distal humeral fractures. Surgical technique. J Bone Joint Surg Am. 2004;86-A (Suppl 1):18–29.

12. Hierholzer C, Sama D, Toro JB, Peterson M, Helfet DL. Plate fixation of ununited humeral shaft fracture: effect of type of bone graft on healing. J Bone Joint Surg Am. 2006;88(7):1442–7.
[PubMed]

13. Crawford CH 3rd, Seligson D. Atrophic nonunion of humeal diaphysis treated with locking plate and recombinant bone morphogenetic protein: nine cases. Am J Orthop. 2009;38(11):567–70.
[PubMed]

14. Mansat P, Bonnevialle N, Rongieres M, Mansat M, Bonnevialle P. Experience with the Coonrad-Morrey total elbow arthroplasty: 78 consecutive total elbow arthroplasties reviewed with an

average 5 years of follow-up. J Shoulder Elbow Surg. 2013;22(11):1461–8.
[Crossref][PubMed]

15. Espiga X, Antuna SA, Ferreres A. Linked total elbow arthroplasty as treatment of distal humerus nonunions in patient older that 70 years. Acta Ortho Belg. 2011;77(3):304–10.

16. Baksi DP, Pal AK, Baksi D. Prosthetic replacement of elbow for intercondylar fractures (recent of ununited) of humerus in elderly. Int Ortho. 2011;35(8):1171–7.
[Crossref]

17. Anmin A, Suresh S, Sanghrajka A, Cannon SR, Briggs TW, Unwin P. Custom-made endoprosthetic reconstruction of the distal humerus for non-tumorous pathology. Acta Orthop Belg. 2008;74(4):446–50.
[PubMed]

18. Brinker MR, O'Connor DP, Crouch CC, Mehlhoff TL, Bennett JB. Ilizarov treatment of infected nonunions of the distal humerus after failure of internal fixation: an outcomes study. J Orthop Trauma. 2007;21(3):178–84.
[Crossref][PubMed]

19. Ring D, Jupiter JB, Toh S. Salvage of contaminated fractures of the distal humerus with thin wire external fixation. Clin Orthop Relat Res. 1999;359:203–8.
[Crossref]

20. Gerwin M, Hotchkiss RN, Weiland AJ. Alterative operative exposures of the posterior aspect of the humeral diaphysis with reference to the radial nerve. J Bone Joint Surg Am. 1996;78(11):1690–5.
[Crossref][PubMed]

21. Chen RC, Harris DL, Leduc S, Borrelli J, Tornetta P, Ricci WM. Is ulnar nerve transposition beneficial during open reduction internal fixation of distal humerus fractures? J Ortho Trauma. 2010;24(7):391–4.
[Crossref]

22. Sanders R, DiPasquale T. A technique for obtaining bone graft. J Ortho Trauma. 1989;3(4):287–9.
[Crossref]

23. Westrich GH, Geller DS, O'Malley MJ, Deland JT, Helfet DL. Anterior iliac crest bone graft harvesting using the corticocancellous reamer system. J Ortho Trauma. 2001;15(7):500–5.
[Crossref]

24. Gossman DG, Rosenblum W, Arosarena O, Valentino J. The acetabular reamer: a unique tool for anterior iliac crest bone graft harvesting. Laryngoscope. 2005;115(3):557–9.
[Crossref][PubMed]

25. Brawley SC, Simpson RB. Results of an alternative autologous iliac crest bone graft method. Orthopedics. 2006;29(4):342–5.
[PubMed]

26. Murena L, Canton G, Vulcano E, Surace MF, Cherubino P. Treatment of humeral shaft aseptic nonunions in elderly patients with opposite structural allograft, BMP-7, and mesenchymal stem

cells. Orthopedics. 2014;37(2):201–5.
[Crossref]

27. Starman JS, Bosse MJ, Cates CA, Norton HJ. Recombinant human bone morphogenetic protein-2 use in the off-label treatment of nonunions and acute fractures: a retrospective review. J Trauma Acute Care Surg. 2012;72(3):676–81.
[Crossref][PubMed]

28. Giannoudis PV, Kanakaris NK, Dimitriou R, Gill I, Kolimarala V, Montgomery RJ. The synergistic effect of autograft and BMP-7 in the treatment of atrophic nonunions. Clin Orthop Relat Res. 2009;467(12):3239–48.
[Crossref][PubMed][PubMedCentral]

29. Bong MR, Capla EL, Egol KA, Sorkin AT, Distefano M, Buckle R, et al. Osteogenic protein-1 (bone morphogenic protein-7) combined with various adjuncts in the treatment of humeral diaphyseal nonunions. Bull Hosp Jt Dis. 2005;63(1–2):20–3.
[PubMed]

30. Lindenhovius AL, Doornberg JN, Brouwer KM, Jupiter JB, Mudgal CS, Ring D. A prospective randomized controlled trial of dynamic versus static progressive elbow splinting for posttraumatic elbow stiffness. J Bone Joint Surg Am. 2012;94(8):694–700.
[Crossref][PubMed]

31. Bash DS, Spur ME. An alternative to turnbuckle splinting for elbow flexion. J Hand Ther. 2000;13(3):237–40.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_6

# 6. Nonunions of the Forearm

Fred G. Corley[1] and Ben S. Francisco[1]

(1) Department of Orthopedics, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Dr., MC 7774, San Antonio, TX 78229, USA

**Fred G. Corley**
**Email:** corley@uthscsa.edu

**Keywords** Nonunion – Forearm – Radial shaft fracture – Ulnar shaft fracture – One-bone forearm

## 6.1 Introduction

Although nonunion of forearm fractures is an uncommon outcome, it does occur and proper treatment of the nonunion is essential to preserve function. The forearm itself is considered by many to be a functional unit, or even a joint, and like articular fractures, forearm nonunions should be restored to anatomic alignment.

## 6.2 Anatomy

### 6.2.1 Overview

The forearm is composed of the radius and ulna, and the “forearm joint” is the result of the interplay between these two bones at their two distinct articulations proximally and distally, the proximal radioulnar joint (PRUJ) and the distal radioulnar joint (DRUJ) . Interposed between these two joints and the two bones is the interosseous membrane (IOM) , which provides

stability to the forearm and transfers forces from the distal radius and ulna proximally, as well as stabilizing the PRUJ and DRUJ [1–4]. The coordinated action and stability provided by these three structures allow the forearm to function appropriately. The forearm axis of rotation passes from the center of the radial head through the fovea of the distal ulna [5], thereby allowing the mobile radius to rotate around the fixed ulna 150–180° as the forearm musculature moves the hand from pronation to supination. Working simultaneously with the carpus, this degree of freedom provides the forearm with the ability to position the hand intricately in space.

Various authors have determined forearm range of motion with 68–70° pronation and 75–85° supination being average [6–8]. However, Morrey et al. noted that only 55° of supination and 50° of pronation are needed for most activities of daily living, indicating that supination is needed more than pronation, one of the reasons for that being, if one abducts the shoulder, it essentially pronates the forearm.

### 6.2.2 Bony Anatomy

The radius is a long bone with proximal and distal epiphyses. At the proximal end, the radial head is nearly round and articulates with the ulna in its radial fossa and also with the capitellum of the humerus. As the radius moves distal, it narrows to form the radial neck. On its ulnar side, there is a bony prominence called the radial or bicpital tuberosity, where the biceps tendon inserts. In its midsubstance, the radius is triangular in shape. The ulnar border is the apex of the triangle and is the origin of the IOM.

In addition, the radius has three bows. At the distal one-fifth, the radius has a convex dorsal bow, and at the proximal one-fifth, the radius has a convex ventral bow. The middle three-fifths contain the most prominent radial bow. This bow corresponds to the insertion sites of the pronator teres and supinator muscles, and hence, the radial bow is vital in ensuring correct forearm rotation. A fracture in this area leads to the most disability if the fracture is not anatomically aligned, as the lever arms of these respective muscles are shortened [9]. Interestingly, in Burwell and Charnley's series of 231 fractures in 150 adult patients that 93% of radius fractures occurred in the middle three-fifths of the radius [10] and Sage and Smith reported that three-fifths of their radius fractures were found in the middle third [11].

Therefore, in forearm fractures, the radial bow must be restored or loss of rotation will ensue. Schemitsch et al. [12] demonstrate the method by which

the apex of the radial bow can be found. This is foundational in the treatment of forearm fractures and the prevention of anatomic and rotational malunion.

At its distal end, the radius flares to receive articulations with the ulna at the sigmoid notch, the lunate and the scaphoid at the lunate and scaphoid fossae, and also forms the radial styloid on the radial side to receive the insertion of the brachioradialis. The radius rotates about the ulna 150–180°. This arc of rotation has a longitudinal axis that is centered in the radial head proximally and at its distal end passes through the center of the ulnar head and consequently the index finger. As the radius rotates around the ulna, the ulna moves in a varus–valgus direction about 9° at the elbow, thus allowing the ulna to move out of the way of the rotating radius distally [13].

The ulna is also a long bone. At its proximal end is the olecranon, which receives the insertion of the triceps. Anterior to this is the trochlear notch, which forms the stable articulation with the trochlea of the humerus. The trochlear notch terminates in the coronoid process. On its radial side is the radial notch or fossa, which, together with the radial head, forms the PRUJ. On the ulnar side of the trochlear notch is a small tuberosity, which receives the insertion of the brachialis. The diaphysis of the ulna is triangular in shape, with the apex pointed to the radius. This apex receives the insertions of the IOM. At its distal aspect, the ulna flares to form the ulnar head and ulnar styloid. On its radial side, the ulna articulates with the stigmoid notch of the radius to form the DRUJ. The ulnar styloid forms the origin for the triangular fibrocartilage complex (TFCC).

### 6.2.3 Muscle Anatomy

The volar forearm is comprised of fourteen muscles, whereas the dorsal forearm has thirteen involved muscles. As one moves from proximal to distal along the shaft of the radius, the following muscles are encountered: insertion of brachioradialis, origin of pronator quadratus, origin of flexor pollicis longus, origin of flexor digitorum superficialis, insertion of pronator teres, insertion of supinator, and insertion of biceps.

As one moves distal to proximal along the dorsal radius, the following muscles are encountered: insertion of brachioradialis, origin of extensor pollicis brevis, insertion of pronator teres, insertion of abductor pollicis longus, and insertion of supinator [14].

The musculature of the forearm is the driving force of forearm motion and plays vital roles in hand and wrist function. Their coordinated actions

contribute to the needed pronation, supination, flexion, and extension needed to perform the many gross and fine movements needed in the complex functions involved in daily life.

It is also these muscles that can contribute to the formation of anatomic and rotational malunions that are a result of forearm fractures. The muscles responsible for pronation are mainly the pronator teres and to a lesser extent the pronator quadratus, while the biceps brachii and supinator are supinators. It is these muscles that produce the majority of deformity in fractures of the forearm, causing the fracture ends to approach each other centrally toward IOM. Furthermore, the proximal fragments tend to be flexed, the ulna by the brachialis and the radius by the biceps brachii [9].

The brachioradialis is also a major deforming force. Its action is best demonstrated in a Galeazzi fracture, where the distal one-third of the radius is pulled into valgus, as there is no opposing force. The pronator quadratus is also involved in this fracture, wherein it pulls the distal fragment into pronation as a result of unopposed action.

### 6.2.4 Distal Radial Ulnar Joint Anatomy

Distally, the ulna articulates with the radius at the sigmoid notch to form the DRUJ. This joint is stabilized primarily by the TFCC. The palmar radioulnar ligament, dorsal radioulnar ligament, articular disk, ulnocarpal ligaments, extensor carpi ulnaris subsheath, and meniscus homolog comprise the TFCC. The ligamentous complex is the primary stabilizer of the DRUJ, whereas the fibrocartilage component transmits force across the ulnocarpal joint. The differences in curvatures in the ulnar head and sigmoid notch allow for DRUJ incongruity and thus the ability of these two structures to rotate and translate relative to one another, thereby providing a portion of the rotation necessary for forearm movements [15].

### 6.2.5 Interosseous Membrane Anatomy

The IOM and its anatomy have been studied both anatomically and biomechanically by various authors [1, 16–21]. Biomechanically, the IOM serves as an origin for forearm musculature, stabilizes the DRUJ [2–4] and the longitudinal forearm, transmits loads from the radius to the ulna, and allows for smooth forearm rotation [1, 3, 4, 8–11]. Anatomically, the IOM can be divided into distal membranous, middle ligamentous, and proximal

membranous portions [19]. Together, these structures average roughly 22 cm in length, with the radial origin being an average of 10.6 cm in length and ulnar insertion measuring 10.6 cm [20]. The width of the IOM is roughly 3.5 cm in width and 0.94 mm at its thickest point [16].

The distal membranous portion is composed of the distal oblique bundle. This portion of the IOM is found under the pronator quadratus, inserts on the inferior rim of the sigmoid notch, and blends with the DRUJ, TFCC, and dorsal and palmar ligaments at its most distal aspect [19]. Working in concert, these structures serve to stabilize the DRUJ [2–4].

The proximal oblique cord and dorsal oblique accessory cord comprise the proximal membranous portion [19]. The dorsal oblique accessory cord has also been called the proximal ascending bundle [17], or the proximal interosseous band [20]. The proximal oblique cord is found between the origin of flexor digitorum profundus and the supinator, originates from the anterolateral aspect of the coronoid process, and inserts just distal to the radial tuberosity. The dorsal oblique accessory cord is located below the origin of the abductor pollicus longus muscle [19].

The central ligamentous complex is composed of several distinct bands: the stout central band, one to five accessory bands, membranous portions, and the proximal interosseous band [19, 20]. The central ligamentous complex is divided into the central and accessory bands. The central band is the most robust of the bands of IOM and is always present; as such, it is considered to be of prime importance. Furthermore, it comprises 40–60% of the total IOM [21]. Hotchkiss et al. reported that it provided 71% of the stiffness of the IOM [16]. The central band, which is 3.5 cm in width or 2.6 cm if measured perpendicular to its fibers, originates on the radius an average of 7.7 cm distal to the articular surface of the radial head. As the central band moves distally toward its ulnar insertion at a 21 degree angle relative to the longitudinal axis of the ulna, the fibers fan out and form an insertion 4.2 cm in length on the ulna. The average insertion point of the central band is 13.7 cm distal to the tip of the olecranon [20]. The accessory bands are distinct anatomic structures, separate from the central band, and vary in number. Furthermore, they are less robust of structures [19, 20].

### 6.2.6 Proximal Radioulnar Joint Anatomy

Proximally, the radius articulates with the ulna at the PRUJ, which is composed of the radial head, the capitellum of the humerus, and the lesser

sigmoid notch of the ulna. The PRUJ is constrained and stabilized by the annular ligament, the lateral ulnar collateral ligament, the radial collateral ligament, and the surrounding elbow joint capsule and musculature. The intrinsic bony anatomy of the proximal ulna and its articulation with the distal humerus allow the ulna to be a fixed construct around which the radius can rotate.

## 6.3 Causes of Nonunion

Overall rates of nonunion of forearm fractures involving the diaphysis are less than 2% for the radius and 4–6% for the ulna [22].

With the advent of AO (Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation) principles of compression plating and implants that resist deforming forces, most diaphyseal fractures of the radius and ulna will heal in a timely manner when fixed surgically.

Nonunions that do occur can be classified into hypertrophic, oligotrophic, or atrophic nonunion s. Hypertrophic nonunion s are rare in the forearm, whereas atrophic nonunions are far more common. The blood supply to atrophic nonunions is poor, and little callous is formed adding to the challenge of healing [23].

Causes of nonunion have been enumerated, but bone loss; comminution; infection; poor soft tissue coverage or absence of soft tissue coverage; inadequate stabilization; patient comorbidities, including smoking , diabetes , malnutrition, renal dysfunction, and poor surgical technique with devascularization of the bone; and inadequate stabilization and lack of compression all contribute to nonunion [23].

Often, the question arises when one should use bone graft acutely to prevent a nonunion. We employ acute bone grafting when there is a robust soft tissue envelope and fracture characteristics are such that it does not allow the fragments to be reduced and maintained adequately. We do not have a specific bone defect size to employ acute bone grafting. If the patient has a contaminated open fracture and there is a segmental bone loss that does not allow for reduction and stabilization, one can consider using a Masquelet technique, wherein an antibiotic-impregnated cement spacer is placed in the bone defect. The surgeon can then bring the patient back to the operating room after 4–6 weeks for removal of the antibiotic cement spacer and

insertion of bone graft. The bone graft, in both instances, can be harvested from the anterior iliac crest. Another option that we have often employed if there is an open fracture and/or significant bone loss is simply plating the fracture at the appropriate length without inserting bone graft at the index procedure. The patient can then be brought back to the operating room at 4–6 weeks, bone graft can then be placed, and the fracture replated.

There is a higher incidence of nonunions of the ulna for several reasons. First, the ulna has a poorer blood supply due to a less-robust soft tissue envelope. Second, it is more common for it to be an open fracture. Third, it is fixed at both ends, resulting in increased strain and stress on the ulna as compared to the radius. However, a patient may function well with a nonunion of the ulna if that patient is able to perform their activities of daily living without incident or significant pain. If the patient is functioning well with a nonunion, we do not advocate for surgical intervention.

Maes et al. [24] reported on 133 fractures of the forearm initially treated nonoperatively. Of that total, 81 involved both the radius and ulna, 22 were isolated to the ulna, and 30 to the radius. Interestingly, the nonunion incidence was 9/81 (11%) for combined radial and ulnar shaft fractures, 6/22 (27%) for isolated ulnar shaft fractures, and 1/30 (3%) for isolated radial shaft fractures. These types of results for nonoperative treatment encouraged early authors to pursue surgical treatment to improve clinical outcomes [9–11, 25–30].

Subsequently, Anderson et al. [31] reported on 244 patients with 330 fractures of the forearm that were treated with acute open reduction and internal fixation using ASIF (Association for the Study of Internal Fixation) compression techniques with or without some other form of fixation, such as a Sage nail and acute bone grafting if more than one-third of the circumference of the shaft was comminuted. There were 112 fractures of both the radius and ulna, 82 isolated fractures of the radial shaft, and 50 isolated fractures of the ulna. 97.1% of fractures healed. There were 7 patients who developed nonunions, 4 nonunions of the radius and 5 of the ulna. This report demonstrated much improved results with regard to nonunion for compression plating of forearm fractures, in particular fractures involving both the radius and ulna.

When clinically assessing a patient with a diaphyseal forearm fracture, the surgeon must ask himself the following question: Have the bones had adequate time to heal? Four to six months are the usual period given for

forearm bony and functional healing. Other questions that should be asked: Is the fracture unstable? Is there adequate soft tissue coverage? Were there multiple injuries? Has the patient had continual pain? Has there been any progression of healing from month one to month six?

In considering the radiograph, the surgeon must ask certain questions:

1. Are there gaps on the X-ray?

2. Has there been any softening of the fracture surfaces (a sign of healing)?

3. Is there any hardware loosening?

4. Is there any hardware in the fracture site?

5. Is there any sign of infection?

6. Is the fracture site atrophic or hypertrophic?

---

## 6.4 Surgical Treatment

### 6.4.1 Preoperative Planning

After determination that a nonunion exists, the surgical alternatives are discussed with the patient and the surgery is planned.

Preoperative planning includes, if needed: perfect orthogonal *anteroposterior* and lateral radiographs of both forearms. Computed tomography is also a valuable option to help the surgeon in determining the surgical approach, as well as in evaluating the nonunion.

If the patient was surgically treated at another institution, operative reports and other pertinent records should be sought and obtained.

If the possibility of infection exists, complete blood count, erythrocyte sedimentation rate, and C-reactive protein tests should be ordered to help determine the need for more advanced imaging studies.

Thought should be given to the implants that will be used. For example, will dynamic compression plate (DCP), limited-contact dynamic compression

plate (LC-DCP), and/or locking plates be applied? If infection is a concern, one should also consider the need for external fixation, cement spacers, and the possibility of staging the surgical procedures.

If the nonunion is the result of a prior surgical failure, instruments for screw removal should be on hand, as well as possible power tools for cutting titanium and stainless steel.

Thought should also be given to the need for bone graft. If autograft is chosen, the planned harvest site should be prepped. We recommend not to take bone graft from proximal ulna/distal radius because it creates a stress riser, and thus, the likelihood of iatrogenic fracture increases. If bone graft is needed, we either use anterior iliac crest if cortical is desired, or if cancellous bone is needed, a significant amount can be obtained from the proximal tibia by making a bone window at Gerdy's tubercle and then curetting the cancellous bone. Care must be taken to not penetrate the knee joint. Bone graft substitutes should be on hand if autograft is not chosen. If there is a possibility of infection, the fracture site should be exposed first before graft taken.

Appropriate medical evaluation, surgical risk stratification, and laboratory work should be done, and blood products, if needed, should be available.

## 6.4.2 Operating Room Setup

An adequate sized room should be obtained to allow for an arm table, C-arm, and back tables for bone graft. If an arm table is used, the C-arm is positioned to enter from the foot to obtain adequate views. The monitor is placed at the location most convenient and freely viewed by the surgeon.

The arm should be draped out at the shoulder and a sterile tourniquet applied. The draping of the patient should also allow the forearm to be positioned across the chest. If needed, access to the opposite side of the table allows the arm to be positioned over the chest.

For the procedure, the surgeon should arrange for the presence of capable assistants and the engagement of an anesthesiologist.

With regard to instruments that should be available, bone tools, rongeurs, osteotomes, burrs, curettes, screw removal and broken screw removal sets, and saws and/or burrs capable of cutting stainless steel and titanium should all be readily accessible in the operating room suite.

There should also be a discussion with the patient about the type of anesthesia to be used. Will regional or general anesthesia be administered? If

no autogenous iliac graft is needed, regional anesthesia is preferred.

In addition, the surgeon should discuss postoperative pain, bleeding, swelling, and compartment syndrome with patient and the appropriate treatment and response to these postoperative conditions.

### 6.4.3 Surgical Approach and Exposure

If there is concern about the previous skin incision(s), a fresh incision can be made through virgin tissue. Potential reasons for doing this would be that you did not perform the index procedure, and based on the skin incision, there is potential for damage to important structures. Otherwise, the previous incision and/or incisions can be utilized.

For the ulna diaphysis, the approach should be just dorsal to the subcutaneous border and over the muscle and not the bone. Choose the interval between the flexor carpi ulnaris and extensor carpi ulnaris. This is adequate for exposure of all the ulnar diaphysis.

For the radius, the distal two-thirds of the shaft can be approached through a volar-Henry approach.

1. The skin incision should follow a line drawn from the radial styloid distally to the biceps tendon proximally.

2. Sharply incise the skin and subcutaneous tissue to the forearm fascia.

3. Incise the fascia over the flexor carpi radialis tendon down to the tendon itself.

4. Use a moist lap sponge to dissect the subcutaneous tissue off the fascia, exposing the interval between the flexor carpi radialis and the radial artery.

5. The artery does not need to be dissected out completely unless the incision approaches the mid-forearm.

6. If the fracture requires exposure proximal to the mid-forearm, the radial artery needs to be dissected so that it can be mobilized and retracted

either radially or ulnarly.

7. The deep interval between the flexor pollicis longus and the brachioradialis is developed proximally and distally.

8. The pronator quadratus is dissected and subperiosteally cleared off the distal radius, from its radial styloid attachment.

9. If proximal exposure is needed, the pronator teres is sharply incised and its tendinous insertion dissected off the bone.

10. Volar exposure can be extended proximal to the bicipital tuberosity by ligating the radial recurrent vessels and subperiosteally dissecting the supinator off the radius, with protection of the posterior interosseous nerve.

11. The volar approach to the radius allows exposure of the entire diaphysis. One must be aware of and protect the radial artery, the superficial sensory branch of the radial nerve, and the posterior interosseous nerve proximally, as well as the brachial artery and median nerve.

The posterior Thompson approach can be used for those nonunions that require exposure of the entire route of the posterior interosseous nerve.

1. The skin incision is made along a line with the forearm pronated, starting at the lateral epicondyle of the elbow and ending over Lister's tubercle.

2. The skin is incised, and a moist lap sponge is used to dissect the subcutaneous tissue off the fascia.

3. The interval between the extensor digitorum comminus and the extensor carpi radialis brevis is more easily found distally.

4. In large individuals, you can use the bovie to stimulate the muscle bellies

proximally and easily separate the extensor digitorum comminus from the extensor carpi radialis brevis.

5. The dissection through the muscle bellies is more easily done from distal to proximal.

6. The glistening fascia over the supinator is easily identified, and the distal border of the muscle is the anatomical point where the posterior interosseous nerve arborizes.

7. Prior to branching, the posterior interosseous nerve lies between the two muscle layers of the supinator accompanied by its artery and vein. It can be easily found and freed up to the radial head.

8. The supinator then can be easily elevated off the proximal radius.

Pitfalls occur when the proper interval is not recognized, and denervation of the extensor digitorum comminus can occur. Vigorous retraction of the posterior interosseous nerve can result in a posterior interosseous nerve palsy.

## 6.4.4 Essentials of Exposure

1. Adequate draping to allow full exposure of the limb

2. Appropriate functioning tourniquet and equipment

3. Functioning C-arm and easily available screens

4. Comfortable seating and height for the surgeon and assistant along with loupe magnification

5. Draw incisions with a marker.

6. If two incisions are needed, allow at least a six to eight centimeter interval between the incisions.

7. The secret to soft tissue dissection is adequate tension on the tissues in the correct vector.

8. Dissect from normal to abnormal tissue. Never seek to identify structures in scar tissue.

9. Dissect with the tips of your scissors.

10. Scissors work best in normal tissue, and a scalpel is needed in scar tissue.

11. Hemostasis can be obtained with clips, sutures, or the bovie. To avoid any intimal damage to the artery, bovie at least one centimeter away from the artery.

12. Most exposures in normal tissue can be done by dividing fascia and mesentery, avoiding proximity to major nerves and vessels.

13. Place retractors appropriately and remember “the bone and periosteum are your friends.”

14. Keep tissues moist.

15. If, after adequate exposure, there is concern about bleeding, let your tourniquet down to control it. It is often easier to ligate vessels that may be difficult to reach if the fracture is not stabilized with a plate. After hemostasis is obtained, you can reinflate the tourniquet and place the fixation.

## 6.4.5 Bone Preparation

The surgeon should avoid extensive subperiostal stripping. The periosteal elevator should be used against the acute angle of the muscle attachments. Once the fracture site is exposed and the plate(s), if any, removed, use bone

hooks to bring the bone to you. Meticulous attention should be placed upon preserving the soft tissues and blood supply.

Technique for bone preparation:

1. Curette the screw holes.

2. Using a rongeur, debride the bone ends back to bleeding bone at fracture site.

3. Reconstitute the medulary canal in both fragments to allow ingress of pluripotent cells.

4. Use an osteotome to “rose petal” the cortical bone for one inch on both sides of nonunion site [32].

5. Select a plate that has at least six cortices in nonviolated bone on both sides of the nonunion site. Do not use previous drill holes.

6. Use the plate to reestablish length, and if a gap exists at the nonunion site, it should be filled with cancellous or corticocancellous graft. It is preferable to use autologous graft and insert it immediately after harvesting it. Do not let it sit on the back table. Defects up to four to six centimeters should be treated with corticocancellous grafts that give structural support. Defects larger than six centimeters should be considered for vascularized grafts [33–38].

7. Perform a routine closure without drains and apply a bulky sterile dressing with a sugartong splint.

8. Depending upon the stability of the fixation, active range of motion can be initiated at 10 days with a removable orthosis.

## 6.4.6 Technical Points

1. Preserve soft tissues as much as possible.

2. Begin dissection in normal tissue

3. Keep tissues moist.

4. Achieve meticulous hemostasis.

5. The tourniquet should be released at 60 min and possibly reinflated if needed.

6. Antibiotics should be administered preoperatively and postoperatively for 24 h.

7. Leave sutures in place for two weeks.

8. Obtain radiographs at two months unless otherwise indicated.

9. The expected healing time is 6–12 months.

---

## 6.5 Patient Variables

There are many factors that are patient-dependent and they include:

1. Smoking

2. Adequate control of comorbidities: diabetes, hypertension, cardiac disease, liver disease, chronic obstructive pulmonary disease, and alcoholism.

3. Preoperative assessment of nutritional status should be included in the postoperative course.

4. Insomnia, depression, anxiety, bipolar, and fibromyalgia all have some influence on the fracture healing.

## 6.6 Anticipated Outcomes/Recurrent Nonunions

Successful healing in uncomplicated nonunions treated appropriately should be within the 85–100% range [23, 34–39]. Failure of a nonunion following appropriate surgical intervention is usually related to infection, hardware failure, smoking, and chronic illness. If the nonunion is treated appropriately, these nonunions are beyond the treating surgeon's control. Bone stimulators can be of some benefit, but our experience with them is limited.

These recurrent nonunions must be evaluated as to the cause and can be treated with vascularized grafts, possible plate replacement, and autologous grafts. Difficult nonunions, infected nonunions, and nonunions with missing bone can be treated with one-bone forearm.

## 6.7 Case Discussions

**Case 1** (Fig. 6.1)

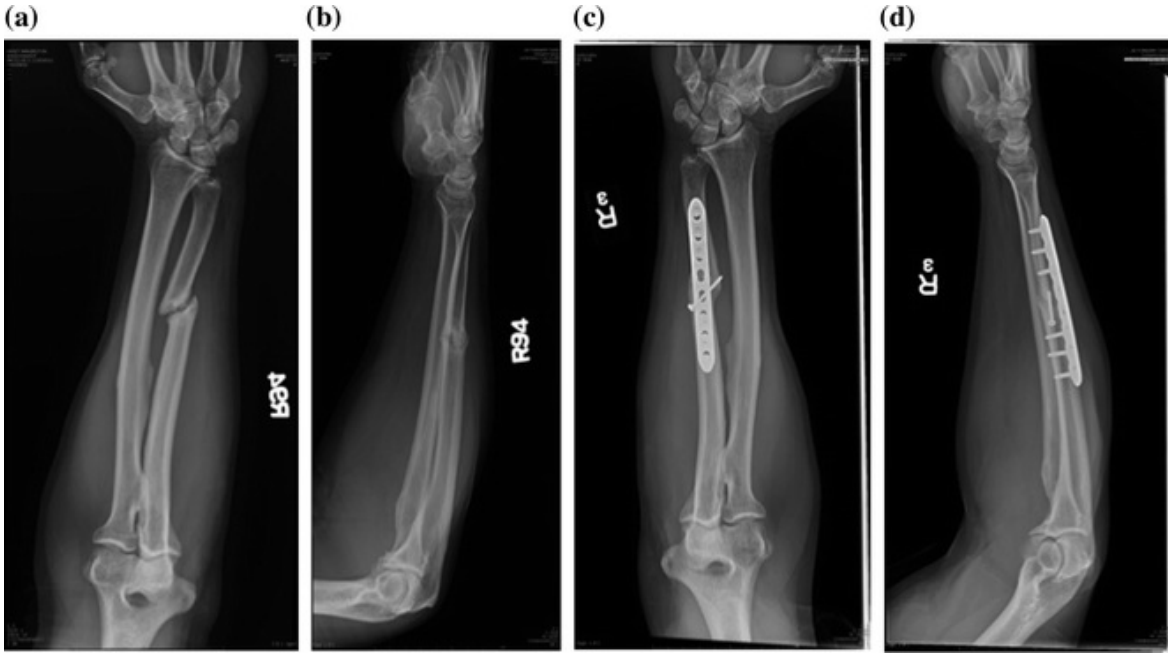



***Fig. 6.1*** **a**, **b** *Anteroposterior* (AP) and lateral of nonunion after failed closed treatment. **c**, **d** AP

and lateral of after repair of nonunion

A 45-year-old, right-hand dominant, day laborer, who was a 2-pack-per-day smoker, sustained a direct blow to his ulnar forearm, resulting in a fracture of the shaft of the ulna. It was decided, in conjunction with the patient, to treat his fracture with nonoperative methods. He subsequently went on to develop an atrophic nonunion. Once nonoperative treatment was exhausted, the patient underwent nonunion takedown, rose-petaling of both fracture ends, reestablishing of the canals, application of a corticocancellous autograft, open reduction and internal fixation with an interfragmentary compression screw, and then the fracture was neutralized with a 3.5 mm locking compression plate The patient stopped smoking as well and went on to heal his nonunion uneventfully.

**Case 2** (Fig. 6.2)


***Fig. 6.2*** **a**, **b** *Anteroposterior* (AP) and lateral after gunshot wound. **c**, **d** AP and lateral with established "asymptomatic" nonunion

A 37-year-old, right-hand dominant male sustained a gunshot wound to his ulna. He refused surgical intervention and was treated with closed methods. At his last follow-up, he had 55° of supination and 35° of pronation. He continued to refuse surgical interventions, stating that he had adequate function and eventually was lost to follow-up.

**Case 3** (Fig. 6.3)


***Fig. 6.3*** **a**, **b** *Anteroposterior* (AP) and lateral of delayed presentation of right proximal radius fracture. **c**, **d** AP and lateral at 1 year with healed proximal radius fracture despite a delayed presentation

A 63-year-old, left-hand dominant male had end-stage renal disease as a result of poorly controlled diabetes mellitus. He sustained multiple orthopedic and general surgery injuries, including a proximal radius fracture that was identified 4 months after his initial accident. The patient was treated by closed methods for medical reasons and also because the patient refused surgical intervention. He was definitively managed in an orthosis which

eventually healed at 12 months after his accident. He was able to obtain 15° of pronation and 40° of supination and had function appropriate for his daily needs.

**Case 4** (Fig. 6.4)


***Fig. 6.4*** **a**, **b** *Anteroposterior* (AP) and lateral after initial plating. Length was re-established. **c**, **d** AP and lateral with nonunion of radius and failure of hardware. **e**, **f** AP and lateral after repeat osteosynthesis and iliac crest bone graft. **g**, **h** AP and lateral after healing of nonunion

A 26-year-old, right-hand dominant male sustained a handgun gunshot wound to the forearm, resulting in a 3-cm bone defect of the radius. Initially, this was plated out to length and not grafted. The fixation ultimately failed,

and a nonunion ensued. He also had a significant decline in his forearm rotation, resulting in 25° of supination and 35° of pronation. The patient was taken back to the operating room, a corticocancellous graft was harvested from the patient's ipsilateral iliac crest, and a repeat osteosynthesis was performed. The patient went on to heal, and his supination improved to 55° and pronation to 50°.

**Case 5** (Fig. 6.5)


(a)
(b)
49
R
PORTABLE
(c)
(d)
RT52
Portable
R 32
Portable
OK'D BY
DR. CROMACK
(e)
(f)

***Fig. 6.5*** **a**, **b** *Anteroposterior* (AP) and lateral of initial gunshot wound to right radius. **c**, **d** Immediate post-op AP and lateral of revision open reduction internal fixation with vascularized fibular strut and plate. **e, f** 11-month post-op AP and lateral showing complete healing of nonunion with complete incorporation of vascularized fibula

A 34-year-old, left-hand dominant male sustained a gunshot wound to the radius with a resulting 4-cm bone defect. The patient was treated initially with irrigation and debridement of devitalized bone and soft tissue, application of an antibiotic-impregnated cement spacer, and plate fixation. The patient was lost to follow-up and returned a year later. The construct had ultimately failed and the patient had decreased function with 45° of supination and 55° of pronation. He desired to have repeated surgical treatment and returned to the operating room for a joint intervention with plastic surgery. A vascularized fibula was harvested and then insetted. He subsequently went on to heal successfully with the vascularized fibular graft. His supination improved to 60°, and pronation stayed the same at 55°.

**Case 6** (Fig. 6.6)

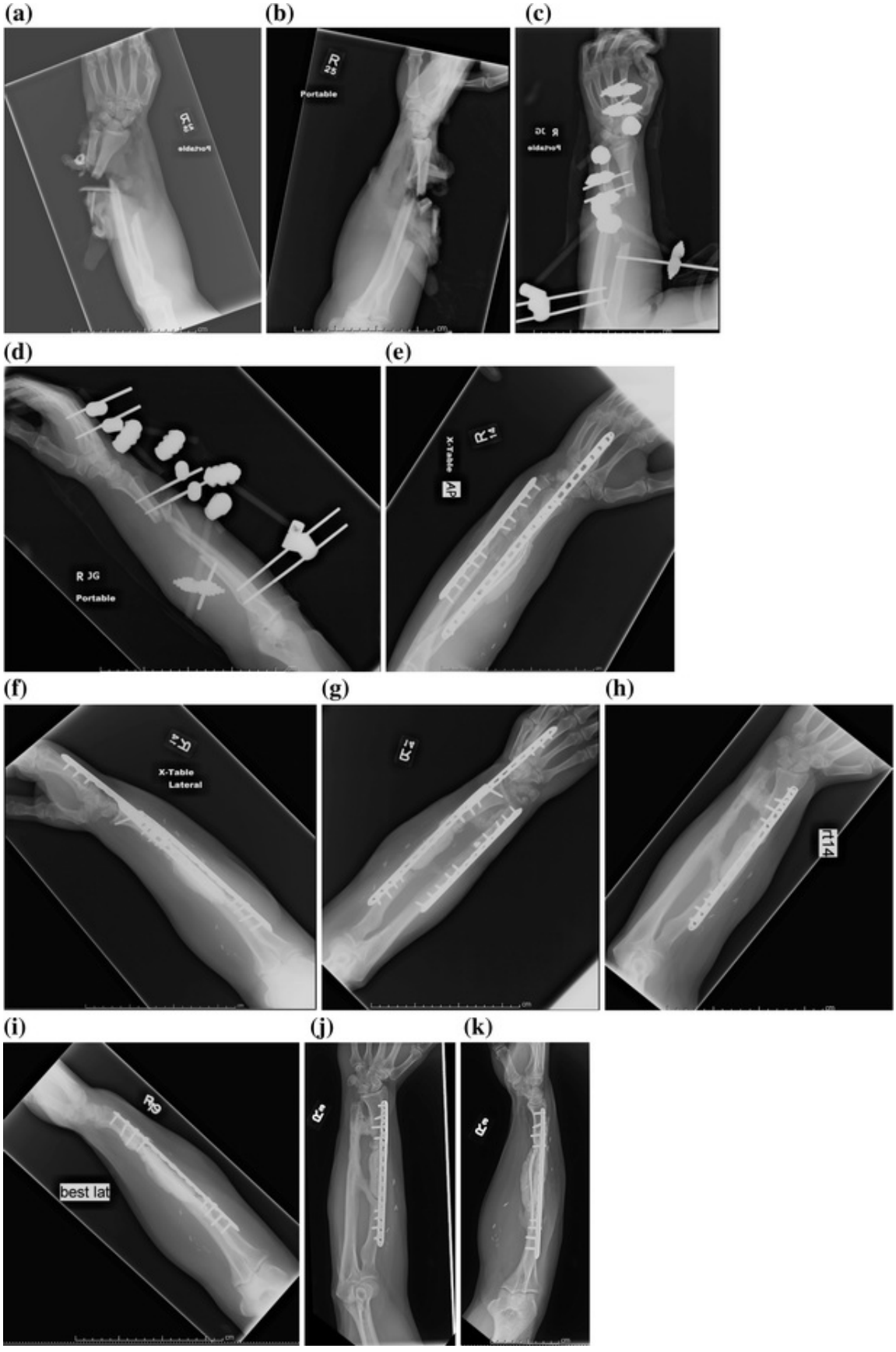

(a)
(b)
(c)
Portable
(d)
(e)
Portable
X-Table
AP
(f)
(g)
(h)
X-Table
Lateral
(i)
(j)
(k)
best lat

***Fig. 6.6*** **a, b** *Anteroposterior* (AP) and lateral at initial injury showing obvious soft tissue defect and significant bony injury. **c**, **d** AP and lateral showing temporizing external fixation and the significant bone loss from the injury and after debridement. **e**, **f** AP and lateral after "bridging" internal fixation of wrist and open reduction internal fixation of radius and ulna. Cement spacer placed in radial defect. **g** Follow-up radiograph showing development of radioulnar synostosis. (**h**, **i**) AP and lateral of subsequent removal of hardware from ulna and Darrach procedure. **j**, **k** Final follow-up AP and lateral with healed ulna, stable radius with retained cement and establishment of radioulnar synostosis

A 39-year-old, right-hand dominant sustained multiple traumatic injuries, including lower extremity amputations and open fractures of his right radius and ulna and concomitant massive soft tissue injury to the right forearm. The patient's forearm fractures were initially treated with a temporary external fixator. Once the patient was stable medically, he returned to the operating room, where the external fixator was removed, the radius was plated out to length, and an antibiotic-impregnated cement spacer was inserted. The definitive plan was to take the patient back to the operating room to remove the cement spacer and insert a corticocancellous graft; however, a radioulnar synostosis began to form. The bridging wrist plate was removed. The patient continued with symptomatic ulna hardware and ulnar-sided wrist pain, necessitating hardware removal from the ulna and a Darrach procedure. At that point, an intra-operative fluoroscopic stress test was performed, which confirmed that the radioulnar synostosis had matured and the decision was made to leave the cement spacer in place as the patient had essentially developed a one-bone forearm.

**Case 7** (Fig. 6.7)

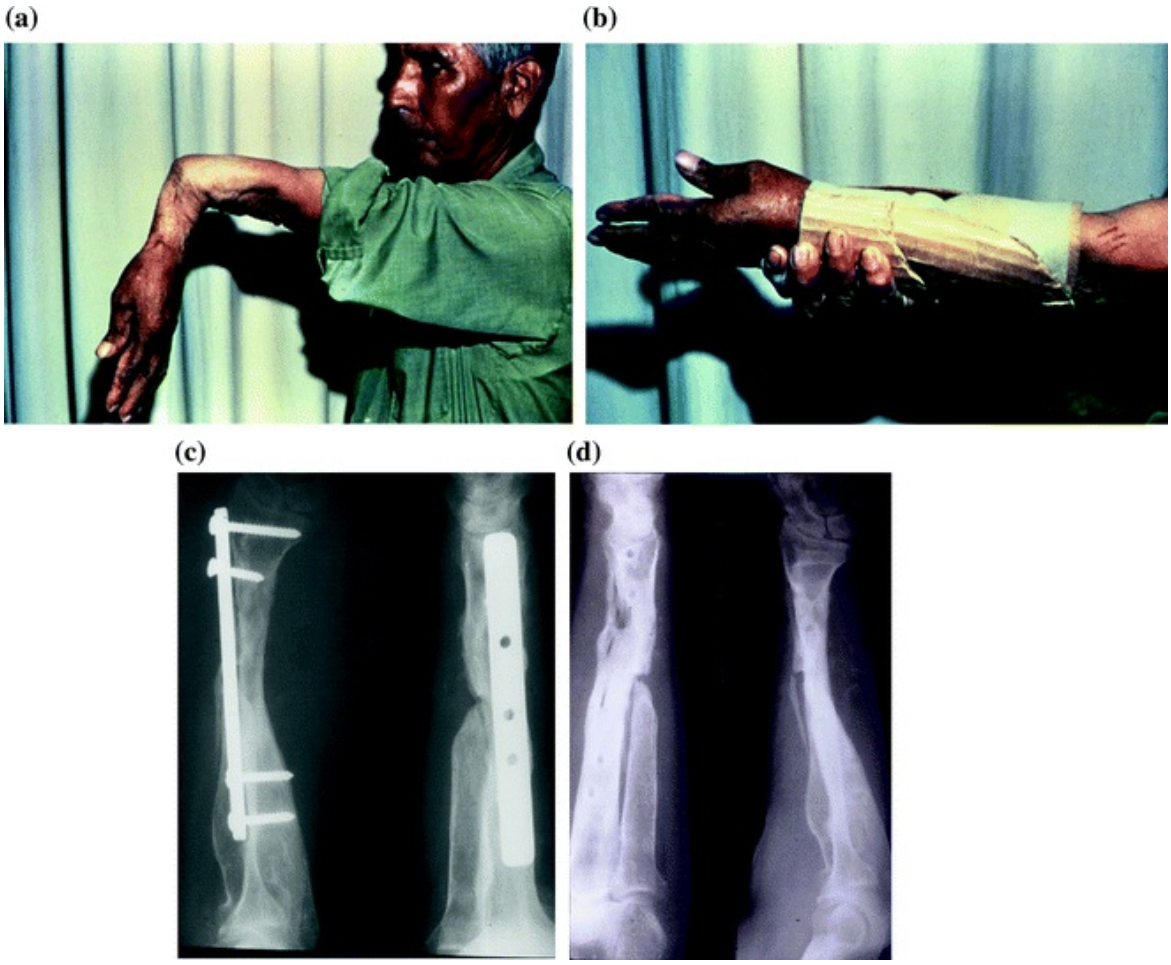



***Fig. 6.7*** **a** Clinical picture of *left* forearm-infected nonunion. **b** Clinical picture with splint application showing correction of deformity. **c** Creation of a healed one-bone forearm with proximal ulna plated to distal radial shaft. **d** Healed one-bone forearm after plate removal secondary to hardware irritation

A 43-year-old, left-hand dominant male day laborer sustained open fractures of his left radius and ulna in a work-related accident. This was treated nonoperatively and went on to become an infected nonunion. His treating physicians at that time continued to treat him nonoperatively, and the patient was able to work with his homemade splint. He subsequently presented to our institution for evaluation. He was taken to the operating room for evaluation and operative treatment. His nonunion site was taken down, and the distal radius was inserted into the proximal ulna, and then, the fracture was plated. In addition, he was treated with appropriate antibiotic therapy and his fracture subsequently healed. He returned 2 years later with complaints of hardware irritation, and the plate was then removed (see Fig. 6.7d).

# References


1. McGinley JC, Kozin SH. Interosseous membrane anatomy and functional mechanics. Clin Orthop. 2001;383:108–22.
[Crossref]

2. Ward LD, Ambrose CG, Masson MV, Levaro F. The role of the distal radioulnar ligaments, interosseous membrane, and joint capsule in distal radioulnar joint stability. J Hand Surg Am. 2000;25(2):341–51.
[Crossref][PubMed]

3. Watanabe H, Berger RA, Berglund LJ, Zobitz ME, An KN. Contribution of the interosseous membrane to distal radioulnar joint constraint. J Hand Surg Am. 2005;30(6):1164–71.
[Crossref][PubMed]

4. Kihara H, Short WH, Werner FW, Fortino MD, Palmer AK. The stabilizing mechanism of the distal radioulnar joint during pronation and supination. J Hand Surg Am. 1995;20(6):930–6.
[Crossref][PubMed]

5. Soubeyrand M, Wassermann V, Hirsch C, Oberlin C, Gagey O, Dumontier C. The middle radioulnar joint and triarticular forearm complex. J Hand Surg Eur. 2011;36(6):447–54.
[Crossref]

6. Morrey BF, Askew LJ, Chao EY. A biomechanical study of normal functional elbow motion. J Bone Joint Surg Am. 1981;63(6):872–7.
[Crossref][PubMed]

7. Tarr RR, Garfinkel AI, Sarmiento A. The effects of angular and rotational deformities of both bones of the forearm. An in vitro study. J Bone Joint Surg Am. 1984;66(1):65–70.
[Crossref][PubMed]

8. Youm Y, Dryer RF, Thambyrajah K, Flatt AE, Sprague BL. Biomechanical analyses of forearm pronation-supination and elbow flexion-extension. J Biomech. 1979;12(4):245–55.
[Crossref][PubMed]

9. Jinkins WJ Jr, Lockhart LD, Eggers GW. Fractures of the forearm in adults. South Med J. 1960;53:669–79.
[Crossref][PubMed]

10. Burwell HN, Charnley AD. Treatment of forearm fractures in adults with particular reference to plate fixation. J Bone Joint Surg Br. 1964;46:404–25.
[PubMed]

11. Sage FP, Smith H. Medullary fixation of forearm fractures. J Bone Joint Surg Am. 1957;39-A(1):91–8.

12. Schemitsch EH, Richards RR. The effect of malunion on functional outcome after plate fixation of fractures of both bones of the forearm in adults. J Bone Joint Surg Am. 1992;74(7):1068–78.
[Crossref][PubMed]

13. Boland, MR. Open reduction and internal fixation of diaphyseal forearm fractures. In: Wiesel, SW, editor. Operative techniques in orthopaedic surgery. 1st ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2011. p. 2127–9.

14. Llusa Perez M, Meri Vived A, Ruano Gil D. Surgical atlas of the musculoskelatal system. 1st ed. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2008. p. 61–5.

15. Huang JI, Hanel DP. Anatomy and biomechanics of the distal radioulnar joint. Hand Clin. 2012;28(2):157–63.
[Crossref][PubMed]

16. Hotchkiss RN, An KN, Sowa DT, Basta S, Weiland AJ. An anatomic and mechanical study of the interosseous membrane of the forearm: pathomechanics of proximal migration of the radius. J Hand Surg Am. 1989;14(2 Pt 1):256–61.
[Crossref][PubMed]

17. Poitevin LA. Anatomy and biomechanics of the interosseous membrane: its importance in the longitudinal stability of the forearm. Hand Clin. 2001;17(1):97–110, vii.

18. Nakamura T, Yabe Y, Horiuchi Y. Functional anatomy of the interosseous membrane of the forearm—dynamic changes during rotation. Hand Surg. 1999;4(1):67–73.
[Crossref][PubMed]

19. Noda K, Goto A, Murase T, Sugamoto K, Yoshikawa H, Moritomo H. Interosseous membrane of the forearm: an anatomical study of ligament attachment locations. J Hand Surg Am. 2009;34(3):415–22.
[Crossref][PubMed]

20. Skahen JR 3rd, Palmer AK, Werner FW, Fortino MD. The interosseous membrane of the forearm: anatomy and function. J Hand Surg Am. 1997;22(6):981–5.
[Crossref][PubMed]

21. Schneiderman G, Meldrum RD, Bloebaum RD, Tarr R, Sarmiento A. The interosseous membrane of the forearm: structure and its role in Galeazzi fractures. J Trauma. 1993;35(6):879–85.
[Crossref][PubMed]

22. Chapman MW, Gordon JE, Zissimos AG. Compression-plate fixation of acute fractures of the diaphyses of the radius and ulna. J Bone Joint Surg Am. 1989;71(2):159–69.
[Crossref][PubMed]

23. Stewart RL. Operative treatment of radius and ulna diaphyseal nonunions. In: Wiesel SW, editor. Operative techniques in orthopaedic surgery. 1st ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011. p. 2156–61.

24. Maes JM, Van Velthoven V. Clinical and statistical research on the occurrence of pseudarthrosis of ulna and radius in forearm fractures. Acta Orthop Belg. 1977;43(6):767–80.
[PubMed]

25. Knight RA, Purvis GD. Fractures of both bones of the forearm in adults. J Bone Joint Surg Am. 1949;31A(4):755–64.
[Crossref][PubMed]

26. Sage FP. Medullary fixation of fractures of the forearm. A study of the medullary canal of the radius and a report of fifty fractures of the radius treated with a prebent triangular nail. J Bone Joint Surg Am. 1959;41-A:1489–516.

27. Hughston JC. Fracture of the distal radial shaft; mistakes in management. J Bone Joint Surg Am. 1957;39-A(2):249–264; passim.

28. Caden JG. Internal fixation of fractures of the forearm. J Bone Joint Surg Am. 1961;43(8):1115–21.
[Crossref]

29. Hicks JH. Fractures of the forearm treated by rigid fixation. J Bone Joint Surg Br. 1961;43-B:680–7.

30. Sargent JP, Teipner WA. Treatment of forearm shaft fractures by double-plating; a preliminary report. J Bone Joint Surg Am. 1965;47(8):1475–90.
[Crossref][PubMed]

31. Anderson LD, Sisk D, Tooms RE, Park WI 3rd. Compression-plate fixation in acute diaphyseal fractures of the radius and ulna. J Bone Joint Surg Am. 1975;57(3):287–97.
[Crossref][PubMed]

32. Richard MJ, Ruch DS, Aldridge JM 3rd. Malunions and nonunions of the forearm. Hand Clin. 2007;23(2):235–243, vii.

33. Shelton WR, Sage FP. Modified Nicoll-graft treatment of gap non-unions in the upper extremity. J Bone Joint Surg Am. 1981;63(2):226–31.
[Crossref][PubMed]

34. Moroni A, Rollo G, Guzzardella M, Zinghi G. Surgical treatment of isolated forearm non-union with segmental bone loss. Injury. 1997;28(8):497–504.
[Crossref][PubMed]

35. Ring D, Allende C, Jafarnia K, Allende BT, Jupiter JB. Ununited diaphyseal forearm fractures with segmental defects: plate fixation and autogenous cancellous bone-grafting. J Bone Joint Surg Am. 2004;86-A(11):2440–5.

36. Wood MB. Upper extremity reconstruction by vascularized bone transfers: results and complications. J Hand Surg Am. 1987;12(3):422–7.
[Crossref][PubMed]

37. Jupiter JB, Gerhard HJ, Guerrero J, Nunley JA, Levin LS. Treatment of segmental defects of the radius with use of the vascularized osteoseptocutaneous fibular autogenous graft. J Bone Joint Surg Am. 1997;79(4):542–50.
[Crossref][PubMed]

38. Dell PC, Sheppard JE. Vascularized bone grafts in the treatment of infected forearm nonunions. J Hand Surg Am. 1984;9(5):653–8.
[Crossref][PubMed]

39. Davey PA, Simonis RB. Modification of the Nicoll bone-grafting technique for nonunion of the

radius and/or ulna. J Bone Joint Surg Br. 2002;84(1):30–3.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_7

# 7. Nonunions of the Wrist and Hand

Matthew Lyons[1], Ahmad Fashandi[1] and Aaron M. Freilich[1]

(1) Department of Orthopedics, University of Virginia, P.O. Box 800159, Charlottesville, VA 22908-0159, USA

**Aaron M. Freilich**
**Email:** amf7z@virginia.edu

**Keywords** Distal radius – Distal radioulnar joint (DRUJ) – Scaphoid – Scaphoid nonunion advanced collapse (SNAC) – Scaphocapitate – Hamate – Metacarpal – Phalange

## 7.1 Introduction

Nonunions involving the hand and wrist can occur after injury and result in continued pain, weakness, and delay in recovery of function. If unrecognized and undertreated, they can be a source of frustration for both the patient and the treating clinician. Injury to any of the bones in the hand and wrist can be result in nonunion; however, some, such as scaphoid and hamate, as a consequence of their unique vascularity, are more likely to be involved than others. Depending on which bone is involved, different strategies for treatment need to be adopted. In all cases, the underlying cause of the nonunion should be assessed. This can be a problem with soft tissue coverage, vascularity, motion at the fracture, infection, or metabolic causes. In the carpal bones of the wrist, osteonecrosis becomes an important complicating factor that needs to be addressed. In many patients, obtaining healing of the fracture will be the goal; however, salvage procedures such as

arthrodesis or even amputation should be considered depending on circumstances.

---

## 7.2 Distal Radius Fracture Nonunion

### 7.2.1 Background

Distal radius fractures are the most common injury of the upper extremity. In the literature, management of these fractures has commonly focused on restoring key parameters of skeletal alignment, rather than obtaining consolidation of the fracture itself [1–3]. This is due to the fact that treatment of these injuries rarely results in fracture nonunion. The low incidence of nonunion is often attributed to the excellent intraosseous blood supply of the distal radius, healing potential of cancellous metaphyseal bone, impaction of fracture fragments, and minimal soft tissue disruption caused by the typical mechanism of a low-energy fall [1]. Nonunion is more common in the setting of attempted surgical fixation than closed management and appears to be increasing over the past two decades [1, 2, 4]. A potential causative factor for this increase is the advent of fixed angle volar plating, which has allowed surgeons to restore and maintain radial length in the setting of high-energy injuries with extensive comminution, resulting in gap creation in the metaphyseal bone [1, 5]. Other risk factors include unstable fracture patterns with inadequate fixation, concomitant fracture of the distal ulna, open comminuted fractures, infections, pathologic lesions, and inadequate period of immobilization [1, 2, 4]. Whatever the cause, nonunions of the distal radius often result in significant pain, stiffness, and dysfunction, which necessitate surgical stabilization. Treatment of these injuries is aimed at obtaining bony stability and restoring alignment and can be difficult due to small, osteopenic bone fragments, bony reabsorption at the fracture site, and associated soft tissue contracture [1].

### 7.2.2 Anatomy

The failure to recognize key fracture components that predict fracture instability is a risk factor for nonunion with both conservative and operative management of these injuries. Therefore, an understanding of the anatomic alignment and relationships between the distal radius and radiocarpal articulation is paramount to the treatment of distal radius fractures. The distal

radius forms an articular platform on which the carpus rests. The stability provided by the radiocarpal articulation and its surrounding ligaments allows the complex functions performed by the carpus and hand. The distal radius has three concave surfaces, which form the foundations of this articulation, the scaphoid fossa, lunate fossa, and sigmoid notch. The scaphoid and lunate fossas are divided by a sagittal plane ridge. The radiocarpal articulation is further stabilized by the strong volar radial ligamentous structures, including the radioscaphocapitate (RSC), radiolunotriquetral (RLT), radioscapholunate (RSL), and dorsal radiotriquetral (RT) ligaments. The sigmoid notch acts an articulation for the distal ulna, allowing forearm motion through rotation of the radius around the ulna. It has well-defined dorsal, volar, and distal walls, with further stability provided by the components of the triangular fibrocartilage complex (TFCC), including deep and superficial volar and dorsal radioulnar ligaments [5].

Key radiographic parameters predict stability and dictate treatment of the distal radius. These include radial height, radial inclination, radial tilt, ulnar variance, articular congruity, and distal radioulnar joint (DRUJ) stability. Radial height is measured on the posterioranterior (PA) radiograph by drawing two lines tangential to the radial styloid and ulnar head articular surface and perpendicular to their shaft axes. The distance between the lines is measured. The average normal value is 11 mm with an acceptable limit of 4 mm. Radial inclination, also measured on a PA radiograph, is the angle formed by one line perpendicular to the longitudinal axis of the radial shaft and a second line along the distal radius articular surface. The normal value for this angle is 22° with an acceptable change of 15° in either direction. Measured on the lateral radiograph, radial tilt is the angle between the distal radius articular surface and a line perpendicular to the longitudinal axis of the radial shaft. It has a normal value of 11° of volar tilt with an acceptable limit of 15° of dorsal or 20° of volar tilt. Ulnar variance is calculated on the PA radiograph by the axial difference in length from lines drawn parallel to the ulnar head articular surface and ulnar most edge of articular distal radius. It averages neutral to 1 mm of ulnar negative variance with an acceptable limit of ±4 mm [6, 7]. Articular incongruity is determined by breaks in the dense subchondral bone on the PA radiograph, less than 2 mm of articular gap or step-off is acceptable. If these parameters are met and able to be maintained by either closed or open means, the outcome of treatment will be determined by the degree of soft tissue injury rather than skeletal deformity.

### 7.2.3 Classification Systems

Multiple classification systems have been proposed to describe distal radius injuries patterns, each with its own inherent strengths and weaknesses. The goal of any surgeon taking care of these injuries is to become familiar enough with common injury patterns as to recognize the personality of the individual fracture [3]. This is a combination of the energy, deformity, comminution, soft tissue disruption, and stability inherent in the fracture pattern that are predictive of outcome and help determine treatment.

Given that the majority of distal radius fracture nonunions involve high-energy injuries and intra-articular fracture patterns, we find the columnar classification proposed by Rikli and Regazzoni and the fragment-specific system of Medoff most useful in approaching these injuries [8–10]. Rikli and Regazzoni's classification system divides the wrist into three columns. The radial column is composed of the radial styloid and scaphoid facet of the distal radius. Realignment of this column effectively restores radial height and inclination. The intermediate column contains the primary load bearing portion of the distal radius and is encompassed by the lunate facet of the distal radius. Fractures of this column may also disrupt the sigmoid notch and distal radioulnar articulation. Finally, the medial column is composed of the distal ulna, TFCC, and radioulnar ligaments. Injuries to the medial column may result in DRUJ instability. The integrity and stability of the DRUJ should be assessed in the setting of every distal radius fracture, and the treatment of DRUJ instability will be covered later in this chapter.

The fragment-specific classification system proposed by Medoff is a direct extension of the Melone Classification. Originally proposed in 1984, Melone's system divides the distal radius into four components: the shaft, radial styloid, and volar medial and dorsal medial facets [11]. Medoff expanded this system to include five major fracture fragments, including the radial styloid, dorsal wall, impacted articular segment, dorsal ulnar corner, and volar rim [9, 10]. The system provides a treatment algorithm for fragment-specific fixation that can be useful with obtaining stability in the setting of a nonunion.

### 7.2.4 Clinical Evaluation

As with any initial assessment, clinical evaluation should begin with a detailed history of the patient's injury mechanism and any attempted

treatment. Any previous attempt at surgical treatment should be elicited as it may play a role in preoperative planning. Patients frequently present with a combination of pain and dysfunction, related to deformity and instability. It should be noted that a number of patients in the latent nonunion period between 3 and 6 months after injury may complain of only stiffness, as they have experienced insignificant time or activity level to develop other symptoms. Patients should be questioned about potential risk factors for nonunion, including advanced age, medical comorbidities, smoking , nonsteroidal anti-inflammatory use, metabolic disease, and nutritional deficiency. A complete medical history should be elicited, with particular attention given to the patient's occupational demands, recreational activities, and goals for treatment. The preoperative evaluation of nonunion should include a characterization of biologic healing capacity, deformity, presence or absence of infection, and host status. Correction of modifiable risk factors, such as tobacco cessation and nutritional status, should be attempted prior to surgical intervention [12]. As signs and symptoms of infection can be subtle , we recommend obtaining screening laboratories, including complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C reactive protein (CRP).

The physical examination may be difficult in the setting of a nonunion. It should attempt to focus on the strength, range of motion, and stability of the upper extremity from the shoulder to the digits utilizing the unaffected side for comparison whenever possible. Wrist range of motion, including flexion, extension, pronation, and supination, should be tested. A complete motor and sensory examination should be performed. When combined with provocative tests for carpal tunnel syndrome, it can expose an underlying median nerve injury. Skin inspection should be performed for any previous surgical incisions, which may have an effect on the choice for later surgical approach. Also, given the association of distal radius fractures with complex regional pain syndrome, special attention should be given to disproportionate pain, finger stiffness, swelling, allodynia, or paresthesias. When the patient describes a history of instability, stress tests should seek to localize it to the radiocarpal, midcarpal, or DRUJs. Although TFCC injuries commonly occur with distal radius fractures, radiocarpal or midcarpal instability is extremely rare. Increased anterior to posterior translation of the ulna on the radius compared to the contralateral side can be indicative of DRUJ instability. Finally, an Allen test should be performed to assess the specific vascular

supply crossing the zone of injury [13].

Radiographic examination constitutes the second core component of the clinical examination. All previous radiographs should be obtained and reviewed. A current series of wrist radiographs should be obtained, including PA, lateral, and oblique views. High-quality, appropriately aligned radiographs allow measurement of the key parameters outlined earlier that portend fracture stability and help determine surgical approach. These include radial height, radial inclination, radial tilt, ulnar variance, articular congruity, and distal radial ulnar joint stability [6, 7]. Radiographs may reveal persistent fracture lines, scalloping, or bone reabsorption in the distal segment, as well as loose or broken hardware. Wrist flexion and extension radiographs may provide additional benefit by revealing fracture site motion. Hypertrophic, oligotrophic, and atrophic radiographic appearance allows the clinician to make inferences about the degree of fracture stability, the presence of infection, and the biologic viability of the fracture fragments prior to developing a treatment plan. The presence of an atrophic nonunion should raise concern for an underlying infectious process. Also, comparison views of the contralateral wrist can aid with preoperative planning. While the radiographic parameters serve as a useful benchmark in treatment, each patient's symptoms and function should be taken in account when determining a treatment course.

The role of advanced imaging is difficult to define and should be considered on a case-by-case basis. Rotational deformity and articular congruity are often difficult to assess on plain radiographs, and computed tomography (CT) can be a useful adjunct. Three-dimensional reconstruction images provide information on both articular displacement and axial plane deformities (Fig. 7.1). CT also aids in assessment of bone reabsorption and osteopenia, which may influence fixation method.

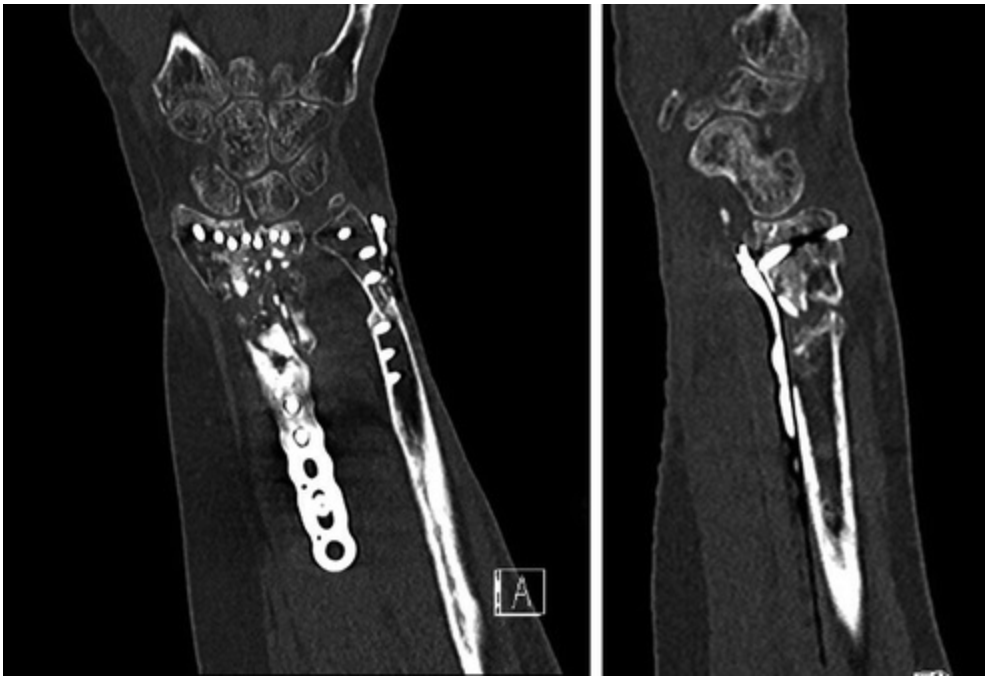


***Fig. 7.1*** Computed tomography evaluation of distal radius nonunion after attempted fixation and allograft bone grafting for comminuted open fracture

## 7.2.5 Treatment

Given the rarity of distal radius nonunions, it is not possible to provide treatment recommendations based on statistical analysis. Instead, each patient should be considered individually, with the appropriate treatment determined by the fracture personality, patient demands, and presence or absence of infection. Nonoperative treatment is typically appropriate only in the setting of elderly patients with very low functional demands. Historically, the most common treatment of nonunion was with wrist arthrodesis. However, advances in implant technology, such as fixed angle volar locking plates, have improved fracture union rates. Open reduction and internal fixation may have a significantly positive impact on upper extremity function through the resulting preservation of wrist motion [1, 14, 15]. Similar to malunited distal radius fractures, patients with nonunion often experience activity limiting pain and instability, with the majority of problems arising from fracture malalignment. In the setting of acute distal radius fractures, the radiographic parameters of radial height, radial inclination, radial tilt, ulnar variance, and articular congruity are used to correct malalignment. These same parameters are useful in the treatment of nonunion. The goal of treatment should be to

provide acceptable, stable fracture alignment with a soft tissue envelope devoid of infection and a biologic environment that is capable of fracture healing.

As discussed earlier in the chapter, failure of healing of metaphyseal distal radius fractures is rare. The definition of delayed union and nonunion of the distal radius is not clearly defined in the literature. While the severity of injury plays an obvious role in the rate of healing, one would expect to see radiographic evidence of progressive healing within 3 months after initial injury. Even in fractures with extensive comminution, one would expect evidence of healing after 4 months. When there is lack of progressive healing in the latent nonunion period between 3 and 6 months from injury or initial treatment, continued immobilization and limb disuse are likely to have a detrimental effect on function and range of motion. Further conservative treatment also fails to address the primary problem if malalignment or atrophic changes are present on radiographs. Therefore, we recommend a low threshold to surgical intervention during this time period.

Surgical treatment of nonunited distal radius fractures with multiple fracture fragments is challenging due the osteopenic quality of the bone, potential presence of fracture reabsorption, and limited options for fracture fixation. The majority of these injuries will present after a failed attempt at previous surgical fixation. In this setting, the orthogonal approach to plating as outlined by Fernandez, Ring, and Jupiter can prove useful [1, 16]. The use of a dual plating technique with fixed angle locking plates provides a greater number of fixation points in the distal segment and greater stability in the setting of atrophic or osteopenic bone (Fig. 7.2). Using this technique, the distal radius is approached through a volar or combined volar and dorsal approach, depending on the type of deformity present. The hybrid volar approach proposed by Chhabra et al. can also be utilized to release the carpal tunnel if median nerve symptoms are present preoperatively or if substantial deformity correction or soft tissue contracture may place the median nerve at risk postoperatively [17]. The fracture ends are identified, fibrous tissue and synovial membrane are removed, and sclerotic or necrotic bone is resected. The medullary canal of each end of the metaphyseal component of the fracture is opened to facilitate intraosseous ingress of blood and growth factors for fracture healing. Release or z-lengthening of the brachioradialis and flexor carpi radialis tendons may be required to correct loss of radial height and inclination. An external fixator with 2.5-mm Schanz screws is

often used to aid with reduction and maintain alignment prior to internal fixation. Care should be taken to not place Schanz pins where they may interfere with plate fixation. Kirschner wires are used to stabilize individual fracture fragments. Using the Rikli and Regazzoni columnar classification, one plate is placed on the radial styloid and lateral radius to control the lateral column, while a second plate is placed on the volar or dorsal cortex to stabilize the intermediate column [8]. We recommend initial fixation of the more stable column to the radial shaft, as this may aid with determination of alignment for the more complex or comminuted column. Autogenous cancellous bone graft is used to pack the defect. Following fracture fixation, the distal radial ulnar joint should be assessed for congruency and arthrosis. If uncorrectable malalignment or advance arthritic changes of the sigmoid notch–distal ulna articulation are present, a salvage procedure such as a Darrach distal ulnar resection or Bowers hemiresection interposition arthroplasty may be required.

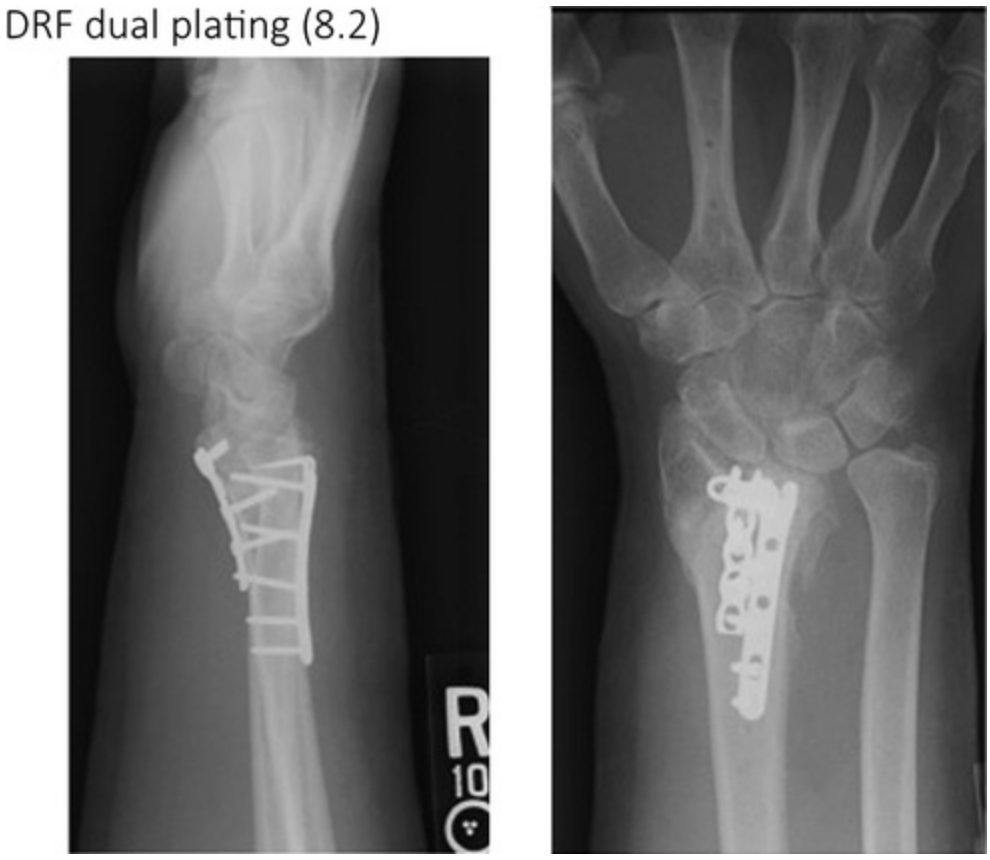



***Fig. 7.2*** Volar and dorsal dual plating of a distal radius fracture

Ring and Jupiter reported on a total of 23 patients treated with this technique over a 10-year period [18]. At an average of 28-month follow-up, all but one had gone on to a successful union. The one persistent nonunion was eventually treated with a wrist fusion. Two patients were treated with DRUJ salvage procedures (Darrach or Bowers). While patients demonstrated significant improvement in range of motion and function, they never regained normal motion and only 7 of 23 had good or excellent results, according to the rating system of Fernandez. In another group of 10 patients, Fernandez et al. reported similar results [1]. All 10 successfully healed their fractures. Distal ulnar salvage procedures were performed in 4 patients. Average wrist flexion was 50°, wrist extension was 55°, and pronation and supination were 70 and 75°. According to the Fernandez functional result system, there were 3 excellent, 4 good, 2 fair, and 1 poor result.

Given that the majority of distal radius fracture nonunions have undergone previous surgical intervention, failure to adequately address patterns of fracture instability should be considered in the preoperative evaluation. The most common example of this is the failure to recognize and stabilize the volar ulnar corner fragment. While the advance in fixed angle volar plating techniques has overall improved fracture fixation and allowed earlier return of function, it can be difficult to maintain reduction in complex intra-articular fractures with a volar ulnar corner or rim fracture of the distal radius. Stability of the volar ulnar corner is critical to providing structural support to the carpus and failure to maintain reduction leads to volar carpal subluxation or dislocation and catastrophic effects on wrist function [19–21]. The fragment-specific fixation method proposed by Medoff allows use of two or more low-profile implants to strategically capture specific fracture fragments [10]. This technique may be useful in the setting of a latent nonunion before osteopenia and fracture reabsorption have developed. Newer fixed angle, low-profile hook plates may allow more stable fixation of the volar rim in osteoporotic bone, as long as significant metaphyseal comminution is not present [22]. In the setting of significant metadiaphyseal comminution or fracture reabsorption, the use of low-profile implants is contraindicated, but the principles of fracture-specific fixation remain the same and control of the volar-ulnar fragment is critical for stabilization of the carpus.

Dorsal distraction bridge plating is a useful technique in nonunions with extensive comminution and bone loss [23, 24]. Segelman and Clark [2] have

suggested that union may not be possible if less than 5 mm of subchondral bone is present beneath the lunate facet, as there is inadequate space available for implant fixation. Dorsal distraction plating alleviates this problem and allows for both correction of severe radial shortening and bridging of osteopenic metaphyseal bone (Fig. 7.3). An initial 4-cm dorsal incision is made over the second or third metacarpal, and the extensor tendon is retracted. Choice of the second or third metacarpal is a matter of surgeon preference and may be influenced by individual fracture characteristics. A 4-cm second dorsal incision is centered over the distal radius, at least 4 cm proximal to the level of fracture comminution. Fluoroscopic superimposition of the dorsal plate can aid with plate selection and positioning of the proximal incision. Typically, a 12 or 14 hole limited contact, dynamic compression locking plate is used. Blunt dissection of the proximal incision between the brachioradialis and second dorsal compartment tendons is performed until the dorsal distal radius is exposed. Care should be taken to avoid damage to the superficial sensory nerve, which emerges from deep to the brachioradialis.

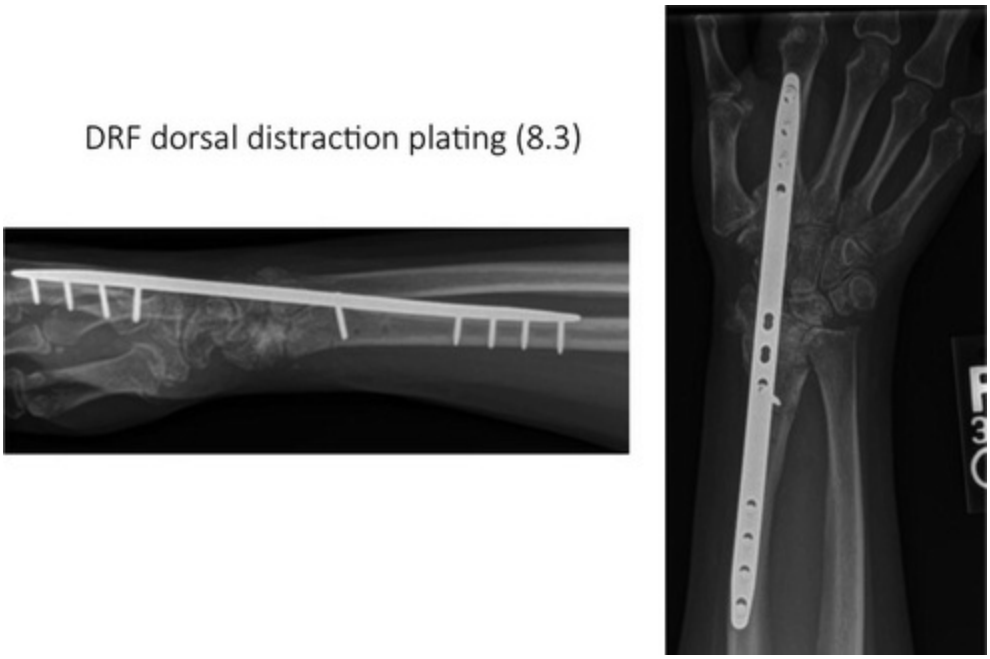



***Fig. 7.3*** Dorsal distraction (bridge) plating of a distal radius nonunion

If the second metacarpal is chosen, the plate will be placed in the second dorsal compartment, and if the third metacarpal is chosen, the plate will be placed deep to the tendons of the fourth compartment. A Freer or Cobb

elevator can be used to create a path from the distal to the proximal incisions, and the plate is slide from distal to proximal, avoiding impingement of the extensors. A cortical screw is placed in the center hole of the plate distally to stabilize it to the metacarpal. Fracture reduction is performed with longitudinal traction to restore length, palmar translation of the hand relative to the forearm to correct radial tilt, and pronation of the hand relative to the radius to counteract supination of the carpus. Once reduction is confirmed, a second cortical screw is placed in the middle hole of the proximal end of the plate. The remaining holes are filled with locking screws. Fluoroscopy and physical examination are used to rule out over distraction. The radiocarpal space should not be greater than 5 mm, and full passive finger flexion should be present. Supplemental fixation, including Kirschner wires and fragment-specific plates, can be used to attempt to improve alignment and articular congruency. Iliac crest autogenous bone graft can be used to fill defects. Following surgery, patients are placed in a short arm splint and digit motion is initiated immediately. The plate is removed at the time of radiographic fracture consolidation, and wrist range motion and progressive strengthening are started.

Mithani et al. [23] evaluated a total of 8 patients treated with this technique, reporting healing in all, with plate removal at an average of 148 days from surgery. Patients reported statistically significant improvement in range of motion and DASH scores from preoperative values. Despite successful healing, this technique has some potential complications. Patients must be instructed that plate immobilization for 5–6 months results in a significant loss of wrist range of motion, which requires committed and prolonged therapy to recover. Additionally, over distraction of the wrist can lead to digit contracture, radial nerve neuritis, and complex regional pain syndrome. Finally, there is a risk of tendon irritation and even tendon rupture if the plate is placed in a position that causes tendon entrapment.

Wrist arthrodesis was historically recommended as first-line treatment for distal radius fracture nonunion (Fig. 7.4) [16, 17]. However, since preservation of even minimal wrist range of motion results in a significant improvement in upper extremity function, it is now more commonly employed as a salvage procedure. It is effective a achieving bone stability when reconstruction is either not possible or not recommended. Potential indications include advanced radiocarpal or midcarpal degenerative changes and extensive bone loss or reabsorption.


***Fig. 7.4*** **a** Chronic nonunion of distal radius fracture after volar plating. **b** Treated with hardware removal and wrist arthrodesis

Management of infection in the setting of nonunion adds an additional layer of complexity. A low threshold should be maintained for the suspicion of infection, especially with the appearance of an atrophic malunion on radiographs. Screening white blood cell count, CRP, and ESR are recommended in all patients. If elevated, CRP and ESR are both independently predictive of infection, and the likelihood of infection increases with each additional positive test [25]. Radionucleotide bone scans have been recommended as an additional screening tool for infection, but they are not cost-effective and do not increase the predictive value in the setting of positive laboratory values [25]. Intra-operative tissue cultures from the nonunion site and any associated purulence can be obtained to provide a definitive diagnosis and aid with antibiotic therapy. There is no literature to guide whether infections of the distal radius with retained hardware can be treated with a single operation or require a staged approach. In the setting of gross contamination, eradication of the infection is advisable prior to proceeding with definitive fixation. An external fixator can be useful in correcting and maintaining alignment, while allowing for repeat surgical debridement. Treatment of an infected distal radius nonunion should be viewed as a collaboration between the hand surgeon and an infectious disease team well experienced in the care of orthopedic infections.

Autogenous cancellous bone graft is used to improve biology and fill gaps created by fracture realignment. The distal ulna, olecranon, and iliac crest can all be used a potential donor sites, depending on the amount of graft needed. In the setting of a large segmental defect, tricortical iliac crest graft

can be harvested, but with increased donor site morbidity. Bone graft substitutes have also been developed, including bone morphologic protein, demineralized bone matrix, and synthetics, such as calcium phosphate, calcium sulfate, and hydroxyapatite. While they remove the risk of donor site morbidity, all have significantly increased cost and have not proven superior to autograft bone in fracture treatment [26, 27]. Calcium phosphate grafts have an osteoconductive potential and high compressive strength and may be useful when combined with an osteoinductive substance to fill a large segmental defect.

### 7.2.6 Postoperative Care

Given that many patients will have significant preoperative stiffness and dysfunction from deformity and prolonged immobilization, early postoperative range of motion should be emphasized, with initiation of digit and forearm range of motion on the morning after surgery. Patients are immobilized in a postoperative short arm splint for a total of 10–14 days. After this, a removable splint can be placed and rehabilitation is initiated, focusing on early progressive range of motion. Strengthening exercises are restricted until there is radiographic evidence of healing, usually 12 weeks from surgery. With more extensive bone loss, healing may be detailed. Patients should be counseled that this is a salvage procedure in the setting of significant preoperative stiffness, deformity, and dysfunction and that recovery of full range of motion and strength is unlikely.

---

## 7.3 Distal Radioulnar Joint Instability

### 7.3.1 Background

The DRUJ is a diarthrodial articulation that acts as the distal stabilizing structure between the radius and ulna. It functions as a pivot point, allowing the radius to rotate round the ulna in supination and pronation. The ligamentous structures, which confer its stability, can be injured in a mechanism similar to that, which produces distal radius fractures. This typically consists of an axial load with wrist pronation and extension, such as a fall on an outstretched wrist. As a result, DRUJ instability can occur either in isolation or the setting of a distal radius fracture [28].

Chronic instability can occur from both a nonunited fracture of the base

of the ulnar styloid or a purely ligamentous injury to the TFCC and DRUJ joint capsule. In the latter setting, the instability can be viewed as a consequence of soft tissue “nonunion” of the DRUJ. Given the frequency with which DRUJ instability accompanies a distal radius fracture, as high as 11% in one study, the management of distal radius nonunion and malunion should include a clear understanding of the evaluation and management of this injury [29].

## 7.3.2 Anatomy and Biomechanics

The articulation between the distal radius through the sigmoid notch and the ulnar head encompasses the bony architecture of the DRUJ. The relative asymmetry of this relationship results in minimal conferred stability. The sigmoid notch is significantly more shallow than the ulnar head, with a radius of curvature that is 50–100% greater (15–19 mm vs. 10 mm) [28, 30]. Given the size mismatch with the ulnar head, the volar and dorsal rims of the sigmoid notch must contribute to stability. Post-traumatic deficiencies of either rim lead to decreased joint stability in biomechanical testing [28, 31–34]. The dorsal rim has an acute angulation, while the volar rim is rounded with a fibrocartilaginous lip [28]. There is significant variation between the coronal and axial alignment of the DRUJ articular surface. In the coronal plane, the joint assumes one of three slopes relative to the long axis of the radius and ulna: parallel, oblique, or reverse oblique [33]. While, at baseline, the shape has no effect on stability or function, changes in relative length of the radius or ulna may result in a mismatch between the articular surfaces and increased contact pressure. For instance, an ulnar shortening osteotomy in a patient with a reverse oblique configuration can result in loading and increased contact pressure in the proximal ulnar head and sigmoid notch [35].

In the axial or transverse plane, there are 4 potential configurations of the sigmoid notch: flat face, ski slope, C type, and S type. The flat notch shape has the least inherent stability and is more prone to failure with soft tissue reconstruction alone in a cadaveric model [36]. The ulnar head is the stable unit of the DRUJ articulation, acting as a platform around which the radius rotates. The contour of the ulnar head articular surface is often slightly asymmetric when compared to that of the sigmoid notch, which can lead to a CAM effect with forearm rotation and further propagate instability with ligamentous injury [28]. The end result of these anatomic factors is that only

20% of DRUJ constraint is provided by the ulnar head and sigmoid notch articulation, with the majority of stability contributed by soft tissue attachments, including the TFCC [32].

Along with the TFCC, the DRUJ capsule, pronator quadratus, extensor carpi ulnaris, and interosseus membrane all contribute to the stability of the DRUJ. The components of the TFCC are the most important in providing stability and maintaining DRUJ kinematics [30, 37]. The TFCC is comprised of several confluent structures that provide distinct functions. They include extending the articular surface of the radius to cover the ulnar head, absorbing and transmitting axial load forces, linking the distal radius and ulna, and supporting the ulnar carpus [28]. The components of the TFCC are the volar and dorsal radioulnar ligaments, volar and dorsal ulnocarpal ligaments, articular disk, meniscus homolog, and extensor carpi ulnar tendon sheath [28, 37]. The radioulnar ligaments contribute the primary stabilizing force to the DRUJ and are necessary for normal stability with volar and dorsal translation. Cadaveric studies have demonstrated that they maintain normal DRUJ joint kinematics after division of other soft tissue stabilizers [38]. Originating from the volar and dorsal aspects of the sigmoid notch, each ligament divides into two limbs in the coronal plane as they extend across the ulna. The deep radioulnar ligament is the more proximal of the two as it attaches to the fovea, a shallow concavity at the base of the ulnar styloid that is bare of cartilage and contains blood vessels that supply the TFCC. The superficial limb courses distally, inserting on the mid-portion of the ulnar styloid [39]. The differing attachment points of the ligaments exert an effect on the stability of ulnar styloid fractures. Fractures of the base of the styloid often indicate instability of the DRUJ from detachment of the deep limbs from the fovea [28].

The precise mechanism by which the radioulnar ligaments provide stability remains in debate, although both ligaments must be present to prevent translation in either direction [28, 32, 40]. In one proposed mechanism, the dorsal radioulnar ligaments act as the primary restraint from dorsal dislocation of the ulna with pronation, while the volar ligament opposed volar escape in supination. This is supported by a cadaveric study measuring ligament strain at the extremes of rotation [40–42]. The other theory is the exact opposite and was developed by observing bone displacement with application of a translation force. In this scenario, the volar ligaments opposing dorsal displacement in pronation and dorsal ligaments

countering volar displacement in supination via a tethering effect [32, 43]. Both theories have little effect on clinical management, other than reinforcing the idea that the ligaments exert a synergistic effect and that injury to both ligaments should be expected in the setting of bidirectional or severe unidirectional instability that permits joint dislocation [28, 40].

The articular disk of the TFCC is composed of fibrocartilage, which extends from the articular edge of the distal radius until in blends with the deep volar and dorsal radioulnar ligaments. Its primary function is to bear and transmit compressive loads across the DRUJ and provides minimal stability to DRUJ translation [41, 44]. The deep radioulnar ligaments reinforce the disk by preventing splaying with compressive force [35].

The ECU tendon sheath runs from the dorsal ulnar head to the carpus. It not only stabilizes the ECU tendon, but acts to augment the dorsal DRUJ joint capsule. The volar portion of the TFCC also contains three ulnocarpal ligaments named for the carpal bone to which they insert. These are the ulnotriquetral, ulnolunate, and ulnocapitate ligaments. The ulnotriquetral and ulnolunate ligaments originate from the volar radiolunate ligament, while the ulnocapitate ligament runs more volar and originates from the fovea. They theoretically provide a restraint to ulnocarpal translation and rotation, although their contribution to DRUJ stability is unknown [28]. The meniscus homolog is named for the loose connective tissue that occupies the space between joint capsule, disk, and proximal surface of the triquetrum and provides an unknown function [28].

### 7.3.3 Clinical Evaluation

Incompetency of the soft tissue stabilizers of the DRUJ manifests clinically as complaints of pain, decreased grip strength and mechanical symptoms [39]. As with any initial assessment, it should begin with a detailed history of the patient’s injury mechanism and attempted treatment. The location, quality, severity, and frequency of pain and instability should be obtained, along with any factors that alleviate or aggravate symptoms. Patients will typically describe a history of a fall on an outstretched wrist or passive forceful wrist rotation, such as with a jammed power tool [28]. Initial ulnar-sided wrist pain that is aggravated by forearm rotation, may, over time, evolve into activity limiting pain, weakness, and mechanical symptoms. Patients with more severe instability may describe a palpable clunk with forearm rotation activities, such as turning a screwdriver [28].

Given that injuries to the radiocarpal, ulnocarpal, lunotriquetral, and proximal radioulnar joints can cause similar symptoms, a comprehensive examination of the affected extremity from the elbow distal is required. The examiner should begin with inspection of the DRUJ, wrist, and forearm, evaluating for swelling or prominence of the ulnar head compared to the contralateral side. Tenderness to palpation of the ulnar styloid can occur in the setting of fracture nonunion. Tenderness of the fovea, located at the soft depression between the flexor carpi ulnaris, ulnar styloid, and triquetrum, can be indicative of a TFCC injury. Active and passive range of motion of both extremities, including wrist motion and pro-supination, should be measured for comparison. Significant crepitus or decreased and painful motion should be noted, as this may be indicative of DRUJ arthritis, which would be a contraindication to a soft tissue reconstructive procedure [45]. The shuck test is performed to assess for stability. The distal ulna is grasped with one hand, while the distal radius is stabilized by the other hand. The ulna is then forcefully translated in a volar and dorsal direction. Pain or increased translation compared to the contralateral side is indicative of DRUJ injury. Translation can vary depending on forearm rotation, so the test should be repeated in supination, pronation, and neutral positioning [28, 45].

Ulnocarpal joint stress testing should be performed to assess for TFCC disk tears and symptomatic ulnocarpal impaction syndrome. The presence of pain with the press test, in which a patient axially loads the wrist by using his or her arms to push up from a seated to a standing position, is indicative of a TFCC tear [45]. The test may also be informative in the setting of DRUJ instability. With attempted press up from the chair, the ulnar head will appear more depressed on the affected side. Maneuvers in which the wrist is taken through a range of motion (flexion–extension and/or pronation–supination) while the wrist is axially loaded in ulnar deviation are also useful in eliciting TFCC and ulnocarpal pathology [28].

ECU subluxation and tendinitis and lunotriquetral (LT) ligament tears can both result in symptoms similar to DRUJ injury. Instability of the ECU can be elicited by the “ice cream scoop” test with rotation of the wrist from a position of pronation and radial deviation to supination and ulnar deviation. Lunotriquetral ligament injury can be examined using the LT shear test. The lunate is stabilized between the examiner’s index finger and thumb, while the other hand provides a volar to dorsal translated force to the triquetrum. A positive test is manifested by pain [28].

### 7.3.4 Imaging

Initial radiographic evaluation should consist of standard PA and lateral views. Bilateral images are useful for comparison, and care should be taken to insure proper positioning in order to obtain symmetric views. Slight variations in rotation can exert a significant effect on measurement of radiographic parameters. Ulnar variance is best measured using the PA view, although diastasis between the distal radius and ulna, especially when not present on images of the contralateral limb, can be indicative of DRUJ instability. On occasion, a small fleck of bone is avulsed from the fovea, indicating detachment of the deep radioulnar ligaments [28]. In patients with symptoms of ulnocarpal impaction syndrome, the forearm pronated PA or clenched fist view can be helpful in assessing for dynamic ulnar positive variance [46–48]. The goal of an accurate lateral view is for the pisiform to bisect the volar surfaces of the distal pole of the scaphoid and the capitate (scaphopisocapitate lateral view) [28]. While commonly used to assess for DRUJ instability, it is imprecise, as a subluxed ulna can appear reduced and a dislocated ulna can appear subluxed with only 10° of forearm rotation [49]. Suspected instability can be further evaluated by lateral stress view with the patient holding a 5 lb. weight in a position of pronation [50]. Semi-pronated and semi-supinated views allow visualization of the sigmoid notch and ulnar head to assess for fracture, incongruence, and early degenerative changes and osteophyte formation [28]. Radiographs should also be used to assess for a malunited distal radius fracture, which may be the source of DRUJ instability.

Computed tomography can be a useful tool as it is able both to assess the congruency of the distal radioulnar articulation and to evaluate for advanced degenerative changes, which would contraindicate a soft tissue reconstruction. It is most useful in the case of a symptomatic patient with subtle instability [28]. For complete comparison, both wrists should be imaged in identical forearm positions, including neutral, pronation, and supination [28]. Measurement methods utilize the axial images. These include the dorsal and volar radioulnar lines, congruency method, epicenter method, and radioulnar ratio [49, 51, 52]. Numerous studies have demonstrated variability in findings for all methods. So, most authors recommend combing multiple measurements, while continuing to rely most heavily on the patient examination and clinical history [28].

Due to its ability to provide detailed images of the soft tissue structures,

MRI is the primary advanced imaging modality utilized in evaluation of TFCC tears in the acute setting. Arthroscopy remains the gold standard for diagnosis of TFCC injuries, but the sensitivity and specificity of MRI continues to improve [28, 53]. In the subacute or chronic setting, the use of MRI is less well defined. Although it has yet to be formally tested, MRI may be useful for assessing attenuation of the TFCC and resulting inability to perform a primary repair in the patient who present weeks or months from initial injury.

---

## 7.4 Treatment

### 7.4.1 Acute Distal Radioulnar Joint Instability

The treatment of acute instability of the DRUJ falls largely outside the scope of a chapter on nonunion of the wrist and hand, but bears mentioning for sake of completeness. The most common cause of acute DRUJ instability is a distal radius fracture. The majority of these injuries will be stable following accurate fracture reduction and stabilization. Following management of the distal radius fracture, stability should be reassessed over a full range of pronation and supination. If stability is maintained in only full pronation or supination, the DRUJ should be pinned in that position. Commonly, dorsal dislocations are stable in supination, while volar dislocations are stable in pronation [28]. If instability persists, then an open repair of the TFCC, which is discussed in detail later in this chapter, is indicated.

### 7.4.2 Chronic Distal Radioulnar Joint Instability

The goals of any treatment for DRUJ instability should be restoration of stability and a full, pain-free range of motion. Although it is unknown if DRUJ instability predisposes a patient to arthritis, chronic instability symptoms will rarely improve without surgical management [28]. Functional bracing has been proposed, which showed effectiveness in decreasing subluxation and improving range of motion [54]. This may be a consideration in lower demand patients. Surgical treatment options include operative fixation of instability resulting from an ulnar styloid nonunion, direct repair of the TFCC, or soft tissue reconstruction. In the setting of chronic instability, the TFCC is frequently irreparable and a soft tissue reconstruction technique is indicated [39]. Less straightforward is the treatment of subacute injuries or

subtle instability, in which soft tissue attenuation is less pronounced.

Ulnar styloid fractures are a common finding in the setting of a distal radius fracture, occurring 61% of the time [55]. As discussed earlier, fractures of the tip of the styloid often retain stability of the DRUJ, as the deep volar and dorsal radioulnar ligaments remain intact [28, 56]. Fractures of the base of the styloid may involve both the deep and superficial ligaments and result in DRUJ instability, especially in the setting of significant fracture displacement [28, 29, 57]. Ulnar styloid fracture nonunions are often asymptomatic. If the DRUJ is stable, painful tip fractures can be excised without affecting stability [29, 57]. In the setting of a large fragment and stable DRUJ, excision of a symptomatic fragment should be performed with caution (Fig. 7.5). Stability should be re-assessed, and if the DRUJ becomes unstable, the TFCC should be repaired to the fovea of the styloid, using interosseus sutures [57]. In the setting of DRUJ instability, fixation of a styloid base fracture can be attempted. Numerous techniques have been described, including Kirschner wires, compression screws, mini fragment plates, tension band wiring, and sutures anchors [28]. The implant chosen depends on a combination of surgeon preference and the size of the fragment. DRUJ stability should be re-assessed following fragment fixation and, if instability persists, requires a soft tissue reconstruction procedure.

***Fig. 7.5*** Chronic ulnar styloid nonunion that remained asymptomatic

With regard to isolated soft tissue injury, tears of the ulnar-sided attachments of the TFCC are most commonly associated with instability of the DRUJ. While tears of TFCC are common in the setting of distal radius fractures, the majority will not cause acute TFCC instability and do not progress if adequately addressed at the time of injury [28]. Both arthroscopic and open repair techniques have been described for repair of TFCC injuries resulting in DRUJ instability. The indications for arthroscopic TFCC repair in the setting of chronic DRUJ instability have not been completely described, and there is concern that soft tissue repair alone may not confer adequate stability. Newer arthroscopic techniques, such as pushlock anchors, which facilitate repair of the TFCC directly to the fovea through a drill hole in the ulna, may be of benefit, but have not been adequately studied in this setting. Open repair is performed through a dorsal approach to the DRUJ between the fifth and sixth extensor compartments, as described by Adams [28]. The extensor digiti mini tendon is mobilized and retracted ulnarly. An L-shaped capsulotomy is then made in the dorsal capsule, with the longitudinal portion of the incision centered over the radial aspect of the ulnar neck and the transverse limb beginning proximal to the dorsal radioulnar ligament. With retraction of the capsulotomy, the TFCC can be visualized.

If amenable to repair, a second transverse capsular incision is made distal to the dorsal radioulnar ligament to visualize the tear. Sutures are passed in either a horizontal or vertical mattress configuration through the peripheral edge of the tear and adjacent joint capsule. Holes are placed in the ulna using K wires or a small caliber drill, facilitating direct repair of the tear to the bone of the fovea. A suture-passing device is valuable in passing the sutures through the bone tunnels. The sutures are then tied over the bone with ulnar reduced in neutral forearm rotation. The dorsal capsule and extensor retinaculum are closed in a single layer, excluding the extensor digiti minimi, which is left superficial to the closure. Following completion of the case, DRUJ stability should be restored. If not, augmentation with a soft tissue reconstruction should be considered.

Soft tissue reconstruction procedures are indicated in the setting of an irreparable TFCC injury. Numerous surgical techniques have been described, which can be divided into the categories of extra-articular linking of the radius and ulna via tenodesis or ulnocarpal sling and intra-articular

reconstruction of the radioulnar ligaments. Indirect reconstruction techniques have been studied in a cadaveric model by Adams and Petersen and failed to restore native DRUJ stability or kinematics [58]. However, they may be necessary in the setting of a previous ulnar head resection, when an anatomic reconstruction is no longer possible. These include proposed techniques by Boyes and Bunnell and by Hui and Linscheid utilizing a strip of the flexor carpi ulnaris (FCU) tendon to reconstruct the volar ulnocarpal ligaments [59, 60]. Both techniques raise concern for the long-term stability of the DRUJ due to the unknown contribution to DRUJ stability from volar ulnocarpal ligaments. There is an additional risk of loss of motion from the tethering effect of the tendon [39].

Attempted anatomic reconstruction of one or both radioulnar ligaments has been described in techniques by Scheker et al. [50], by Johnston et al. [61], and by Adams and Berger [31]. In the technique by Scheker et al., a tendon graft is used to reconstruct only the dorsal radioulnar ligament. This raises concern for the long-term stability of the construct, as cadaveric models have demonstrated that both ligaments must be ruptured for instability to occur [62]. Nonetheless, they reported that all 14 patients treated with the procedure were satisfied with their outcome, with no recurrent instability, improved grip strength, and near complete resolution of pain at an average of 1.5-year follow-up [50]. The techniques proposed by Johnston et al. and by Adams and Berger seek to reconstruct both the volar and dorsal radioulnar ligaments with a palmaris longus autograft [31, 61]. Both reported similar midterm results. Johnston et al. [61] reported satisfactory results in 13 of 14 patients, with range of motion at least 90% of the unaffected side in all patients. Adams and Berger reported that patients recovered approximately 85% of the grip strength and wrist motion of the contralateral side [31]. In the both studies, 12 of 14 patients were able to return to their previous level of employment [31, 61].

Similar to open treatment of a TFCC injury, the technique described by Adams and Berger utilizes a dorsal approach to the DRUJ between the fifth and sixth extensor compartments [28, 39]. The extensor retinaculum is divided longitudinally for later repair, and the EDM tendon is mobilized and retracted ulnarly. A dorsal L-shaped capsulotomy is performed, and care should be taken to not violate the ECU sheath. The periosteum of the dorso-ulnar distal radius is elevated, deep to the fourth extensor compartment. Depending on the size of the palmaris longus graft, a 3.2–4 mm cannulated

drill bit system is used to place a tunnel in the distal radius from dorsal to volar at a position approximately 5 mm proximal to the lunate fossa and 5 mm radial to the sigmoid notch. The same cannulated drill is used to place a second tunnel in the ulnar beginning in the ulnar neck and exiting at the fovea. C-arm fluoroscopy is valuable in confirming the position of the guide wires prior to drilling. A whipstitch is placed in each end of the graft, and a suture passer is then used to weave the graft through the radius and ulna. The remaining limbs of the graft are passed around the subcutaneous border of the ulnar neck and tied into place with the ulna reduced and the forearm in neutral rotation. Care should be taken to insure that branches of the ulnar nerve are entrapped in the construct. Additionally, if the graft is not long enough to pass around the ulna after passing through the ulnar tunnel, a biocomposite interference screw can be utilized to stabilize the graft within the ulnar tunnel [28]. The dorsal capsulotomy and extensor retinaculum are closed in a single layer, and the EDM tendon is left superficial to the capsular closure.

In patients with flat face alignment of the sigmoid notch or who have sustained a fracture of the rim of the sigmoid notch, an osteoplasty can be considered as an isolated or complimentary procedure to prevent dorsal subluxation of the distal ulna [28]. Axial computed tomography images can be useful in assessing notch alignment and deformity [28]. The procedure proposed by Wallwork and Bain can be combined with reconstruction of the TFCC ligaments [63, 64]. The distal radius is accessed through a dorsal approach to the DRUJ. Osteotomes are used to make 2 parallel transverse osteotomies in the dorsal ulnar corner of the distal radius, one just proximal to the subchondral surface and the second at the proximal edge of the sigmoid notch. A third, longitudinal cut is then performed connecting the parallel osteotomies 5 mm from the sigmoid notch. This produces a thin osteocartilaginous flap, which can be backfilled with cancellous bone graft from the distal radius. To this point, only a case report and a technique article have been published on this subject. Clinical trials would be beneficial to determine long-term results.

#### *7.4.2.1 Postoperative Management*

Following soft tissue reconstruction, patients are immobilized in a long are splint for 3 weeks in neutral forearm rotation. At 3 weeks, the splint is removed and they are converted to a short arm cast and limited forearm

rotation is permitted for an additional 3 weeks. The patient is then converted to a removable wrist brace to be used for an additional 2 months. Therapy is initiated with active and gentle-only passive wrist flexion, extension, pronation, and supination. Strengthening is started early, with care taken to avoid high forces with the arm in full pronation and supination. More aggressive passive range of motion and strengthening are delayed until the 4-month mark, with the goal of recovering 85% of native forearm rotation by 6 months. Return to activities and lifting greater than 5 lbs is delayed until at least 4 months post-surgery [28, 31].

---

## 7.5 Carpal Bones

### 7.5.1 Scaphoid

#### *7.5.1.1 Background*

The scaphoid is the most commonly fractured bone in the carpus, accounting for between 60 and 70% of all carpal bone fractures [65]. Fractures most frequently affect a young, active, male patient population. In a study of US military personal, the incidence was 121 per 100,000 person-years, with fractures most commonly occurring in males ages 21–24 [66]. Achieving union is of paramount importance, as fractures that fail to heal progress to a predictable pattern of disability, carpal collapse, and eventual radiocarpal arthrosis. Healing of these injuries is a complex process, influenced by fracture location and orientation, displacement, and vascular supply to the scaphoid. As a result of the ligamentous connections between the bones of the carpus, a healing scaphoid fracture is subjected to significant shearing and bending forces [65]. Despite this, nondisplaced or minimally displaced fractures that involve the body of the scaphoid and distal can be treated with rigid immobilization in a cast with expected union rates reported at greater than 90% [67]. Delayed diagnosis, fracture displacement greater than 1 mm, angulation greater than 15°, proximal fracture location, and evidence of osteonecrosis on radiographs represent risk factors for nonunion and are commonly used as indications for surgical management. The rate of union after acute surgical management has been reported in several meta-analyses and approaches 100% [68–72]. As a result, correct initial assessment and management of these fractures is of paramount importance. When fractures fail to heal as expected or present in a delayed fashion care of the nonunited

scaphoid can prove challenging. The correct approach to a delayed union or nonunion of the scaphoid is a topic of continuing research and debate among hand specialists and will be discussed in this section.

### *7.5.1.2 Anatomy and Biomechanics*

The bones of the carpus are aligned in two matching rows, supported by both intrinsic ligaments and a complex system of volar and dorsal extrinsic ligaments. The scaphoid has a complex three-dimensional anatomy, closely resembling a peanut, with articular cartilage covering 80% of its surface. It is the only carpal bone that bridges both carpal rows. There are three anatomic regions into which the bone is divided: the proximal pole, waist or body, and distal pole or tubercle. The proximal pole articulates with scaphoid fossa of the distal radius and the lunate, while the distal pole forms the scapho-trapezial-trapezoid articulation. The bone of the proximal pole is the most dense, as it transmits axial load across the carpus from the distal radius. In contrast, the bone of the waist has the lowest density, which may explain why a majority of fractures occur in this region [73, 74]. Morphologic evaluation has demonstrated that male scaphoids are longer than those of females, which may have an impact on screw length with surgical fixation [73].

As a result of the scaphoid being almost entirely covered with articular cartilage, there is minimal space for perforating blood vessels to enter. This unique anatomy results in a complex blood supply. A cadaveric study performed by Gelberman et al. determined that approximately 80% of blood flow was provided via a branch of the radial artery entering the dorsal ridge at the scaphoid waist and supplying the proximal pole in a retrograde fashion. The remaining 20% is supplied by further volar radial branches entering the distal pole. The tenuous blood supply of the proximal pole results in a unique susceptibility to nonunion and avascular necrosis following fractures of the scaphoid waist or proximal [75].

The precise mechanism by which a scaphoid fracture occurs has not been clearly elucidated, although it appears to be a combination of axial load and either wrist hyperextension or, less commonly, wrist flexion [76, 77]. As described earlier, healing potential is dependent on a number of factors including location, displacement, angulation, and vascularity [28]. Intramembranous ossification is the mechanism by which scaphoid fractures heal. The resulting lack of protective callus formation renders the scaphoid susceptible to mechanical forces throughout the healing process. This can

lead to fracture displacement or angulation if proper immobilization or stabilization is not provided. A scaphoid waist fracture is exposed to a combination of bending, shearing, and translation forces. Axial load applied to the wrist prior to fracture healing will result in progressive flexion and pronation of the distal pole. Over time, the distal pole will continue to angulate as volar bone is reabsorbed, leading to a “humpback deformity.” This is further compounded by the limited blood supply of proximal fractures. The combination of fracture displacement, angulation, and absent blood supply all contribute to the development of a nonunion and eventual avascular necrosis [28].

If left untreated, scaphoid nonunion leads to a predictable pattern of degenerative arthritis within the radiocarpal and midcarpal joints known as scaphoid nonunion advanced collapse (SNAC). The scaphoid is a vital link between the proximal and distal carpal rows. As a result, scaphoid nonunion leads to a significant disruption of carpal mechanics. In a normal wrist, the scaphoid and lunate are connected by the scapholunate interosseus ligament, which draws the lunate into a flexed position with the scaphoid with wrist radial deviation. The volar collapse of the distal pole in a humpback deformity results in reduced carpal height. With radial deviation, the lunate continues to extend along with the proximal pole of the scaphoid, while the distal pole remains in a flexed position. Over time, the resulting dorsal intercalated segmental instability (DISI) pattern becomes fixed and the progression of degenerative changes is similar to that observed in a scapholunate ligament deficient wrist. Degenerative changes begin in the radial styloid articulation with the scaphoid before spreading to the entire radioscaphoid, radiolunate, and scaphocapitate joints, and ending in pancarpal arthritis. Patients may be initially asymptomatic, but will eventually develop progressive activity-related pain. In one study, 97% of patients with at least a 5-year history of scaphoid nonunion demonstrated degenerative changes on radiographs [78].

#### *7.5.1.3 Clinical Evaluation*

The evaluation of patients with known or expected scaphoid nonunion should begin with a detailed history. Most patients will often present with pain, stiffness, or inability to resume normal activities beyond the period of time one would expect for fracture healing, while a subset will remain asymptomatic. Although it can be difficult, an onset of injury should try to be

obtained. Occasionally, patients will not be able to recall a specific event, but rather a decrease in function, onset of pain, or loss of motion. Clinical records should be reviewed to determine any previous treatment. In the case of closed management, the duration and type of immobilization use should be obtained. For patients treated with surgery, the approach and type of fixation used are beneficial for later preoperative planning. Although the majority of these patients are young and active, a complete medical history should be obtained. Particular attention should be applied to use of tobacco products. While not an absolute contraindication to surgery, their use is a risk factor for nonunion and the patient should be counseled for and offered help with tobacco cessation [79, 80].

The physical examination should attempt to localize the source of pain in as gentle a manner as possible. Wrist range of motion should be obtained and compared to the contralateral side. While pain is not always localized to the anatomic snuff box area or either pole of the scaphoid, diffuse pain and significantly decreased range of motion should alert the examiner to the possibility of advanced degenerative changes.

The goal of the radiographic evaluation should be to determine the degree of healing, alignment, and vascularity of the fracture, as well as any evidence of degenerative changes. Initial radiographs should include standard posteroanterior, lateral, 45 degree pronated oblique, and navicular (PA in wrist ulnar deviation) views. They may reveal sclerosis, cyst formation, bone reabsorption, fracture displacement or angulation, or hardware loosening or failure. The lateral radiographs can also be used to evaluate for a DISI deformity, with a scapholunate angle >60° or a radiolunate angle >30° [81, 82]. If present, it factors into preoperative planning, as correction of both the alignment of the scaphoid and the normal scapholunate relationship can be challenging [83]. All previous radiographs, including initial injury films, should be reviewed to determine the progression of healing and any evidence of progressive fracture displacement or angulation.

Computed tomography (CT) scans provide the most detailed images of the osseous anatomy and can be useful in determining nonunion in the setting of equivocal radiographs. CT has demonstrated high intra-observer reliability in determining displacement and fracture union (Fig. 7.6) [84]. CT can also provide valuable information regarding bone reabsorption following collapse and early evidence of degenerative changes. CT images are used to determine angulation of the scaphoid with the lateral intrascaphoid angle or height-to-

length ratio on sagittal images [84]. For accuracy, the CT should be oriented perpendicular to the long axis of the scaphoid, rather than the wrist [85]. The normal lateral intrascaphoid angle is 24°, while an angle greater than 45° is predictive of an increased risk of arthritis, even in healed fractures [86]. Although the height-to-length ratio has demonstrated a greater intra-observer reliability than the intrascaphoid angles, the clinical significance of this is unknown. Scaphoid collapse is considered significant with a height-to-length ratio greater than 0.65 [87, 88]. CT can also evaluate for technical errors, such as screw misplacement and inadequate fracture reduction and compression. Current CT protocols with metal suppression are useful in minimizing hardware artifact. While not as effective as MRI, CT can predict proximal pole osteonecrosis through increased radio-opacity of the proximal pole and lack of bridging trabeculae between fracture fragments [85, 89].

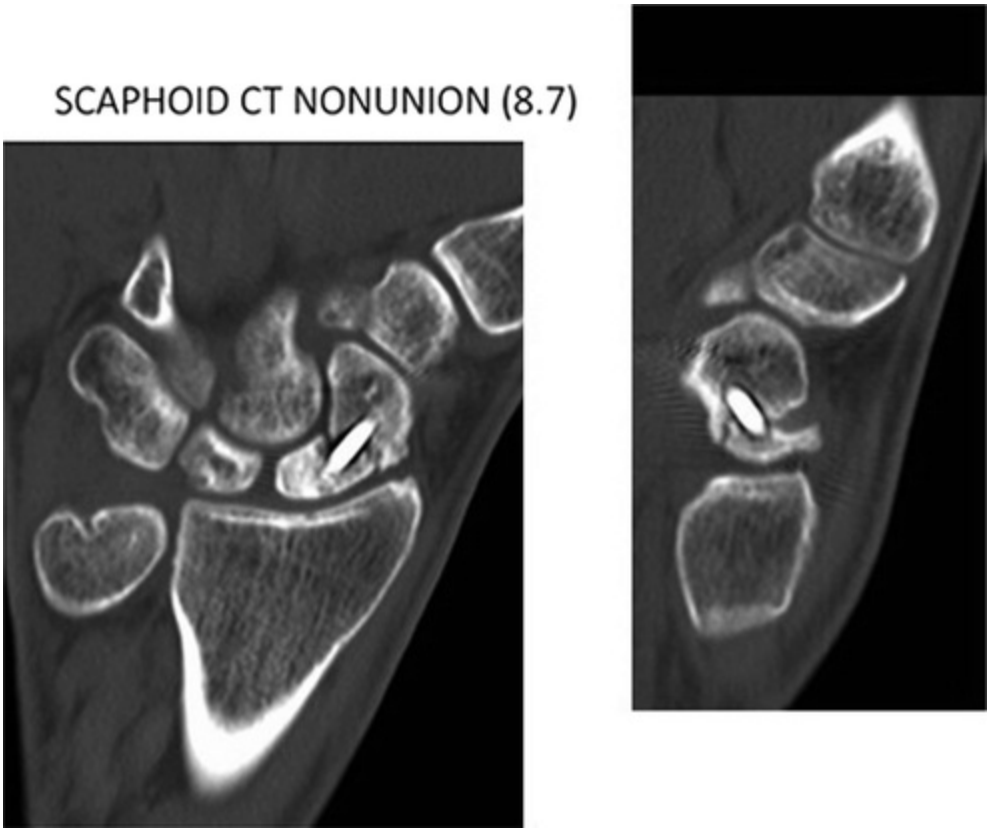



***Fig. 7.6*** Computed tomography scan showing nonunion of a scaphoid waist fracture previously treated with screw fixation

Due to the unreliability of radiographs in predicting osteonecrosis, MRI plays a key role in the preoperative evaluation of scaphoid nonunion. Studies

have established MRI as the most effective imaging modality in determining vascularity of the proximal pole (Fig. 7.7) [90–93]. It can also be effective in diagnosing occult scaphoid fractures and determining the degree of devascularized bone in patients who have undergone previous surgical treatment [83]. Decreased or absent signal intensity on T1 and T2 weight images has been associated with compromised vascular supply [93]. Patients with this finding on preoperative MRI demonstrated suboptimal healing rates when not treated with a vascularized bone graft [93]. Additionally, a clinical study has directly correlated absence of T1 signal on MRI with the presence of osteonecrosis, empty bone lacunae, and poor uptake on bone scan within intra-operative bone biopsy specimens [93]. While MRI is unnecessary if plain radiographs clearly demonstrate osteonecrosis, it is recommended to completely evaluate for the presence of osteonecrosis in any waist or proximal pole fracture with an established diagnosis of nonunion.

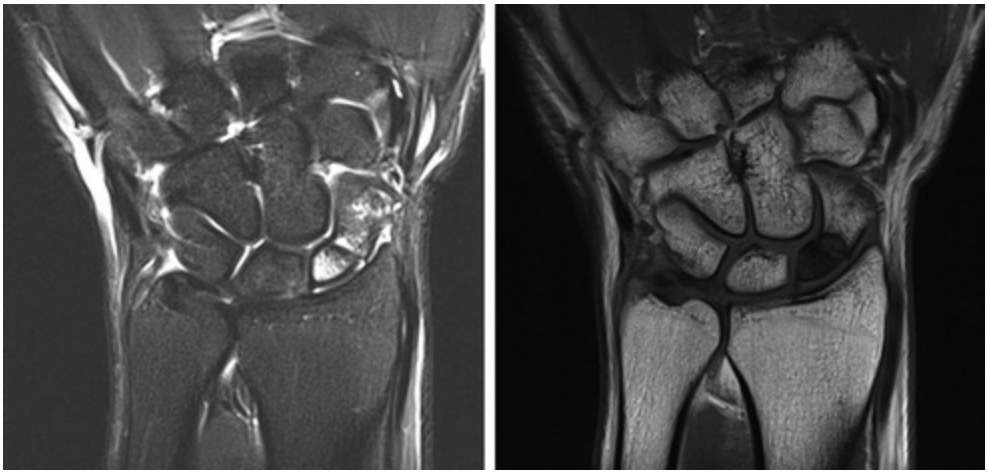


***Fig. 7.7*** Magnetic resonance image of scaphoid nonunion with signal changes on t1 and t2 consistent with avascular necrosis of the proximal pole

### *7.5.1.4 Treatment*

Surgical treatment is indicated in the setting of nonunion, as spontaneous healing is extremely rare [94]. The addition of cast immobilization and/or pulsed electromagnetic field treatment with a bone stimulator does not result in predictable consolidation once the diagnosis of nonunion has been established [95]. The healing rate was only 69% with the use of casting and bone stimulators in nondisplaced nonunions without radiographic evidence of osteonecrosis [95].

Delayed union represents a category of fracture that merits individual discussion. Although there is no clear definition of delayed union, it should be considered when radiographs fail to demonstrate expected progression of healing. Initial conservative treatment of nondisplaced fractures of the waist or distal pole is a reasonable approach. However, surgical intervention should be strongly considered when radiographs fail to show signs of healing after 6–8 weeks of immobilization and certainly by 12 weeks. Determination of healing can be difficult due to the lack of callus deposition, and a CT scan may be necessary for definitive evaluation. This is especially prudent in athletes and young laborers, as research has demonstrated faster return to play and work and decreased overall medical cost with early surgical intervention [96, 97]. Also, prolonged long arm cast immobilization can lead to elbow and wrist stiffness, exerting its own negative impact on function and quality of life. Further, management of delayed union without bone reabsorption, collapse, or osteonecrosis is technically less demanding and may be accomplished with compression screw without the need for bone graft. Whether initial patient presentation is acute or delayed, fracture displacement greater than 1 mm, fracture comminution, fracture of the proximal pole, fracture angulation as manifested by an intrascaphoid angle greater than 45° or height-to-length ratio greater than 0.65, and poor patient compliance all represent predictors of eventual non- or malunion and warrant immediate surgical management [86, 87, 98].

In the setting of established nonunion, the majority of authors recommend open reduction and internal fixation of the fracture with bone graft (Fig. 7.8) [98–102]. Throughout the evolution of surgical stabilization, a number of implants have been used, including Kirschner wires, staples, plates, and compression screws. The implant of choice must be capable of withstanding shearing forces with disrupt fracture healing. Kirschner wires lack the compressive strength necessary to maintain fracture reduction, while the use of staples and plates, although displaying satisfactory healing rates with acute fractures, raise concern hardware impingement and damage to the surrounding articular cartilage and often require later hardware removal [103, 104]. New mini-plate technology may alleviate some of these concerns, while theoretically provided increased torsional stability compared to a screw [105, 106]. Nevertheless, compression screws remain the mainstay for current treatment of acute scaphoid fractures and nonunion. Herbert developed the initial headless screw. Even without a compression design, they reported a

union rate of 100% for acute fractures and 83% overall [107]. Subsequent advances have demonstrating increased compression through a partially threaded or fully threaded, variable pitch design. The addition of a cannulated system utilizing guide wire placement under fluoroscopic guidance has improved accuracy of screw placement. Studies have demonstrated that accurate screw positioning is critical, with the greatest stability imparted by a screw positioned in the center–center position of the bone on PA and lateral views or perpendicular to the fracture line [108, 109]. Additionally, studies by Trumble et al. reported that screw placement within the central third of the proximal pole reduced time to union by 50% [81, 109, 110]. Several companies have now developed compression screws, and little data exists comparing their effectiveness. Therefore, screw choice remains largely dependent on surgeon preference.


***Fig. 7.8*** **a** Chronic nonunion of scaphoid waist fracture without avascular necrosis of the proximal pole. **b** Images 10 weeks postoperatively demonstrate healed fracture after volar nonvascularized bone grafting and compression screw fixation

After implant choice, the next steps in the treatment algorithm are surgical approach and choice of bone graft material. A combination of fracture stability and alignment, bone reabsorption, presence of osteonecrosis, and previous surgical intervention is used to determine the surgical approach

and bone graft source.

### 7.5.2 Scaphoid Nonunion Without Osteonecrosis

Well-aligned, stable nonunions with evidence of substantial bone loss or osteonecrosis represent the one category of fracture in which need for intra-operative bone graft remains controversial. A study by Shah and Jones found that fractures with a stable fibrous nonunion or intact cartilage cap healed without need for bone graft [111]. Additional studies by Slade et al. and Ikeda et al. demonstrated similar results, reporting healing of all nonunions treated with screw fixation alone if sclerosis was less than 1–2 mm on CT scan. Of note, fractures treated within 6 months of initial injury healed faster than older injuries [112, 113]. Fracture location determines surgical approach, with most authors recommending a volar approach for waist fractures in order to preserve the remaining dorsal blood supply and a dorsal approach for proximal pole fractures, which allows improved access and reduces the risk of displacing the proximal pole with guidewire placement [36, 65, 83].

Fractures that are well perfused, but with substantial bone loss, require the use of bone autograft to provide structural stability and enhance healing potential. The choice of cancellous versus corticocancellous structural graft remains an area of controversy. Geissler, Slade, and Gillon have argued that arthroscopically guided percutaneous fixation, and bone grafting is adequate for healing in nondisplaced or minimally displaced fractures with fibrous stabilization of the fracture site [65, 114]. In their technique, cancellous bone autograft is harvested from either the distal radius or iliac crest using a bone biopsy needle. A guidewire is then place within the scaphoid using a percutaneous of mini-incision dorsal approach. A second wire is placed as an anti-rotation wire, and arthroscopy is performed to confirm the presence of the fibrous nonunion, assess vascularity, and rule out ligamentous injury. Under arthroscopic visualization, a dorsal capsular release is performed using a curved hemostat through the 3,4 portal. The arthroscope is then removed, and the scaphoid is drilled for the compression screw. The guidewire is left in place, and bone biopsy needle is placed over it to deliver bone graft. Finally, a compression screw 4 mm shorter than the measured length of the scaphoid is placed. Using this technique on 108 scaphoid fracture nonunions with either no displacement or a reducible humpback deformity, Slade and Gillon reported a 96% healing rate at 9 months. Ten cases of delayed healing

required repeat percutaneous bone grafting [114]. Percutaneous bone grafting can also be performed through a limited volar approach, in the setting of a nondisplaced fracture nonunion. Using this technique, only the scaphotrapezial joint in exposed in the volar approach. The volar beak of the trapezium is removed to allow grafting and retrograde screw placement [36].

Other authors would argue that a corticocancellous graft is required for structural stability and that only an open approach allows complete evacuation of sclerotic bone, which can prevent healing. With open treatment of well-vascularized nonunions, the location of the fracture remains the primary determinant of the surgical approach. The volar approach described by Russe facilitates access to the waist and distal pole of the scaphoid, while preserving dorsal blood supply, especially in a fibrous nonunion or with an intact dorsal cortex [36]. The standard Russe incision starts with a longitudinal incision proximally over the flexor carpi radialis (FCR) tendon s and then angles obliquely in line with the long access of the scaphoid to the level of the scaphotrapezial joint. The FCR tendon sheath is incised to allow ulnar retraction of the tendon and protection of the palmar cutaneous branch of the median nerve. Care is taken to protect the deep branch of the radial artery, which is retracted radially. The volar radiocarpal ligaments are incised obliquely to allow access to the scaphoid and later repair of the ligaments. Preservation of as much RSC ligament as possible prevents volar subluxation of the scaphoid proximal pole [81, 110].

The Matti-Russe procedure describes corticocancellous grafting through the volar approach and is particularly useful in the setting of a fracture with minimal displacement, a large bone defect, and absence of a humpback deformity [115]. Any fibrous tissue at the site of the nonunion is removed, and an osteotome is used to expose the fracture line. Osteotomes or high-speed burr is then used to cut a cortical window, remove a strip of cortical bone, and create a trough for graft placement. All necrotic bone is excised from the proximal fragment, and punctate bleeding is a sign of preserved vascularity. If necessary, the scaphoid is reduced. Kirschner wires can be placed in each fragment as joysticks to help facilitate reduction. A corticocancellous graft equal in size to the cavitary defect is then harvested from either the iliac crest or distal radius. The Green modification of the procedure calls for two parallel grafts placed with the cancellous surfaces facing one another [116]. In the original description, the scaphoid is stabilized with parallel Kirschner wires, although compression screws can

also be used and provide a more stable construct (Fig. 7.9). Upon completion of the procedure, the capsulotomy is closed with nonabsorbable suture. Results of the procedure are varied and appear to rely most heavily on the location of the fracture. Treating a total of 84 patients, Dacho reported union rates of 82% overall and 81% in proximal pole fractures [117]. However, Barton reported a healing rate of only 54%, with the majority of failures occurring in proximal pole fractures [118]. This is supported by Green's study, which reported a healing rate of 71% that was negatively affected by a higher incidence of failure to heal in proximal pole fractures [116].

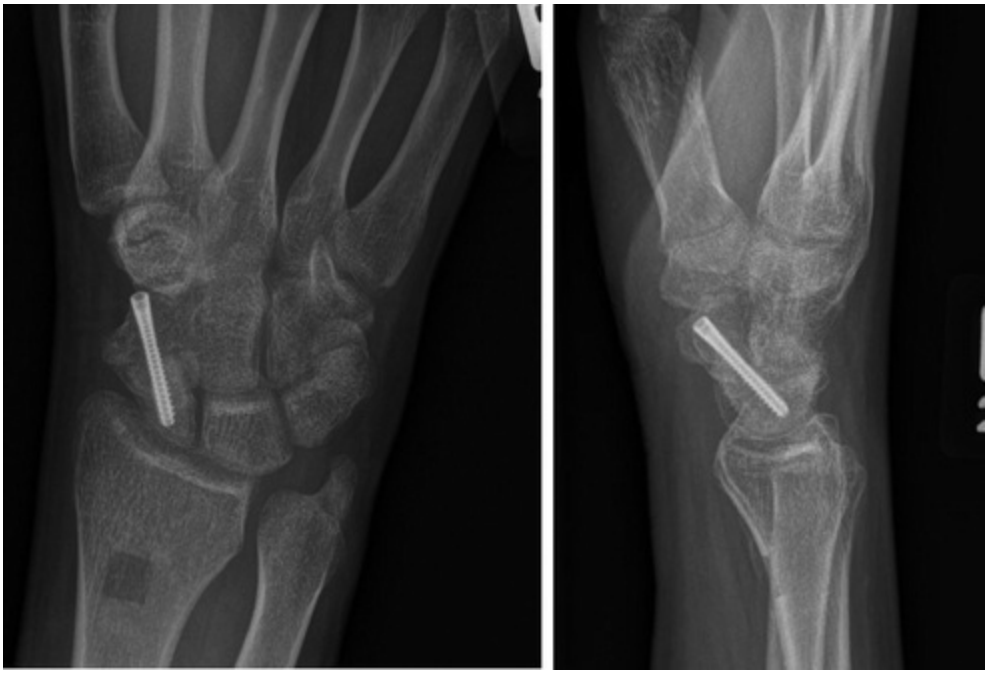


***Fig. 7.9*** Scaphoid nonunion without avascular necrosis treated with corticocancellous graft from distal radius with humpback deformity corrected and screw in place

Fractures that involve a viable proximal pole are best managed through the dorsal approach. The dorsal approach allows improved access to the proximal pole in order to remove nonviable, sclerotic bone and allows anterograde screw placement. The orientation of proximal pole fractures is commonly from distal volar to proximal dorsal, so a volarly placed screw runs the risk of both of displacing the proximal fragment and not crossing the center of the nonunion site [36]. The scaphoid may be approached from either a longitudinal incision in the middle of the wrist centered over the radiocarpal joint or an oblique incision from the Lister's tubercle to the base of the

second metacarpal [36, 65]. The capsule may then be divided longitudinally or with a capsular splitting [119] incision. In the dorsal capsular splitting incision, the dorsal radiocarpal ligament (DRC) is split from just distal to Lister's tubercle to the triquetrum, while the dorsal intercarpal ligament (DIC) is incised from the triquetrum to the distal pole of the scaphoid. The resulting ligamentous flap is elevated from ulnar to dorsal for later repair. The distal aspect of the extensor retinaculum can be incised, and Lister's tubercle can be utilized for either cancellous or corticocancellous bone graft. As with the volar approach, the nonunion site is identified and necrotic bone is removed with curettes or a high-speed burr. Kirschner wires placed in each pole of the scaphoid and used as joysticks may be beneficial in reducing the fracture. A guidewire for a cannulated screw system is placed under fluoroscopic guidance once the fracture has been reduced and bone graft has been placed. Placement of the wire is facilitated by flexion of the wrist. A second eccentric wire can be placed to prevent fracture rotation with drilling and screw placement. Upon completion of the case, the capsulotomy is closed with nonabsorbable suture. The literature examining dorsal approach and bone grafting in the absence of osteonecrosis is limited. Inoue et al. [120] reported on 16 patients, 13 of which went on to radiographic union.

In either a volar or dorsal approach to a fracture with extensive bone loss, resulting in concerns about stability after screw fixation, a second screw or Kirschner wire can be inserted as an augment [121, 122]. The use of a second screw requires prior planning, as the first screw must be positioned to either side of the scaphoid to allow space for the second. The Kirschner wire is typically left in place until there is radiographic appearance of fracture consolidation. Garcia et al. reported on a total of 19 patients treated with placement of 2 compression screws and bone graft through either a volar or dorsal approach. All fractures went on to union at an average of 3.6 months [122].

For patients with a fixed humpback deformity of the scaphoid or with a large cavitary lesion of the volar scaphoid that results in a DISI deformity, a volar approach is required for complete correction and stabilization of the deformity. Fernandez has described a modification of a procedure originally developed by Fisk for treatment of these scaphoid nonunions with carpal instability. The key components of his technique are as follows: (1) the use of preoperative radiographs of the contralateral wrist to assess correct scaphoid length and alignment; (2) the use of a volar approach; (3) the insertion of a

wedge-shaped corticocancellous graft from the iliac crest to correct alignment after resection of the pseudoarthrosis; and (4) stabilization with a compression screw [65, 123]. Fisk's technique used radial styloid bone graft, which Fernandez modified due to the greater compressive strength of the iliac crest. Fernandez reported radiographic healing in 19 of 20 patients treated with this technique with an average time of from work of 8.9 weeks [123]. Eggli et al. found similar results, with 35 of 37 patients achieving union. Preexisting osteonecrosis was present in both of the 2 cases that failed to heal [124].

### 7.5.3 Scaphoid Nonunions with Osteonecrosis

It is generally accepted that vascularized bone graft is necessary in the setting of proximal pole osteonecrosis, or when traditional bone grafting has failed to achieve union (Fig. 7.10). This is supported by studies that demonstrated unacceptable union rates in the setting of osteonecrosis [115, 116, 120, 124–126]. In a canine study, Sunagawa et al. reported a union rate of 73% if vascularized graft was used and 0% with conventional graft [127]. Multiple graft sources have been described, the most common being grafts harvested from the distal radius and rotated on an intact pedicle. Other techniques include pedicle graft from the second metacarpal and ulna, free grafts from the medial femoral condyle, iliac crest and rib costochondral junction, and arterialization.


***Fig. 7.10*** **a** Chronic nonunion of scaphoid waist fracture. **b** 4 months after compression screw fixation with volar nonvascularized bone graft and continued nonunion of fracture. **c**, **d** Images after revision fixation of nonunion with dorsal 1,2 intercompartmental supraretinacular artery vascularized bone graft and screw and k wire fixation with healing of fracture

Techniques for graft harvest from the distal radius are based on the comprehensive blood supply of the distal radius described by Sheetz et al. in 1995 [101]. Their close proximity to the scaphoid confers some advantages over other techniques. Minimal extra exposure and dissection are required for their harvest, and they are rotated on an intact pedicle, eliminating the need

for microvascular anastomosis [85]. The dorsal graft is typically based on either the 1,2 or 2,3 intracompartmental supraretinacular arteries, while the volar graft is from the volar carpal artery [65, 83, 85]. The nutrient vessels of the distal radius from which the dorsal grafts are based are named by their relationship to the extensor compartments of the wrist and the extensor retinaculum [65]. The 1,2 intercompartmental supraretinacular artery (ICSRA) lies superficial to the extensor retinaculum between the first and second dorsal compartments, while the 2,3 ICSRA is also superficial and between the second and third compartments [65]. The 1,2 ICSRA takes off from the radial artery 5 cm proximal to the radiocarpal joint. The 2,3 ICSRA has a longer pedicle and may allow for greater graft rotation [85]. An additional graft based commonly used in the treatment of lunate osteonecrosis (Kienböck disease).

Harvest of the 1,2 ICSRA was first described by Zaidemberg et al. in 1991 and is likely the most commonly used graft source in part due to its consistent location on the extensor retinaculum [36, 65, 128]. A dorsal radial skin incision is centered over the radiocarpal joint between the first and second dorsal compartments to allow exposure to the scaphoid and distal radius. A tourniquet is typically used for surgery, but exsanguination is not performed in order to allow identification of the artery. Branches of the superficial sensory radial nerve must be identified and protected. Meticulous blunt dissection is performed until the 1,2 ICSRA is identified as it courses dorsally from the radial artery to lie superficial on the extensor retinaculum between the first and second compartments. The artery typically appears as a thin red stripe. The first and second extensor compartment tendons are released. The tendons of the second compartment are retracted ulnarly, and the first compartment tendons are retracted radially. The arterial pedicle is mobilized by making parallel incisions in the periosteum on either side of the artery at the desired length for the graft. The pedicle should not be mobilized more than 10–15 mm proximal to the joint line, as this is the area where the nutrient vessels enter the bone. The pedicle is then freed to nearly the level of the radial artery to allow rotation of the graft. The desired size graft is then harvested.

We recommend the use of osteotomes for graft harvest, although some surgeons will utilize an oscillating saw. The artery is ligated proximally, and the tourniquet should be deflated to confirm blood flow through the pedicle. The nonunion site is prepared with complete excision of any necrotic bone as

previously described. Preparation of the nonunion can also be performed prior to graft harvest. Kirschner wire joysticks are again useful for control of the scaphoid with graft placement and fracture reduction. Compression screws are used for fixation as they have significantly better union rates than Kirschner wires [129]. If fracture fragments are completely unstable, fixation can be placed first, followed by impaction of the graft through a cortical window [65].

The use of the 1,2 ICSRA graft has demonstrated mixed results within the literature. Zaidemberg et al. [128] reported union in all 11 patients treated with the procedure in their original technique article. 34 of 50 nonunions treated by Chang et al. went on to heal at an average of 15.6 weeks post-surgery. Risk factors for persistent nonunion included advanced age, osteonecrosis of the proximal pole of the scaphoid, preoperative humpback deformity, lack of screw fixation, tobacco use, and female gender [129]. Straw et al. [130] reported less successful results, with only 6 of 22 patients achieving union. Of note, they used Kirschner wires for fixation, which may have negatively impacted union rates.

As mentioned previously, vascularized bone graft from the volar distal radius has also been described, based on the volar carpal artery. Gras and Mathoulin reported a union rate of 89.5% in 38 patients treated with the procedure, all of who had undergone previous surgical intervention. The average time to radiographic union was 10.8 weeks [131]. This technique may be particularly useful with the combination of osteonecrosis and a humpback deformity or large volar bone defect of the scaphoid that requires a volar wedge graft for deformity correction. Other options for this problem include a dorsal approach with a radial styloidectomy to facilitate volar placement of the ICSRA, a dorsal approach with a vascularized second metacarpal bone graft pedicle that is rotated volarly, or two separate approaches with a volar wedge graft to correct the deformity and ICSRA graft to provide vascularity [83].

Similar to the grafts of the distal radius, the second metacarpal vascularized graft is a pedicled graft that can be rotated into the scaphoid defect. The graft is based on second dorsal metacarpal artery or the dorsal intercarpal arch and can be harvested from the dorsal head or base of the metacarpal. The pedicle does not cross the wrist joint, theoretically decreasing the risk of occlusion due to vessel kinking. It can be harvested from a single dorsal approach or a dual approach, in which it is rotated and

placed in the volar scaphoid defect [85]. Mathoulin and Brunelli [132] reported a union rate of 93% and time to union of 4 months in 15 patients treated with grafts harvested from the metacarpal head. Despite the high union rate, only 10 patients had acceptable functional outcomes and 2 sustained radial nerve irritation. Sawaizumi et al. [133] used a graft from the proximal metacarpal, believing it allowed harvest of a larger piece of bone. They reported that all 14 patients treated with the procedure went on to union at an average of 10.2 weeks. Of note, the presence of preoperative osteonecrosis was not included in their study.

Ulnar bone graft utilizing the ulnar artery as a pedicle represents another option for treatment of avascular scaphoid nonunions. The procedure has some advantages, including a predictable vascular pedicle, large periosteal layer, and decreased donor site morbidity compared to free vascularized grafts [85, 134]. The greatest disadvantage is that the graft utilizes the ulnar artery as a pedicle, which requires division and reconstruction of the artery with an interpositional vein graft. In a study by Guimberteau and Panconi, all 8 patients treated with the procedure both went on to radiographic union at an average of 4.6 months and returned to their previous level of work or athletic activities without postoperative complications [134]. Given the possible complications associated with division and reconstruction of the ulnar artery and that patients had failed an average of 2 prior surgeries, the procedure might be most useful in the setting of revision surgery, in which distal radius pedicle graft has already been used.

With regard to free vascularized grafts, techniques for harvesting from the medial femoral condyle, iliac crest, and rib costochondral surface have been described. Free vascular grafts provide enhanced structural support, such as that needed to correct a humpback deformity. Potential complications include morbidity from a second surgical incision for graft harvest and failure of microvascular technique for anastomosis of the graft [85]. The medial femoral condyle graft is based on a pedicle from the descending geniculate vessels or the superior medial geniculate vessels [135]. Jones et al. [135] compared 1,2 ICSRA and medial femoral condyle grafts in the treatment of scaphoid nonunions with osteonecrosis of the proximal pole. All 12 patients treated with medial femoral condyle graft healed, compared to only 4 of 10 patients treated with 1,2 ICSRA graft. Average time to healing was 13 weeks in the medial femoral condyle group, compared to 19 weeks in the 1,2 ICSRA. The iliac crest free graft allows harvest of structurally sound

tricortical iliac crest graft utilizing the deep circumflex iliac vessels as a pedicle [136]. Arora et al. [136] treated 21 patients with documented osteonecrosis and who has failed previous surgical intervention. They reported a union rate of 76%, with an average time to union of 17 weeks and no donor site complications. Al-Jabri et al. [137] performed a systematic review of studies in which patients with nonunion were treated with either a medial femoral condyle or iliac crest free graft. They reported a combined union rate of 100% in 56 patients treated with a medial femoral condyle graft and 87.7% in 188 patients treated with an iliac crest graft.

Proponents of rib costochondral free vascularized bone graft tout its use in the setting of significant collapse of the proximal pole of the scaphoid [138–140]. A horizontal incision is placed over the 9th rib, and the osteocartilagenous surface of the costochondral joint is exposed. An osteochondral plug is harvested with an oscillating saw and contoured to match the anatomy of the proximal scaphoid. The proximal pole of the scaphoid is resected, and the graft is implanted and fixed to the distal pole with either a screw or removable Kirschner wires. Sandow reviewed 47 patients treated with the procedure [138]. All patients reported improvement in functional scores, with none requiring additional salvage procedures at an average follow-up of 3 months. In another study with longer follow-up, Veitch et al. reported functional improvement in 13 of 14 patients [139]. Graft union to the residual scaphoid was 100% in both studies. Tropet et al. [140] reported on 18 patients treated with technique at an average follow-up of 4.1 years, finding excellent or good results in 15 cases, fair in 2 cases, and poor in 1 case, in which there was subluxation of the graft.

Direct implantation of the second dorsal metacarpal artery or the dorsal index artery into the scaphoid has also been described and predominately used in the setting of previous surgery and limited vascularized bone graft options. Known as arterialization, the procedure was first described by Hori et al. in a canine model [141]. Fernandez and Eggli [142] reported the use of the procedure in 11 patients, 10 of who went on to heal their fracture. Despite the high union rate, 50% required subsequent operations, including radial styliodectomy and limited carpal arthrodesis. Two other case studies in patients who had undergone previous surgery reported successful fracture healing [143, 144].

**Author's Preferred Treatment Algorithm**: In patients who present with an established scaphoid nonunion with evidence of AVN, our preferred

method is compression screw fixation with vascularized bone grafting using a 1,2 ICSRA pedicled graft. However, in patients who have had previous dorsal wrist surgery or trauma or who have a significant humpback deformity that we do not feel would be correctable from the dorsal side, consideration is given to a volar approach and volar vascularized, pedicled graft [131]. Patients who present with very proximal pole fractures are offered attempted fixation with one of these techniques prior to any consideration of free vascularized reconstruction.

#### *7.5.3.1 Postoperative Management*

Following conventional or vascularized bone graft for scaphoid nonunion, patients are initially placed in a short arm splint. This is converted to a short arm cast at 2 weeks that is maintained until the 3 month postoperative mark or radiographic healing. At 3 months, patients are converted to a removable short arm splint that is removed only for gentle range of motion exercises with therapy. Splinting is continued until radiographs show fracture healing. Additionally, any lifting with the affected extremity is delayed until union is apparent on radiographs. Patients should be counseled preoperatively that they will develop significant wrist stiffness and substantial therapy will be required to regain strength and range of motion. They should also be warned that healing can be delayed in the setting of preoperative radiographs, with average time to union between 3 and 5 months post-surgery [128–131]. Although it is not part of our practice, supplemental use of a bone stimulator (either low-intensity pulsed ultrasound or electrical stimulation) can be considered, especially in patients who have failed previous surgery, significant delay in treatment or with preoperative osteonecrosis. Ricardo et al. have reported accelerated time to union (by 38 days) in patients with osteonecrosis who were treated with a bone stimulator [145]. No current evidence supports use of one stimulator type over another, and no comparative trials have been performed to date.

### 7.5.4 Salvage Procedures for Scaphoid Nonunion Advanced Collapse (SNAC)

With significant radiocarpal or midcarpal degenerative changes, surgical treatment of scaphoid nonunion is unlikely to produce a meaningful outcome. As mentioned earlier in the chapter, scaphoid collapse leads to a DISI pattern

of carpal instability, with degenerative changes progressing in a pattern that resembles scapholunate ligamentous instability advanced collapse (SLAC) [78]. Stage I SNAC degenerative changes are confined to the radial styloid. Stage II involves the entire radioscaphoid joint. In Stage III, degenerative changes spread to the capitolunate joint, and in Stage IV, pancarpal arthritis is seen.

Stage I wrists can be treated with scaphoid fixation and bone grafting combined with radial styloidectomy. With resection of the styloid, it is important to protect volar radial ligament attachments. Resection of greater than 1 cm of the radial styloid can result in destabilization of the origin of the radioscaphocapitate ligament [65].

In Stage II, progression of degenerative changes to involve the entirety of the radioscaphoid joint necessitates the use of a motion preserving salvage procedure. Excision of the distal scaphoid pole is an option in lower demand patients with fractures of the scaphoid waist and distal. Malerich et al. [145] reported improvements in grip strength of 134% and range of motion of 85% in 19 patients treated with the procedure. There was no radiographic progression of DISI deformity at average of 4 years postoperatively. Ruch et al. demonstrated satisfactory results in 13 patients at an average of 4 years after distal pole excision [146]. However, 6 of 13 had radiographic worsening of DISI deformity. For more active patients, proximal row carpectomy (PRC) and scaphoid excision with 4 corner fusion are surgical options. Compared to fusion, proximal row carpectomy has the advantages of being a less technically demanding procedure, with no risk of nonunion, shorter postoperative period of immobilization, and minimal postoperative rehabilitation. Comparison of PRC to scaphoid excision and 4 corner fusion has demonstrated improved patient satisfaction, grip strength, and range of motion in patients treated with PRC [147]. In relation to their contralateral extremity, patients can expect to regain 70–80% of their grip strength and 50–60% of their range of motion [147]. Radiographs will often demonstrate progression of arthritis to the capitolunate joint, but this has little functional impact, as most patients remain asymptomatic [148].

With progression of arthritis to the midcarpal joint in Stage III, proximal row carpectomy is no longer an option, and scaphoid excision and 4 corner fusion are indicated (Fig. 7.11). The articulation between the capitate, lunate, hamate, and triquetrum is the location of the fusion, with the goal of maintaining a congruent radiolunate joint. Biomechanical studies have

demonstrated that load is preferentially transferred to the radiolunate joint after the procedure is performed, so it is important to confirm intraoperatively that the joint is without significant degenerative changes. The procedure is also more technically demanding than PRC and obtaining consolidation of the fusion can be challenging. Inadequate correction of the lunate can lead to fusion of the capitate in a dorsally subluxed position and painful radiocapitate abutment that prevents wrist extension [65]. Implant design is an area of ongoing research. Techniques with Kirschner wires, circular plates, compression screws, and staples have all been utilized with the goal of developing rigid, low-profile implants. An ideal implant will also provide adequate bone compression and minimize soft tissue irritation. Outcomes from the use of circular plates have been mixed. Skie et al. [149] reported union of only 29 of 37 patients (78%). Despite achieving union in 10 of 11 patients treated with a circular plate, Chung et al. found that the majority of patients continued to experience persistent wrist pain and 1 patient required removal of painful hardware [150]. Finally, Shindle et al. reported complications in 9 of 16 patients treated with a circular plate, including nonunion (25%), delayed union (6%), dorsal impingement (25%), radial styloid impingement (6%), and broken screws (13%) [151].

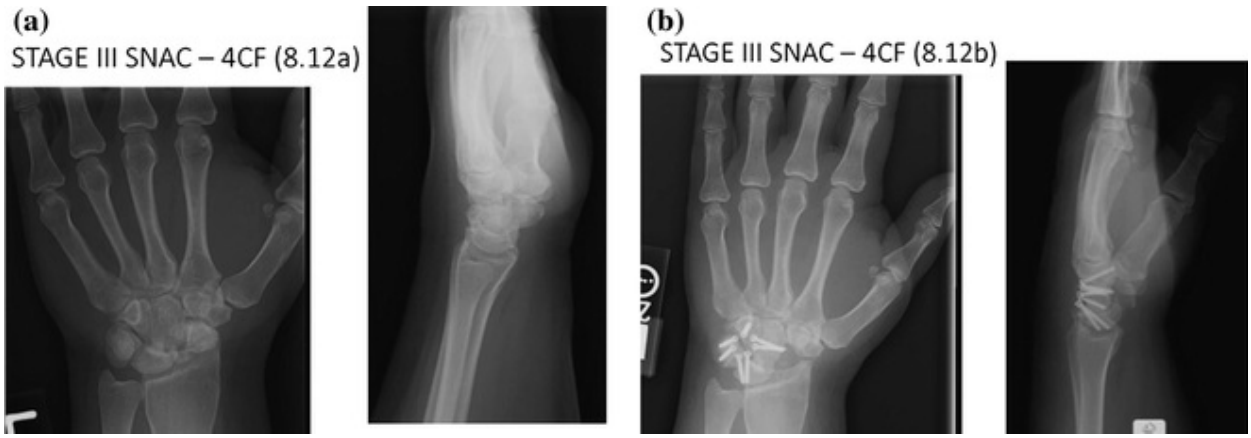



***Fig. 7.11*** **a** Scaphoid nonunion advanced collapse stage III arthritic changes. **b** Treated with scaphoid excision and four corner fusion

The degenerative involvement of the radiolunate joint or presence of pancarpal arthritis in Stage IV is an indication for wrist arthrodesis. It should be noted that this is pain alleviating salvage procedure with significant impact on wrist function and the decision to proceed should be based on a collaborative discussion between the surgeon and patient.

### 7.5.5 Scaphocapitate Fracture Syndrome

The capitate is centered in the carpus where it is well protected and is a rare site of carpal fracture. In fact, capitate fractures account for only 1–2% of all carpal fractures. A capitate fracture may occur in isolation, but more commonly is part of a trans-scaphoid perilunate fracture dislocation. Termed by Fenton as scaphocapitate syndrome, it will result in a capitate nonunion if not correctly diagnosed and adequately addressed [65, 152].

With wrist hyperextension and an axial load resulting in fracture of the scaphoid, the capitate is able to hyperextend. Impaction of the neck of the capitate on the dorsal ridge of the distal radius produces a transversely oriented fracture of the capitate neck. As the wrist returns to neutral position, shortening of the carpus prevents reduction of the proximal fracture fragment. With further wrist flexion, the distal pole of the capitate fragment exerts a flexion force on the proximal fragment, causing the unstable proximal fragment to rotate 180°. The final product is a malrotated fracture, in which the articular surface of the proximal pole faces distally in the fracture site [65, 152]. Clinical diagnosis can be challenging, as initial PA, lateral, and oblique radiographs may have the appearance of a reduced capitate. As a result, these injuries may be missed initially and only diagnosed after fracture reabsorption occurs. Consequently, some surgeons recommend a low threshold to obtain a CT scan for more definitive evaluation of capitate irregularities in the setting of a greater arc injury [153, 154].

The intraosseous blood supply of the capitate is similar to the scaphoid, as dorsal and palmar blood vessels in the head provide vascular supply to the proximal pole by retrograde flow. Also, like the scaphoid, the proximal pole of the capitate is almost entirely intra-articular and the head is nearly completely covered by articular cartilage. The tenuous blood supply from the head renders the capitate vulnerable to osteonecrosis [155]. This is further complicated by the ligamentous instability of a perilunate dislocation and resulting fracture instability.

If recognized acutely, these injuries should be treated with open reduction and internal fixation of the capitate at the same time as definitive management of concomitant injuries including scaphoid fracture and perilunate dislocation. The capitate can be approached through a standard dorsal approach between the third and fourth extensor compartments. The dorsal capsule should be incised though the dorsal capsule splitting approach

described earlier in the chapter, with preservation and later repair of the DRC and DIC ligaments to the triquetrum. There will often be a dorsal capsular tear present in the setting of a perilunate dislocation, and this should be incorporated into the capsular approach whenever possible. Flexion of the wrist facilitates access to the head of the capitate. The fracture is reduced and commonly stabilized with 2 headless screws. Literature on scaphocapitate syndrome consists entirely of small case series and case reports, the largest of which consists of three patients [153, 154, 156–160]. The consensus of these reports is that in the absence of osteonecrosis, both acute and delayed open reduction and internal fixation of these injuries result in good short-term functional outcomes.

Treatment of capitate nonunion, especially with osteonecrosis, is less straightforward. In the setting of delayed presentation, diagnosis, or established nonunion, an MRI is useful to evaluate for osteonecrosis of the proximal pole. The goal of the treatment in capitate nonunion is not only fracture healing, but also re-establishment of capitate height [161]. Failure to do so will result in overload of the scaphotrapezialtrapezoid and triquetral hamate joints and resulting early degenerative changes. A corticocancellous bone graft may be necessary to correct capitate shortening. When significant carpal degenerative changes have developed, limited carpal fusion may be required. Prior to collapse of the proximal pole, attempted restoration of blood flow with vascularized bone grafts has been described, but no long-term outcomes have been published [155]. After collapse of the proximal pole, the problem is best managed by carpal fusion [65, 155].

### 7.5.6 Hook of the Hamate

Hamate fractures are uncommon injuries, accounting for only 2% of all carpal fractures [65]. Fractures of the hook of the hamate occur more frequently than hamate body fractures. Hook of the hamate fractures are more common in athletes than the general population, especially those involved in racquet sports, such as golf, tennis, and baseball. Presentation following injury may be delayed, and without acute immobilization, the risk of nonunion is increased.

The anatomy of the hook of the hamate both places it at risk of injury and contributes to fracture displacement and the potential for nonunion. The hook protrudes volarly from the hamate base into the hypothenar eminence where direct compression from the handle of a club or bat can lead to fracture. The

hook is the origin of the flexor digiti minimi, opponens digiti minimi, hypothenar muscles, and pisohamate ligaments and the distal insertion point of the transverse carpal ligament. Shear forces from each of these muscles, as well as the adjacent flexor tendons of the ulnar digits, can all contribute to fracture displacement. Vascular supply to the hamate has been described by Failla and suggests why fractures of the tip have an increased risk of osteonecrosis compared to the body [162, 163]. The interosseus blood supply to the body of the hamate enters the radial base through a combination of three vascular pedicles, whereas the hook is supplied by a nutrient vessel that originates from the ulnar artery and enters at the tip. In Failla's study, the nutrient vessel to the hook of the hamate was only present in 71% of specimens. Further, minimal collateral flow exists between the nutrient arteries of the base and hook. The incidence of limited blood flow to the tip likely contributes to the risk osteonecrosis.

Initial clinical presentation usually consists of chronic aching pain at the base of hypothenar eminence. The hook of the hamate forms the lateral border of Guyon's canal, which serves to transport the ulnar nerve and artery into the palm. Patients will also frequently complain of ulnar nerve paresthesias in the ring and small finger, as well as a loss of grip strength and exacerbation of pain with grip activities. Some will describe a specific injury, with initial acute pain changing to a dull ache. Many will describe an inability to return to sports activities or decrease in performance, as a result of symptoms. Tenderness to palpation will typically be present over the hook of the hamate, which is distal and radial to the pisiform. Given the close proximity of the ring and small flexor tendons to the hook, pain is often worsened with the hook of the hamate pull test [164]. Resisted flexion of the ring and small finger tendons is applied. Pain is aggravated with resisted finger flexion in wrist ulnar deviation and improved with radial deviation. Infrequently, a chronic fracture will be identified only after rupture of the profundus or superficialis tendons to ring or small fingers.

The injury is often difficult to identify on standard PA, lateral, and oblique hand or wrist radiographs. The PA radiographs may provide some clues to an injury. These include sclerosis of the hamate in the area of the hook and absence of the cortical ring sign in the distal, radial corner of the hamate, which represents the hook radiographically. With suspected injury based on clinical history, the carpal tunnel view, supinated oblique view, and lateral view projected on the first webspace with the thumb abducted have all

been identified as tools to better visualize the hamate [65]. The supinated oblique has the highest sensitivity in clinical studies [165]. When the diagnosis remains unclear, a CT scan is indicated [165, 166]. CT is the most effective of all modalities in diagnosing fracture, with a sensitivity of 100% and specificity and accuracy both greater than 90%. CT is also useful in ruling out a bipartite hamate, an anatomic variant present in a minority of patients.

When diagnosed acutely, nondisplaced fractures of the hook are commonly treated with casting and immobilization. Studies have suggested that delayed presentation of injury leads to poor results with conservative management [167]. In the setting of an established nonunion, healing rates are unacceptably low with cast immobilization alone. Case reports have reported successful healing of a nonunion with cast immobilization and use of low-intensity pulsed ultrasound [168]. As discussed earlier, patients with untreated nonunion are at risk of flexor tendon rupture. This has lead to a trend toward surgical treatment of symptomatic nonunions. Historically, excision of the nonunited fracture fragment has been recommended. This is particularly attractive in athletes, as it may offer a more rapid return to play in comparison with other treatment methods. There is concern that excision may result in a negative effect on flexor tendon function, as the hook functions as a fulcrum for the flexor tendons to the small finger. Biomechanical studies have demonstrated that hook excision results in decreased flexor tendon force, with worsening of the effect as the wrist moves into a position of extension and ulnar deviation [169]. As a result, some authors have proposed attempted open reduction and internal fixation with hook plates or compression screws. Attempted fixation with or without bone grafting has only been studied in small series or single case reports in the literature. Despite adequate healing rates, no clinical benefit has been demonstrated from fixation over fragment excision [169].

The surgical approach to the hook of the hamate is similar to the one used for a decompression of Guyon’s canal. A curvilinear incision is directed over the fragment. Care should be taken to not cross over the proximal palmar flexion crease, in order to avoid scar contracture. The antebrachial fascia of the palm is divided, and the ulnar nerve and artery are identified proximally and followed into Guyon’s canal. The transverse carpal ligament, which attaches to the tip of the hook and comprises the floor of Guyon’s canal, is released from the insertion point on the hook of the hamate. The ulnar artery

and nerve should then be carefully mobilized from around the base of the hook. The deep motor branch to the nerve branches from the dorsal ulnar aspect of the ulnar nerve, passing deep to the flexor digiti minimi. The motor branch frequently lies directly adjacent to a fracture located at the base of the hook. Once the artery and nerve are mobilized and safely retracted without tension, the fracture is subperiosteally excised from the body of the hamate. The remaining periosteum can be closed over the fracture site, or the fracture surface can be smooth with a ronguer to prevent further irritation of the ulnar nerve and flexor tendons.

As mentioned previously, return to play and activities is faster after fragment excision than operative fixation or closed management. Healing of the surgical incision and resolution of pain with impact activities are often determine the time of return to play. In the largest, study of baseball players to date, average return to play after excision was 5.7 weeks [170]. Scar sensitivity is a common complication, and postoperative physical therapy focuses on scar manipulation and desensitization. Athletes will often wear specially padded gloves with initial return to play.

## 7.6 Metacarpal and Phalangeal Nonunions

Nonunion is an uncommon complication after fractures of the metacarpals and phalanges [171]. The risk of nonunion is increased when other complicating factors are involved such as significant soft tissue injury, neurovascular insult, infection, or bone loss. Inadequate fixation, failure of proper reduction and maintenance of reduction, and excessive motion through fracture fragments can prevent fractures from uniting [172].

Recognizing the presence of a nonunion is the first step in treating the complication. By definition, nonunion or delayed union is a fracture without clinical or radiographic signs of healing by 4 months after injury or failure of bony union by 6 months post-injury [171, 172]. The key to this principle is understanding that radiographic evidence of union often lags behind clinical findings and should not be used as the sole indicator of fracture healing. Once a nonunion or delayed union is identified, the causal factors should be explored. Signs and symptoms of infection must be closely evaluated. Bone loss with segmental defects along the metacarpal or phalanx, a dysvascular wound bed with devitalized bone, or significant crush injury with insufficient soft tissue coverage, is all features of the mechanism of injury that hinder

union. However, it is often a failure of fixation that leads nonunion. The use of K wires alone in an unstable fracture pattern or failure of hardware can lead to instability that prohibits healing across fracture lines.

As with other nonunions and delayed unions, classifying the conditions as hypertrophic or atrophic helps direct treatment. Hypertrophic nonunion s of the metacarpals and phalanges are relatively rare, and most are instead atrophic [173]. Hypertrophic nonunions are a result of inadequate immobilization of fracture fragments resulting in excessive relative motion of the fracture fragments, all in the setting of adequate blood supply and a healthy environment for formation of callus. This results in a robust osteoblastic response without bridging fracture lines. More frequently, however, nonunions of the metacarpals and phalanges are atrophic in nature [173]. When an atrophic nonunion is seen in the hand, careful workup is indicated to define the etiology. Fracture biology is compromised in this scenario from factors such as infection, neurovascular compromise, soft tissue stripping or loss, general medical conditions of metabolic disarray.

### 7.6.1 Treatment

Nonoperative treatment of nonunion is indicated in certain cases of significant soft tissue or nerve damage. When grossly contaminated crush injuries or other mechanisms of severe fracture and soft tissue compromise arise, a dysvascular digit often results. Attempted fixation and coverage of the fracture are rarely indicated, and the digit often becomes a hindrance to the overall function of the hand if retained. Consideration should be given to arthrodesis or amputation in these scenarios, especially when the zone of injury is more distal along the ray or involving the lesser digits.

Hypertrophic nonunion s of the metacarpals and phalanges are treated the same way as those involving other bones in the body. The etiology of this type of nonunion is inadequate stability of a fracture which inhibits the formation of bridging callus. Radiographic evidence of callus in the area of the fracture exists, but the fracture line remains clearly visible. The biology of the fracture environment however is sufficient for bone formation and simply requires a more conducive construct for healing. In this setting, treatment consists of increasing stability and compression through the fracture fragments to allow for bone healing. This is often achieved by compression screw fixation if fracture morphology is amenable, followed by rigid neutralization plating, or rigid plating alone.

Atrophic nonunion s requires careful evaluation of contributing factors such as metabolic disarray, poor nutrition, infection, gross instability, or failure to follow prescribed activity restrictions during the initial post-injury/operative period. Furthermore, segmental bone defects or devitalized bone at the fracture site are significant inhibitors of fracture union. After these factors are addressed, stable fixation with a plate-and-screw construct augmented by bone graft to fill any bony deficiency will maximize the odds of functional union [174]. Fracture edges should be debrided, and any signs of fibrous bridging tissue or interposed avascular bone should be removed prior to application of the new construct. Importantly, however, all patients must be counseled in regard to the high likelihood of residual stiffness after this procedure. Surgeons should greatly consider soft tissue releases including tenocapsulolysis at the time of rigid fixation in order to maximize the odds of a successful outcome and to facilitate early motion for guided therapy regimens [175, 176]. Splinting to avoid contracture and appropriate edema control and analgesia to allow for the early phases of therapy is vital to reaching a good functional outcome.

**Author's Preferred bone graft algorithm**: The type of bone graft used or harvested as well as allograft versus autograft is determined by the type of nonunion as well as location of the nonunion (Table 7.1). For example, hypertrophic nonunions may be able to be treated without graft or with limited allograft, such as demineralized bone matrix. However, it is our opinion that nonunions associated with segmental defects or voids or atrophic nonunions are best treated with autograft.

***Table 7.1*** Bone graft algorithm

| Location nonunion | Avascular necrosis (yes/no) | Graft type |
|---|---|---|
| Scaphoid | Yes | 1,2 ICSRA, volar vascularized graft |
| | No | Volar distal radius, olecranon, iliac crest |
| Other carpal | Yes | 5-4 ECA, 2nd metacarpal artery |
| | No | Olecranon, iliac crest |
| Distal radius (small defect) | – | Olecranon, iliac crest cancellous |
| Distal radius (large defect) | – | Iliac crest tricortical graft |

*ICSRA* intercompartmental supraretinacular artery
*ECA* extracompartmental artery

# References


1. Fernandez DL, Ring D, Jupiter JB. Surgical management of delayed union and nonunion of distal radius fractures. J Hand Surg Am. 2001;26(2):201–9.
[PubMed]

2. Segalman KA, Clark GL. Un-united fractures of the distal radius: a report of 12 cases. J Hand Surg Am. 1998;23(5):914–9.
[PubMed]

3. Wolfe SW. Distal radius fractures. In: Wolfe SW, Pederson WC, Hotchkiss RN, Kozin SH, editors. Green's operative hand surgery. 6th ed. Philadelphia: Elsevier; 2010. p. 561–638.

4. McKee MD, Waddell JP, Yoo D, Richards RR. Nonunion of distal radial fractures associated with distal ulnar shaft fractures: a report of four cases. J Orthop Trauma. 1997;11(1):49–53.
[PubMed]

5. Prommersberger KJ, Fernandez DL, Ring D, Jupiter JB, Lanz UB. Open reduction and internal fixation of un-united fractures of the distal radius: does the size of the distal fragment affect the result? Chir Main. 2002;21(2):113–23.
[PubMed]

6. Graham TJ. Surgical correction of malunited fractures of the distal radius. J Am Acad Orthop Surg. 1997;5(5):270–81.
[PubMed]

7. Ring D, Prommersberger KJ, Gonzalez del Pino J, Capomassi M, Slullitel M, Jupiter JB. Corrective osteotomy for intra-articular malunion of the distal part of the radius. J Bone Joint Surg Am. 2005;87(7):1503–9.
[PubMed]

8. Rikli DA, Regazzoni P. Fractures of the distal end of the radius treated by internal fixation and early function. A preliminary report of 20 cases. J Bone Joint Surg Br. 1996;78(4):588–92.
[PubMed]

9. Leslie BM, Medoff RJ. Fracture specific fixation of distal radius fractures. Tech Orthop. 2000;15:336–52.

10. Medoff RJ. Essential radiographic evaluation for distal radius fractures. Hand Clin. 2005;21(3):279–88.
[PubMed]

11. Melone CP Jr. Articular fractures of the distal radius. Orthop Clin North Am. 1984;15(2):217–36.
[PubMed]

12. Hak DJ, Fitzpatrick D, Bishop JA, Marsh JL, Tilp S, Schnettler R, Simpson H, Alt V. Delayed union and nonunions: epidemiology, clinical issues, and financial aspects. Injury. 2014;45(Suppl 2):S3–7.
[PubMed]

13. Bushnell BD, Bynum DK. Malunion of the distal radius. J Am Acad Orthop Surg. 2007;15(1):27–

40.

14. Adams BD, Grosland NM, Murphy DM, McCullough M. Impact of impaired wrist motion on hand and upper-extremity performance. J Hand Surg Am. 2003;28(6):898–903. [PubMed]

15. Prommersberger KJ, Fernandez DL, Ring D, Jupiter JB, Lanz UB. Open reduction and internal fixation of un-united fractures of the distal radius: does the size of the distal fragment affect the result? Chir Main. 2002;21(2):113–23. [PubMed]

16. Ring D. Nonunion of the distal radius. Hand Clin. 2005;21(3):443–7. [PubMed]

17. Gwathmey FW Jr, Brunton LM, Pensy RA, Chhabra AB. Volar plate osteosynthesis of distal radius fractures with concurrent prophylactic carpal tunnel release using a hybrid flexor carpi radialis approach. J Hand Surg Am. 2010;35(7):1082–8. [PubMed]

18. Ring D, Jupiter JB. Nonunion of the distal radius. Tech Hand Up Extrem Surg. 2002;6(1):6–9. [PubMed]

19. Harness NG, Jupiter JB, Orbay JL, Raskin KB, Fernandez DL. Loss of fixation of the volar lunate facet fragment in fractures of the distal part of the radius. J Bone Joint Surg Am. 2004;86-A(9):1900–8.

20. Jupiter JB, Marent-Huber M; LCP Study Group. Operative management of distal radial fractures with 2.4-millimeter locking plates. A multicenter prospective case series. J Bone Joint Surg Am. 2009;91(1):55–65.

21. Jupiter JB, Fernandez DL. Complications following distal radial fractures. J Bone Joint Surg Am. 2001;83:1244–65.

22. Bakker AJ, Shin AY. Fragment-specific volar hook plate for volar marginal rim fractures. Tech Hand Up Extrem Surg. 2014;18(1):56–60. [PubMed]

23. Mithani SK, Srinivasan RC, Kamal R, Richard MJ, Leversedge FJ, Ruch DS. Salvage of distal radius nonunion with a dorsal spanning distraction plate. J Hand Surg Am. 2014;39(5):981–4. [PubMed]

24. Hanel DP, Ruhlman SD, Katolik LI, Allan CH. Complications associated with distraction plate fixation of wrist fractures. Hand Clin. 2010;26(2):237–43. [PubMed]

25. Stucken C, Olszewski DC, Creevy WR, Murakami AM, Tornetta P. Preoperative diagnosis of infection in patients with nonunions. J Bone Joint Surg Am. 2013;95(15):1409–12. [PubMed]

26. Jones AL, Bucholz RW, Bosse MJ, Mirza SK, Lyon TR, Webb LX, et al; BMP-2 Evaluation in Surgery for Tibial Trauma-Allgraft (BESTT-ALL) Study Group. Recombinant human BMP-2

and allograft compared with autogenous bone graft for reconstruction of diaphyseal tibial fractures with cortical defects. A randomized, controlled trial. J Bone Joint Surg Am. 2006;88(7):1431–41.

27. Aro HT, Govender S, Patel AD, Hernigou P, Perera de Gregorio A, Popescu GI, Golden JD, Christensen J, Valentin A. Recombinant human bone morphogenetic protein-2: a randomized trial in open tibial fractures treated with reamed nail fixation. J Bone Joint Surg Am. 2011;93(9):801–8.
[PubMed]

28. Adams BD. Distal radioulnar joint. In: Wolfe SW, Pederson WC, Hotchkiss RN, Kozin SH, editors. Green's operative hand surgery. 6th ed. Philadelphia: Elsevier; 2010. p. 523–60.

29. May MM, Lawton JN, Blazar PE. Ulnar styloid fractures associated with distal radius fractures: incidence and implications for distal radioulnar joint instability. J Hand Surg Am. 2002;27(6):965–71.
[PubMed]

30. Ekenstam F. Anatomy of the distal radioulnar joint. Clin Orthop Relat Res. 1992;(275):14–8.

31. Adams BD, Berger RA. An anatomic reconstruction of the distal radioulnar ligaments for posttraumatic distal radioulnar joint instability. J Hand Surg Am. 2002;27(2):243–51.
[PubMed]

32. Stuart PR, Berger RA, Linscheid RL, An KN. The dorsopalmar stability of the distal radioulnar joint. J Hand Surg Am. 2000;25(4):689–99.
[PubMed]

33. Tolat AR, Stanley JK, Trail IA. A cadaveric study of the anatomy and stability of the distal radioulnar joint in the coronal and transverse planes. J Hand Surg Br. 1996;21(5):587–94.
[PubMed]

34. Wallwork NA, Bain GI. Sigmoid notch osteoplasty for the chronic volar instability of the distal radioulnar joint: a case report. J Hand Surg Am. 2001;26(3):454–9.
[PubMed]

35. Sagerman SD, Zogby RG, Palmer AK, Werner FW, Fortino MD. Relative articular inclination of the distal radioulnar joint: a radiographic study. J Hand Surg Am. 1995;20(4):597–601.
[PubMed]

36. Waitayawinyu T, McCallister WV, Nemechek NM, Trumble TE. Scaphoid nonunion. J Am Acad Orthop Surg. 2007;15(5):308–20.

37. Palmer AK, Werner FW. The triangular fibrocartilage complex of the wrist—anatomy and function. J Hand Surg Am. 1981;6(2):153–62.
[PubMed]

38. Gofton WT, Gordon KD, Dunning CE, Johnson JA, King GJ. Soft-tissue stabilizers of the distal radioulnar joint: an in vitro kinematic study. J Hand Surg Am. 2004;29(3):423–31.
[PubMed]

39. Adams BD, Lawler E. Chronic instability of the distal radioulnar joint. J Am Acad Orthop Surg.

2007;15(9):571–5.
[PubMed]

40. Ward LD, Ambrose CG, Masson MV, Levaro F. The role of the distal radioulnar ligaments, interosseus membrane, and joint capsule in distal radioulnar joint stability. J Hand Surg Am. 2000;25(2):341–51.
[PubMed]

41. Adams BD, Holley KA. Strains in the articular disk of the triangular fibrocartilage complex: a biomechanical study. J Hand Surg Am. 1993;18(5):919–25.
[PubMed]

42. Schuind F, An KN, Berglund L, Rey R, Cooney WP III, Linscheid RL, Chao EY. The distal radioulnar ligaments: a biomechanical study. J Hand Surg Am. 1991;16(6):1106–14.
[PubMed]

43. Ekenstam FAF, Hagert CG. Anatomical studies of the geometry and stability of the distal radioulnar joint. Scand J Plast Reconstr Surg. 1985;19(1):17–25.

44. Chidgey LK, Dell PC, Bittar ES, Spanier SS. Histologic anatomy of the triangular fibrocartilage. J Hand Surg Am. 1991;16(6):1084–100.
[PubMed]

45. Zimmerman RM, Kim JM, Jupiter JB. Arthritis of the distal radioulnar joint: from Darrach to total joint arthroplasty. J Am Acad Orthop Surg. 2012;20(10):623–32.
[PubMed]

46. Epner RA, Bowers WH, Guilford WB. Ulnar variance—the effect of wrist positioning and roentgen filming technique. J Hand Surg Am. 1982;7(3):298–305.
[PubMed]

47. Friedman SL, Palmer AK, Short WH, Levinsohn EM, Halperin LS. The change in ulnar variance with grip. J Hand Surg Am. 1993;18(4):713–6.
[PubMed]

48. Tomaino MM. The importance of the pronated grip X-ray view in evaluating ulnar variance. J Hand Surg Am. 2000;25(2):352–7.
[PubMed]

49. Mino DE, Palmer AK, Levinsohn EM. Radiography and computerized tomography in the diagnosis of incongruity of the distal radio-ulnar joint: a prospective study. J Bone Joint Surg Am. 1985;67(2):247–52.
[PubMed]

50. Scheker LR, Belliappa PP, Acosta R, German DS. Reconstruction of the dorsal ligament of the triangular fibrocartilage complex. J Hand Surg Br. 1994;19(3):310–8.
[PubMed]

51. Lo IK, MacDermid JC, Bennett JD, Bogoch E, King GJ. The radioulnar ratio: a new method of quantifying distal radioulnar joint subluxation. J Hand Surg Am. 2001;26(2):236–43.
[PubMed]

52. Wechsler RJ, Wehbe MA, Rifkin MD, Edeiken J, Branch HM. Computed tomography diagnosis of radioulnar subluxation. Skeletal Radiol. 1987;16(1):1–5.
[PubMed]

53. Amrami KK, Moran SL, Berger RA, Ehman EC, Felmlee JP. Imaging the distal radioulnar joint. Hand Clin. 2010;26(4):467–75.
[PubMed]

54. Millard GM, Budoff JE, Paravic V, Noble PC. Functional bracing for distal radioulnar joint instability. J Hand Surg. 2002;27(6):972–7.

55. Frykman G. Fracture of the distal radius including sequelae—shoulder-hand-finger syndrome, disturbance in the distal radio-ulnar joint and impairment of nerve function. A clinical and experimental study. Acta Orthop Scand. 1967:Suppl 108:3+.

56. Geissler WB, Fernandez DL, Lamey DM. Distal radioulnar joint injuries associated with fractures of the distal radius. Clin Orthop Relat Res. 1996;327:135–46.

57. Hauck RM, Skahen J III, Palmer AK. Classification and treatment of ulnar styloid nonunion. J Hand Surg Am. 1996;21(3):418–22.
[PubMed]

58. Peterson MS, Adams BD. Biomechanical evaluation of distal radioulnar reconstructions. J Hand Surg. 1993;18(2):328–34.

59. Hui FC, Linscheid RL. Ulnotriquetral augmentation tenodesis: a reconstructive procedure for dorsal subluxation of the distal radioulnar joint. J Hand Surg Am. 1982;7(3):230–6.
[PubMed]

60. Boyes JH. Surgical repair of joints. In: Bunnell S, editor. Bunnell's surgery of the hand. Philadelphia: Lippincott; 1970. p. 294–313.

61. Johnston Jones K, Sanders WE. Posttraumatic radioulnar instability: treatment by anatomic reconstruction of volar and radioulnar ligaments. Orthop Trans. 1995–1996;19:832.

62. Kihara H, Short WH, Werner FW, Fortino MD, Palmer AK. The stabilizing mechanism of the distal radioulnar joint during pronation and supination. J Hand Surg Am. 1995;20(6):930–6.
[PubMed]

63. Wallwork NA, Bain GI. Sigmoid notch osteoplasty for chronic volar instability of the distal radioulnar joint: a case report. J Hand Surg Am. 2001;26(3):454–9.
[PubMed]

64. Tham SK, Bain GI. Sigmoid notch osseous reconstruction. Tech Hand Up Extrem Surg. 2007;11(1):93–7.
[PubMed]

65. Geissler WB, Slade JF. Fractures of the carpal bones. In: Wolfe SW, Pederson WC, Hotchkiss RN, Kozin SH, editors. Green's operative hand surgery. 6th ed. Philadelphia: Elsevier; 2010. p. 639–707.

66. Wolf JM, Dawson L, Mountcastle SB, Owens BD. The incidence of scaphoid fracture in a

military population. Injury. 2009;40(12):1316–9.
[PubMed]

67. Doornberg JN, Buijze GA, Ham SJ, Ring D, Bhandari M, Poolman RW. Nonoperative treatment for acute scaphoid fractures: a systematic review and meta-analysis of randomized controlled trials. J Trauma. 2011;71(4):1073–81.
[PubMed]

68. Singh HP, Taub N, Dias JJ. Management of displaced fractures of the waist of the scaphoid: meta-analyses of comparative studies. Injury. 2012;43(6):933–9.
[PubMed]

69. Yin ZG, Zhang JB, Kan SL, Wang P. Treatment of acute scaphoid fractures: systematic review and meta-analysis. Clin Orthop Relat Res. 2007;460:142–51.
[PubMed]

70. Symes TH, Stothard J. A systematic review of the treatment of acute fractures of the scaphoid. J Hand Surg Eur. 2011;36(9):802–10.

71. Suh N, Benson EC, Faber KJ, Macdermid J, Grewal R. Treatment of acute scaphoid fractures: a systematic review and meta-analysis. Hand (N Y). 2010;5(4):345–53.

72. Ibrahim T, Qureshi A, Sutton AJ, Dias JJ. Surgical versus nonsurgical treatment of acute minimally displaced and undisplaced scaphoid waist fractures: Pairwise and network meta-analyses of randomized controlled trials. J Hand Surg Am. 2011;36(11):1759–68, e1.

73. Heinzelmann AD, Archer G, Bindra RR. Anthropometry of the human scaphoid. J Hand Surg Am. 2007;32(7):1005–8.
[PubMed]

74. Pichler W, Windisch G, Schaffler G, Heidari N, Dorr K, Grechenig W. Computer-assisted 3-dimensional anthropometry of the scaphoid. Orthopedics. 2010;33(2):85–8.
[PubMed]

75. Gelberman RH, Menon J. The vascularity of the scaphoid bone. J Hand Surg Am. 1980;5(5):508–13.
[PubMed]

76. Weber ER, Chao EY. An experimental approach to the mechanism of scaphoid waist fractures. J Hand Surg Am. 1978;3(2):142–8.
[PubMed]

77. Horii E, Nakamura R, Watanabe K, Tsunoda K. Scaphoid fracture as a “puncher’s fracture”. J Orthop Trauma. 1994;8(2):107–10.
[PubMed]

78. Mack GR, Bosse MJ, Gelberman RH, Yu E. The natural history of scaphoid non-union. J Bone Joint Surg Am. 1984;66(4):504–9.
[PubMed]

79. Little CP, Burston BJ, Hopkinson-Woolley J, Burge P. Failure of surgery for scaphoid non-union is associated with smoking. J Hand Surg Br. 2006;31(3):252–5.

[PubMed]

80. Nolte PA, van der Krans A, Patka P, Janssen IM, Ryaby JP, Albers GH. Low-intensity pulsed ultrasound in the treatment of nonunions. J Trauma. 2001;51(4):693–702; discussion 702-3.

81. Trumble TE, Clarke T, Kreder HJ. Non-union of the scaphoid: treatment with cannulated screws compared with treatment with Herbert screws. J Bone Joint Surg Am. 1996;78(12):1829–37. [PubMed]

82. Jupiter JB, Shin AY, Trumble TE, Fernandez DL. Traumatic and reconstructive problems of the scaphoid. Instr Course Lect. 2001;50:105–22. [PubMed]

83. Trumble TE, Salas P, Barthel T, Robert KQ III. Management of scaphoid nonunions. J Am Acad Orthop Surg. 2003;11(6):380–91. [PubMed]

84. Buijze GA, Wijffels MM, Guitton TG, Grewal R, van Dijk CN, Ring D; Science of Variation Group. Interobserver reliability of computed tomography to diagnose scaphoid waist fracture union. J Hand Surg Am. 2012;37(2):250–4.

85. Moon ES, Dy CJ, Derman P, Vance MC, Carlson MG. Management of nonunion following surgical management of scaphoid fractures: current concepts. J Am Acad Orthop Surg. 2013;21(9):548–57. [PubMed]

86. Amadio PC, Berquist TH, Smith DK, Ilstrup DM, Cooney WP III, Linscheid RL. Scaphoid malunion. J Hand Surg Am. 1989;14(4):679–87. [PubMed]

87. Bain GI, Bennett JD, Richards RS, Slethaug GP, Roth JH. Longitudinal computed tomography of the scaphoid: a new technique. Skeletal Radiol. 1995;24(4):271–3. [PubMed]

88. Bain GI, Bennett JD, MacDermid JC, Slethaug GP, Richards RS, Roth JH. Measurement of the scaphoid humpback deformity using longitudinal computed tomography: intra- and interobserver variability using various measurement techniques. J Hand Surg Am. 1998;23(1):76–81. [PubMed]

89. Smith ML, Bain GI, Chabrel N, Turner P, Carter C, Field J. Using computed tomography to assist with diagnosis of avascular necrosis complicating chronic scaphoid nonunion. J Hand Surg Am. 2009;34(6):1037–43. [PubMed]

90. Morgan WJ, Breen TF, Coumas JM, Schulz LA. Role of magnetic resonance imaging in assessing factors affecting healing in scaphoid nonunions. Clin Orthop Relat Res. 1997;336:240–6.

91. Perlik PC, Guilford WB. Magnetic resonance imaging to assess vascularity of scaphoid nonunions. J Hand Surg Am. 1991;16(3):479–84. [PubMed]

92.

Sakuma M, Nakamura R, Imaeda T. Analysis of proximal fragment sclerosis and surgical outcome of scaphoid non-union by magnetic resonance imaging. J Hand Surg Br. 1995;20(2):201–5.
[PubMed]

93. Trumble TE. Avascular necrosis after scaphoid fracture: a correlation of magnetic resonance imaging and histology. J Hand Surg Am. 1990;15(4):557–64.
[PubMed]

94. Roolker W, Ritt MJ, Bos KE. Spontaneous healing of a non-union of the scaphoid. J Hand Surg Br. 1998;23(1):86–7.
[PubMed]

95. Adams BD, Frykman GK, Taleisnik J. Treatment of scaphoid nonunion with casting and pulsed electromagnetic fields: a study continuation. J Hand Surg Am. 1992;17(5):910–4.
[PubMed]

96. Rettig AC, Weidenbener EJ, Gloyeske R. Alternative management of midthird scaphoid fractures in the athlete. Am J Sports Med. 1994;22(5):711–4.
[PubMed]

97. Davis EN, Chung KC, Kotsis SV, Lau FH, Vijan S. A cost/utility analysis of open reduction and internal fixation versus cast immobilization for acute nondisplaced mid-waist scaphoid fractures. Plast Reconstr Surg. 2006;117(4):1223–35.
[PubMed]

98. Bunker TD, McNamee PB, Scott TD. The Herbert screw for scaphoid fractures: a multicentre study. J Bone Joint Surg Br. 1987;69(4):631–4.
[PubMed]

99. Hunter JC, Escobedo EM, Wilson AJ, Hanel DP, Zink-Brody GC, Mann FA. MR imaging of clinically suspected scaphoid fractures. AJR Am J Roentgenol. 1997;168(5):1287–93.
[PubMed]

100. Kuhlmann JN, Mimoun M, Boabighi A, Baux S. Vascularized bone graft pedicled on the volar carpal artery for non-union of the scaphoid. J Hand Surg Br. 1987;12(2):203–10.
[PubMed]

101. Sheetz KK, Bishop AT, Berger RA. The arterial blood supply of the distal radius and ulna and its potential use in vascularized pedicled bone grafts. J Hand Surg Am. 1995;20(6):902–14.
[PubMed]

102. Stark A, Brostrom LA, Svartengren G. Surgical treatment of scaphoid non-union: review of the literature and recommendations for treatment. Arch Orthop Trauma Surg. 1989;108(4):203–9.
[PubMed]

103. Korkala OL, Kuokkanen HO, Eerola MS. Compression-staple fixation for fractures, non-unions, and delayed unions of the carpal scaphoid. J Bone Joint Surg Am. 1992;74(3):423–6.
[PubMed]

104. Huene DR, Huene DS. Treatment of nonunions of the scaphoid with the Ender compression blade

plate system. J Hand Surg Am. 1991;16(5):913–22.
[PubMed]

105. Leixnering M, Pezzei C, Weninger P, Mayer M, Bogner R, Lederer S, et al. First experiences with a new adjustable plate for osteosynthesis of scaphoid nonunions. J Trauma. 2011;71(4):933–8.
[PubMed]

106. Reigstad O, Thorkildsen R, Grimsgaard C, Reigstad A, Røkkum M. Is revision bone grafting worthwhile after failed surgery for scaphoid nonunion? Minimum 8 year follow-up of 18 patients. J Hand Surg Eur. 2009;34(6):772–7.

107. Herbert TJ, Fisher WE. Management of the fractured scaphoid using a new bone screw. J Bone Joint Surg Br. 1984;66(1):114–23.
[PubMed]

108. Faucher GK, Golden ML III, Sweeney KR, Hutton WC, Jarrett CD. Comparison of screw trajectory on stability of oblique scaphoid fractures: a mechanical study. J Hand Surg Am. 2014;39(3):430–5.
[PubMed]

109. McCallister WV, Knight J, Kaliappan R, Trumble T. Central placement of the screw in simulated fractures of the scaphoid waist. J Bone Joint Surg Am. 2003;85(1):72–8.
[PubMed]

110. Trumble TE, Gilbert M, Murray LW, Smith J, Rafijah G, McCallister WV. Displaced scaphoid fractures treated with open reduction and internal fixation with a cannulated screw. J Bone Joint Surg Am. 2000;82(5):633–41.
[PubMed]

111. Shah J, Jones WA. Factors affecting the outcome in 50 cases of scaphoid nonunion treated with Herbert screw fixation. J Hand Surg Br. 1998;23(5):680–5.
[PubMed]

112. Slade JF III, Geissler WB, Gutow AP, Merrell GA. Percutaneous internal fixation of selected scaphoid nonunions with an arthroscopically assisted dorsal approach. J Bone Joint Surg Am. 2003;85-A(Suppl 4):20–32.

113. Ikeda K, Osamura N, Tomita K. Percutaneous screw fixation without bone graft for cystic-type scaphoid fractures. J Trauma. 2008;65(6):1453–8.
[PubMed]

114. Slade JF III, Gillon T. Retrospective review of 234 scaphoid fractures and nonunions treated with arthroscopy for union and complications. Scand J Surg. 2008;97(4):280–9.
[PubMed]

115. Russe O. Fractures of the carpal navicular. Diagnosis, non-operative treatment, and operative treatment. J Bone Joint Surg Am. 1960;42-A:759–68.

116. Green DP. The effect of avascular necrosis on Russe bone grafting for scaphoid nonunion. J Hand Surg Am. 1985;10(5):597–605.
[PubMed]

117. Dacho A, Germann G, Sauerbier M. The reconstruction of scaphoid pseudoarthroses with the operation of Matti-Russe. A retrospective follow-up analysis of 84 patients. Unfallchirurg. 2004;107(5):388–96 (article in German).

118. Barton NJ. Experience with scaphoid grafting. J Hand Surg Br. 1997;22(2):153–60. [PubMed]

119. Berger RA, Bishop AT, Bettinger PC. New dorsal capsulotomy for the surgical exposure of the wrist. Ann Plast Surg. 1995;35(1):54–9. [PubMed]

120. Inoue G, Shionoya K, Kuwahata Y. Ununited proximal pole scaphoid fractures. Treatment with a Herbert screw in 16 cases followed for 0.5-8 years. Acta Orthop Scand. 1997;68(2):124–7. [PubMed]

121. Garcia RM, Leversedge FJ, Aldridge JM, Richard MJ, Ruch DS. Scaphoid nonunions treated with 2 headless compression screws and bone grafting. J Hand Surg Am. 2014;39(7):1301–7. [PubMed]

122. Manske PR, McCarthy JA, Strecker WB. Use of the Herbert bone screw for scaphoid nonunions. Orthopedics. 1988;11(12):1653–61. [PubMed]

123. Fernandez DL. Anterior bone grafting and conventional lag screw fixation to treat scaphoid nonunions. J Hand Surg Am. 1990;15(1):140–7. [PubMed]

124. Eggli S, Fernandez DL, Beck T. Unstable scaphoid fracture nonunion: a medium-term study of anterior wedge grafting procedures. J Hand Surg Br. 2002;27(1):36–41. [PubMed]

125. Inoue G, Shionoya K. Herbert screw fixation by limited access for acute fractures of the scaphoid. J Bone Joint Surg Br. 1997;79(3):418–21. [PubMed]

126. Robbins RR, Ridge O, Carter PR. Iliac crest bone grafting and Herbert screw fixation of nonunions of the scaphoid with avascular proximal poles. J Hand Surg Am. 1995;20(5):818–31. [PubMed]

127. Sunagawa T, Bishop AT, Muramatsu K. Role of conventional and vascularized bone grafts in scaphoid nonunion with avascular necrosis: a canine experimental study. J Hand Surg Am. 2000;25(5):849–59. [PubMed]

128. Zaidemberg C, Siebert JW, Angrigiani C. A new vascularized bone graft for scaphoid nonunion. J Hand Surg Am. 1991;16(3):474–8. [PubMed]

129. Chang MA, Bishop AT, Moran SL, Shin AY. The outcomes and complications of 1,2-intercompartmental supraretinacular artery pedicled vascularized bone grafting of scaphoid nonunions. J Hand Surg Am. 2006;31(3):387–96.

[PubMed]

130. Straw RG, Davis TR, Dias JJ. Scaphoid nonunion: treatment with a pedicled vascularized bone graft based on the 1,2 intercompartmental supraretinacular branch of the radial artery. J Hand Surg Br. 2002;27(5):413.
[PubMed]

131. Gras M, Mathoulin C. Vascularized bone graft pedicled on the volar carpal artery from the volar distal radius as primary procedure for scaphoid non-union. Orthop Traumatol Surg Res. 2011;97(8):800–6.
[PubMed]

132. Mathoulin C, Brunelli F. Further experience with the index metacarpal vascularized bone graft. J Hand Surg Br. 1998;23(3):311–7.
[PubMed]

133. Sawaizumi T, Nanno M, Nanbu A, Ito H. Vascularised bone graft from the base of the second metacarpal for refractory nonunion of the scaphoid. J Bone Joint Surg Br. 2004;86(7):1007–12.
[PubMed]

134. Guimberteau JC, Panconi B. Recalcitrant non-union of the scaphoid treated with a vascularized bone graft based on the ulnar artery. J Bone Joint Surg Am. 1990;72(1):88–97.
[PubMed]

135. Jones DB Jr, Moran SL, Bishop AT, Shin AY. Free-vascularized medial femoral condyle bone transfer in the treatment of scaphoid nonunions. Plast Reconstr Surg. 2010;125(4):1176–84.
[PubMed]

136. Arora R, Lutz M, Zimmermann R, Krappinger D, Niederwanger C, Gabl M. Free vascularised iliac bone graft for recalcitrant avascular nonunion of the scaphoid. J Bone Joint Surg Br. 2010;92(2):224–9.
[PubMed]

137. Al-Jabri T, Mannan A, Giannoudis P. The use of the free vascularised bone graft for nonunion of the scaphoid: a systematic review. J Orthop Surg Res. 2014;9:21.
[PubMed][PubMedCentral]

138. Sandow MJ. Costo-osteochondral grafts in the wrist. Tech Hand Up Extrem Surg. 2001;5(3):165–72.
[PubMed]

139. Veitch S, Blake SM, David H. Proximal scaphoid rib graft arthroplasty. J Bone Joint Surg Br. 2007;89(2):196–201.
[PubMed]

140. Tropet Y, Lepage D, Gallinet D, Obert L, Garbuio P, Vichard P. Articular reconstructions by a costochondral grafting (or osteochondral costal grafting). Bull Acad Natl Med. 2006;190(7):1439–56 (article in French).

141. Sunagawa T, Bishop AT, Muramatsu K. Role of conventional and vascularized bone grafts in scaphoid nonunion with avascular necrosis: a canine experimental study. J Hand Surg Am. 2000;25(5):849–59.

[PubMed]

142. Fernandez DL, Eggli S. Non-union of the scaphoid: revascularization of the proximal pole with implantation of a vascular bundle and bone-grafting. J Bone Joint Surg Am. 1995;77(6):883–93. [PubMed]

143. Smith BS, Cooney WP. Revision of failed bone grafting for nonunion of the scaphoid: treatment options and results. Clin Orthop Relat Res. 1996;327:98–109.

144. Hori Y, Tamai S, Okuda H, Sakamoto H, Takita T, Masuhara K. Blood vessel transplantation to bone. J Hand Surg Am. 1979;4(1):23–33. [PubMed]

145. Malerich MM, Clifford J, Eaton B, Eaton R, Littler JW. Distal scaphoid resection arthroplasty for the treatment of degenerative arthritis secondary to scaphoid nonunion. J Hand Surg Am. 1999;24(6):1196–205. [PubMed]

146. Ruch DS, Papadonikolakis A. Resection of the scaphoid distal pole for symptomatic scaphoid nonunion after failed previous surgical treatment. J Hand Surg Am. 2006;31(4):588–93. [PubMed]

147. Wyrick JD, Stern PJ, Kiefhaber TR. Motion-preserving procedures in the treatment of scapholunate advanced collapse wrist: proximal row carpectomy versus four-corner arthrodesis. J Hand Surg Am. 1995;20(6):965–70. [PubMed]

148. Mulford JS, Ceulemans LJ, Nam D, Axelrod TS. Proximal row carpectomy vs four corner fusion for scapholunate (Slac) or scaphoid nonunion advanced collapse (SNAC) wrists: a systematic review of outcomes. J Hand Surg Eur. 2009;34(2):256–63.

149. Skie MC, Gove N, Ciocanel DE, Smith H. Management of non-united four-corner fusions. Hand (N Y). 2007;2(1):34–8.

150. Chung KC, Watt AJ, Kotsis SV. A prospective outcomes study of four-corner wrist arthrodesis using a circular limited wrist fusion plate for stage II scapholunate advanced collapse wrist deformity. Plast Reconstr Surg. 2006;118(2):433–42. [PubMed]

151. Shindle MK, Burton KJ, Weiland AJ, Domb BG, Wolfe SW. Complications of circular plate fixation for four-corner arthrodesis. J Hand Surg Eur. 2007;32(1):50–3.

152. Fenton RL. The naviculo-capitate fracture syndrome. J Bone Joint Surg Am. 1956;38-A(3):681–4.

153. Kim YS, Lee HM, Kim JP. The scaphocapitate fracture syndrome: a case report and literature analysis. Eur J Orthop Surg Traumatol. 2013;23(Suppl 2):S207–12. [PubMed]

154. Hamdi MF. The scaphocapitate fracture syndrome: report of a case and a review of the literature. Musculoskelet Surg. 2012;96(3):223–6.

[PubMed]

155. Vander Grend R, Dell PC, Glowczewskie F, Leslie B, Ruby LK. Intraosseous blood supply of the capitate and its correlation with aseptic necrosis. J Hand Surg Am. 1984;9(5):677–83. [PubMed]

156. Burke NG, Cosgrave CH, O'Neill BJ, Kelly EP. Transstyloid, transscaphoid, transcapitate fracture: a variant of scaphocapitate fractures. BMJ Case Rep. 2014 Mar 31;2014. pii: bcr2014204041.

157. Natera Cisneros L, Lamas Gómez C, Proubasta Renart I, Moya Gómez E. Fenton syndrome. Rev Esp Cir Ortop Traumatol. 2012;56(5):369–73 (article in Spanish).

158. Schliemann B, Langer M, Kösters C, Raschke MJ, Ochman S. Successful delayed surgical treatment of a scaphocapitate fracture. Arch Orthop Trauma Surg. 2011;131(11):1555–9. [PubMed]

159. Arbter D, Piatek S, Wichlas F, Winckler S. The scaphocapitate fracture syndrome (Fenton). Handchir Mikrochir Plast Chir. 2009;41(3):171–4 (article in German).

160. Sawant M, Miller J. Scaphocapitate syndrome in an adolescent. J Hand Surg Am. 2000;25(6):1096–9. [PubMed]

161. Geissler WB. Carpal fractures in athletes. Clin Sports Med. 2001;20(1):167–88. [PubMed]

162. Failla JM. Osteonecrosis associated with nonunion of the hook of the hamate. Orthopedics. 1993;16(2):217–8. [PubMed]

163. Failla JM. Hook of hamate vascularity: vulnerability to osteonecrosis and nonunion. J Hand Surg Am. 1993;18(6):1075–9. [PubMed]

164. Pajares-López M, Hernández-Cortés P, Robles-Molina MJ. Rupture of small finger flexor tendons secondary to asymptomatic nonunion of the hamate hook. Orthopedics. 2011;34(2):142. [PubMed]

165. Andresen R, Radmer S, Scheufler O, Adam C, Bogusch G. Optimization of conventional X-ray images for the detection of hook of hamate fractures. Rontgenpraxis. 2006;56(2):59–65. [PubMed]

166. Andresen R, Radmer S, Sparmann M, Bogusch G, Banzer D. Imaging of hamate bone fractures in conventional X-rays and high-resolution computed tomography. An in vitro study. Invest Radiol. 1999;34(1):46–50. [PubMed]

167. Carroll RE, Lakin JF. Fracture of the hook of the hamate: acute treatment. J Trauma. 1993;34(6):803–5. [PubMed]

168. Sakuma Y, Iwamoto T, Momohara S. Ununited fracture of the hook of hamate treated with low-intensity pulsed ultrasound in an older middle-aged patient. Clin J Sport Med. 2014;24(4):358–9. [PubMed]

169. Demirkan F, Calandruccio JH, Diangelo D. Biomechanical evaluation of flexor tendon function after hamate hook excision. J Hand Surg Am. 2003;28(1):138–43. [PubMed]

170. Bachoura A, Wroblewski A, Jacoby SM, Osterman AL, Culp RW. Hook of hamate fractures in competitive baseball players. Hand (N Y). 2013;8(3):302–7.

171. Green DP. Complications of phalangeal and metacarpal fractures. Hand Clin. 1986;2(2):307–28. [PubMed]

172. Jupiter JB, Koniuch MP, Smith RJ. The management of delayed union and nonunion of the metacarpals and phalanges. J Hand Surg. 1985;10(4):457–66.

173. Ring D. Malunion and nonunion of the metacarpals and phalanges. J Bone Joint Surg Am. 2005;87:1380–8.

174. Smith FL, Rider DL. A study of the healing of one hundred consecutive phalangeal fractures. J Bone Joint Surg. 1935;17:91–109.

175. Weckesser EC. Rotational osteotomy of the metacarpal for overlapping fingers. J Bone Joint Surg. 1965;47(4):751–6. [PubMed]

176. Creighton JJ, Steichen JB. Complications in phalangeal and metacarpal fracture management. Results of extensor tenolysis. Hand Clin. 1994;10(1):111–6. [PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_8

# 8. Acetabular and Pelvic Nonunions

Kyle F. Dickson[1]

(1) Southwest Orthopedic Group, Baylor University, 6560 Fannin St, Suite 1016, Houston, TX 77030, USA

**Kyle F. Dickson**
**Email:** kyledickson99@gmail.com

**Keywords** Nonunion – Acetabulum – Pelvis – Traumatic hip arthritis – Deformity

## 8.1 Nonunion of the Pelvis : Clinical Assessment and Pain

Although pain is not always present in nonunions, it is often the primary reason for a patient to seek medical consultation (Figs. 8.1, 8.2, 8.3 and 8.4). The pain is commonly secondary to instability of the pelvis, or malreduction, and is most frequently located posteriorly in the sacroiliac (SI) region [1]. A nonunion can initially be nondisplaced, but, due to lack of healing, may displace and develop an associated deformity (see Fig. 8.3). Furthermore, a nonunion of one part of the pelvis, especially in osteoporotic patients, can lead to insufficiency fractures in other areas of the pelvis (Figs. 8.4 and 8.5). Posterior pelvic pain associated with malunion often improves after correction of the malunion, although the reason for this is less apparent than with correction of nonunions [2, 3]. Some residual chronic pain often occurs. In an acute injury, instability is readily apparent on physical examination of the pelvis. This is more difficult to appreciate in chronic nonunions. In these situations, the physician’s hands are placed on each of the anterior superior

iliac spine (ASIS) and the pelvis is rocked from side to side. Subtle motion of the pelvis can be detected in this manner or pain is induced at the nonunion site. In these chronic cases, radiographic single-leg stance anteroposterior (AP) views are sometimes more helpful to show instability, as will be reviewed later.

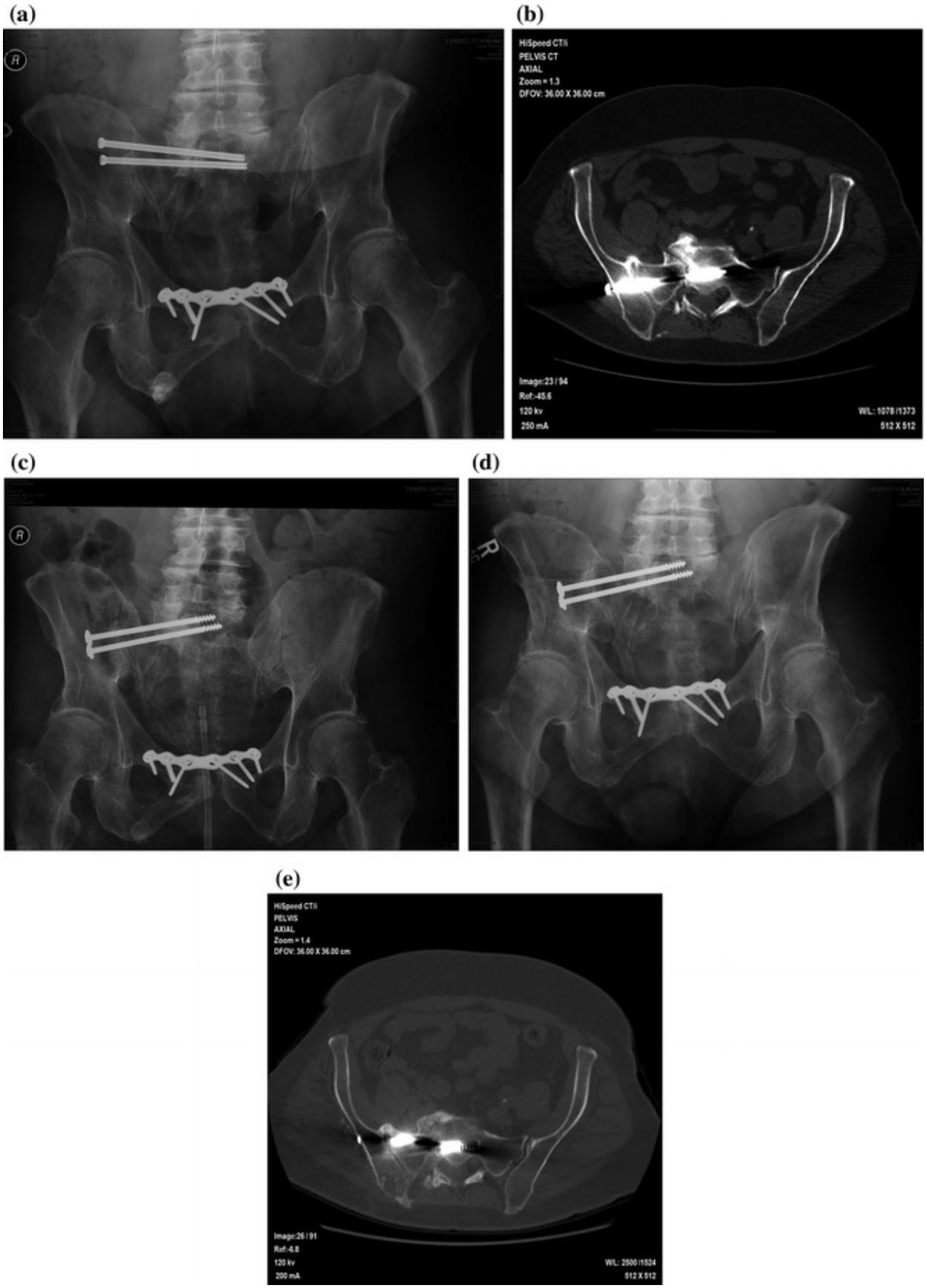

(a)
(b)
HiSpeed CTi
PELVIS CT
AXIAL
Zoom = 1.3
DFOV: 36.00 X 36.00 cm
Image:23 / 94
Ref:-45.6
120 kv
250 mA
WL: 1078 /1373
512 X 512
(c)
(d)
(e)
HiSpeed CTi
PELVIS
AXIAL
Zoom = 1.4
DFOV: 36.00 X 36.00 cm
Image:26 / 91
Ref:-6.8
120 kv
200 mA
WL: 2500 /1024
512 X 512

***Fig. 8.1*** **a** Anteroposterior (AP) pelvis at presentation 2 years after the accident with pain in the sacrum treated previously with cannulated screws. **b** Axial CT scan showing nonunion of the sacrum. **c** Initial postoperative AP pelvis after open reduction internal fixation of the sacral nonunion with iliac crest bone graft and burring sacrum for bleeding bone through a posterior approach. **d** AP pelvis 1 year postoperative showing no lucency around the screws and solid fixation. Patient ambulating with foot pain with minimal pelvic pain. **e** Axial CT 6 months postop showing bridging bone

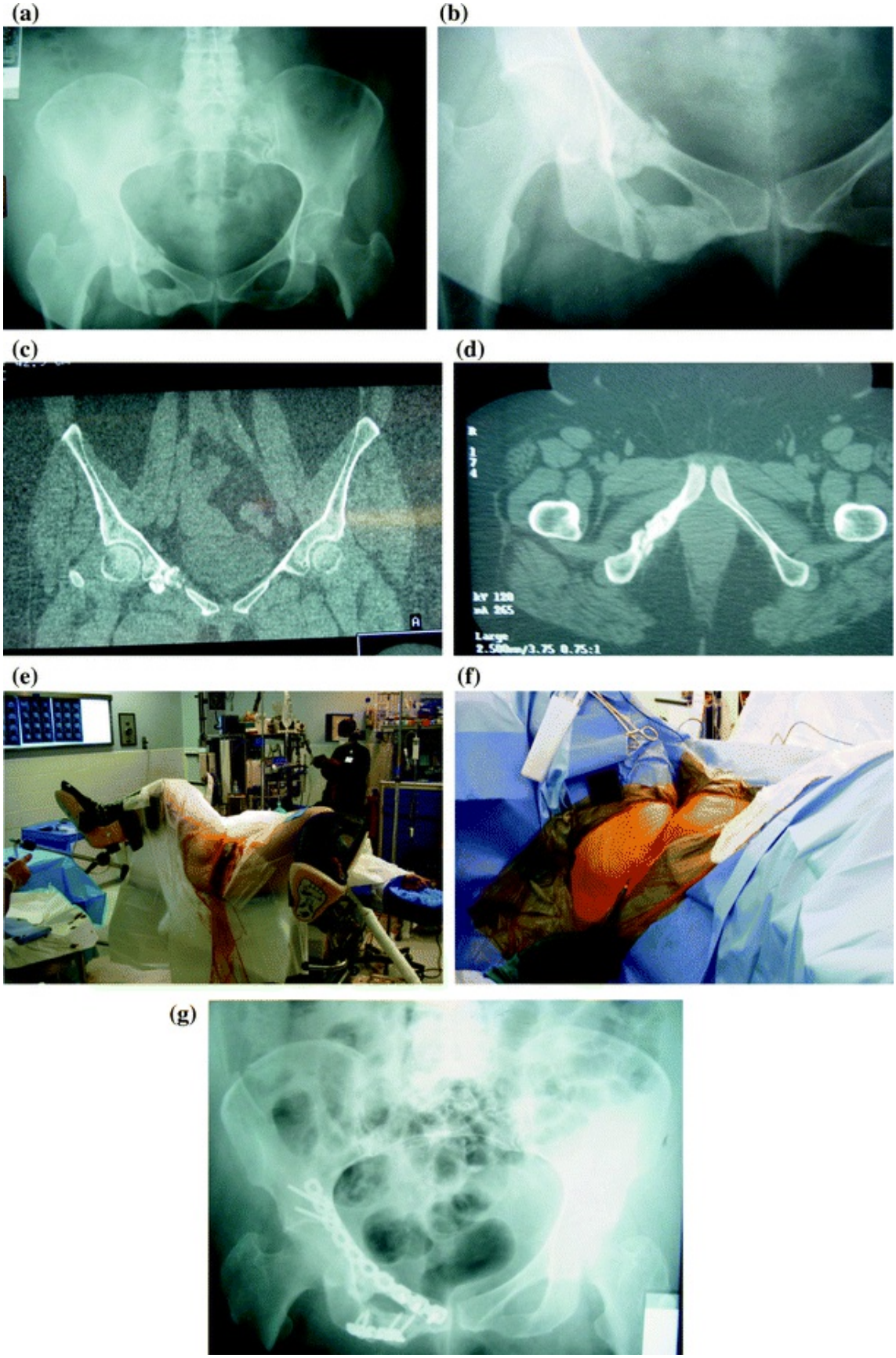
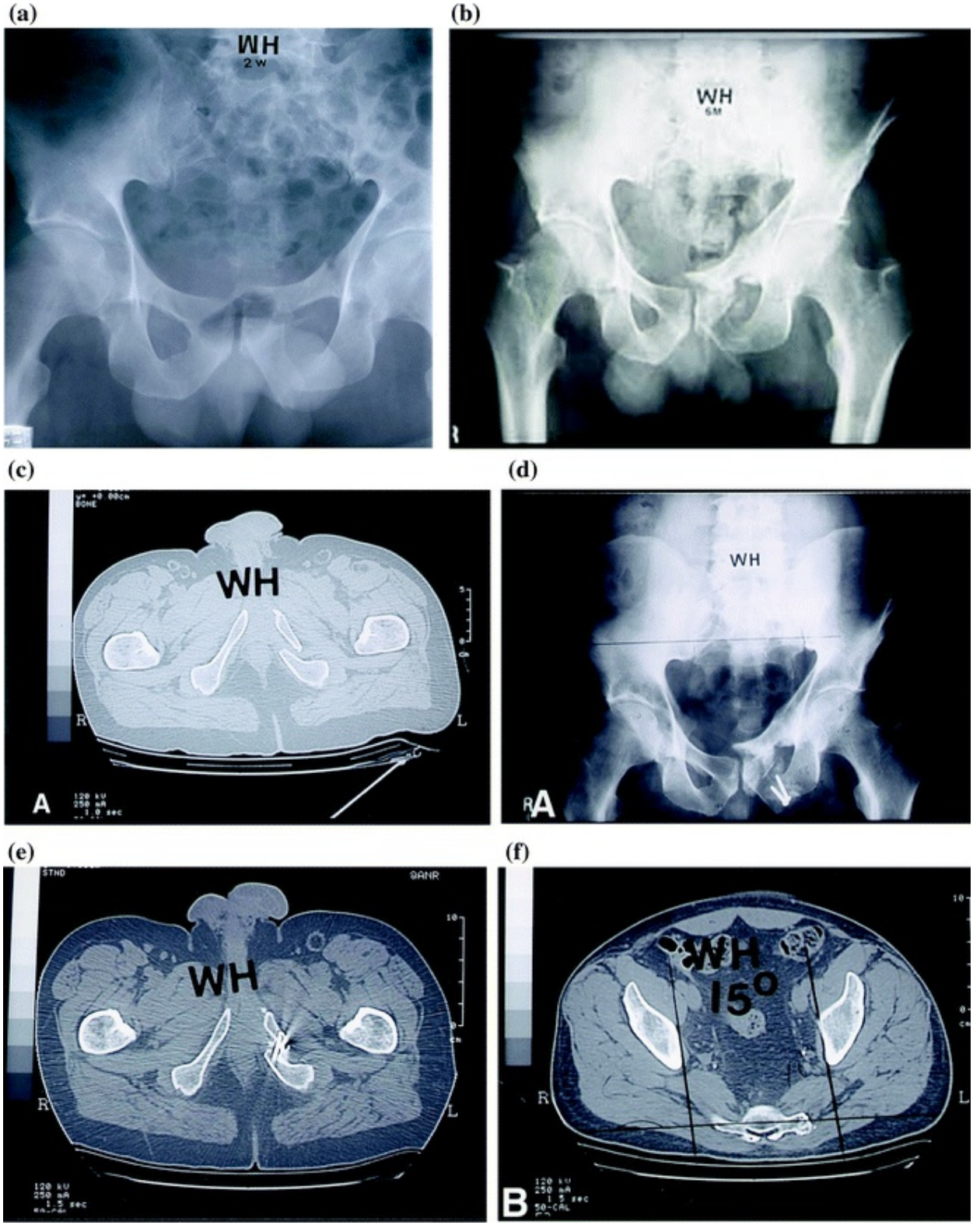

(a)
(b)
(c)
(d)
(e)
(f)
(g)

***Fig. 8.2*** **a** Anteroposterior (AP) view of the pelvis demonstrating normal alignment with a nonunion of the superior and inferior rami after 1 year of symptoms. **b** Magnified view of the nonunion. **c** Coronal CT scan image illustrating nonunion of the superior rami. **d** Axial CT scan image illustrating nonunion of the inferior rami. **e** Intra-operative photograph demonstrating positioning of the patient. **f** Intra-operative photograph demonstrating location of the inferior rami. **g** Postoperative AP view of the pelvis


(a)
WH
2 w
(b)
WH
6M
(c)
WH
A
(d)
WH
A
(e)
WH
(f)
WH
15°
B

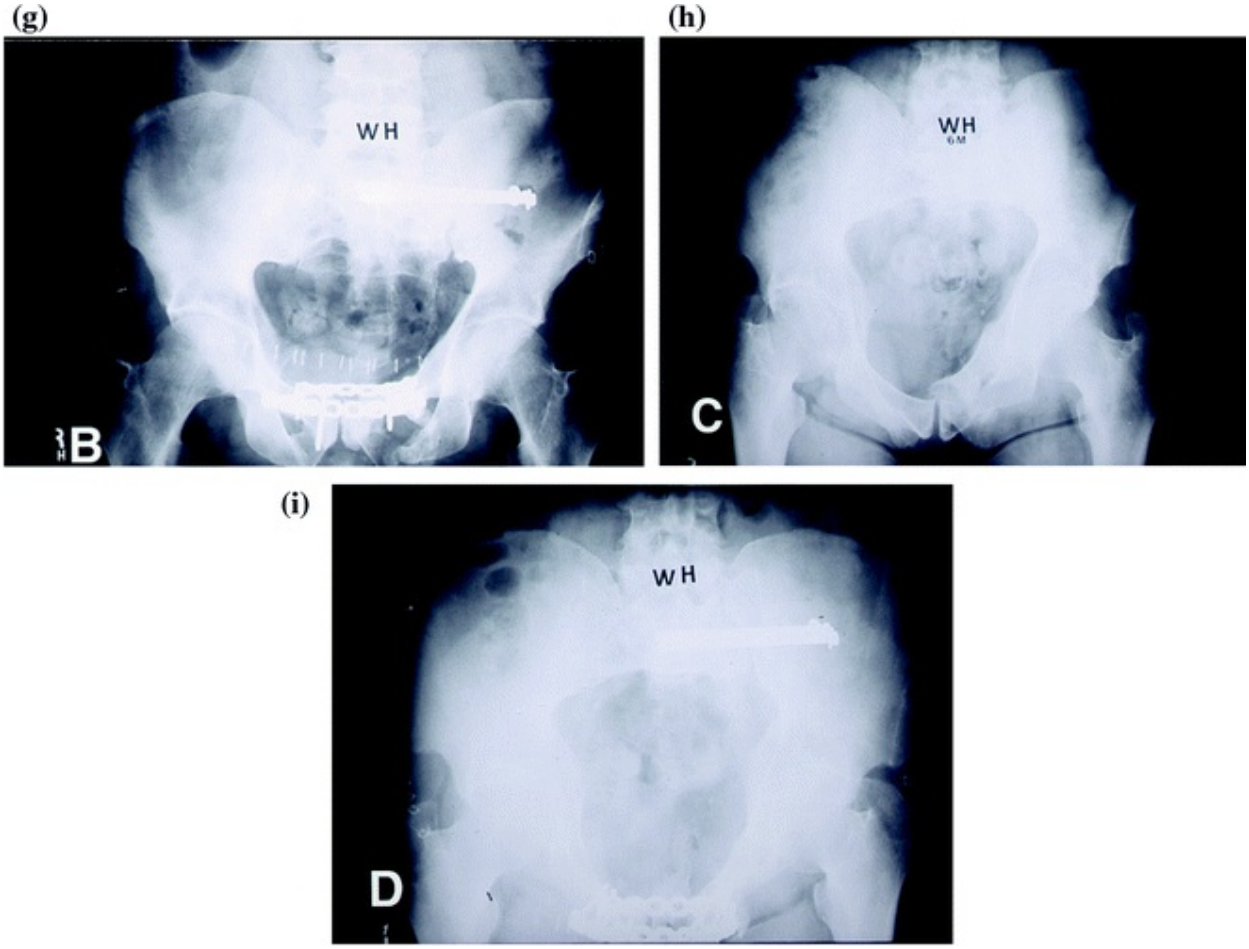



***Fig. 8.3*** **a** Anteroposterior (AP) X-ray of the pelvis from the time of injury. **b** AP X-ray at 6 months postinjury demonstrating malunion of the sacrum and sacroiliac joint and a painful inferior rami nonunion. A rotational deformity of the hemipelvis is also apparent. **c** Axial CT scan image demonstrating ischial nonunion. **d** AP X-ray after screw fixation of the nonunion (surgeon mistakenly fixed a malunion). **e** Axial CT demonstrates a healed nonunion of inferior rami. Groin pain resolved and impotence improved but had an increase posterior SI pain and abnormal gait with intoeing. **f** Axial CT scan image demonstrating 15° internal rotation deformity of the left hemipelvis. **g** AP X-ray status postsacral osteotomy and correction of deformity. **h**, **i** Pre- and postoperative pelvic inlet X-rays illustrating correction of rotational malalignment

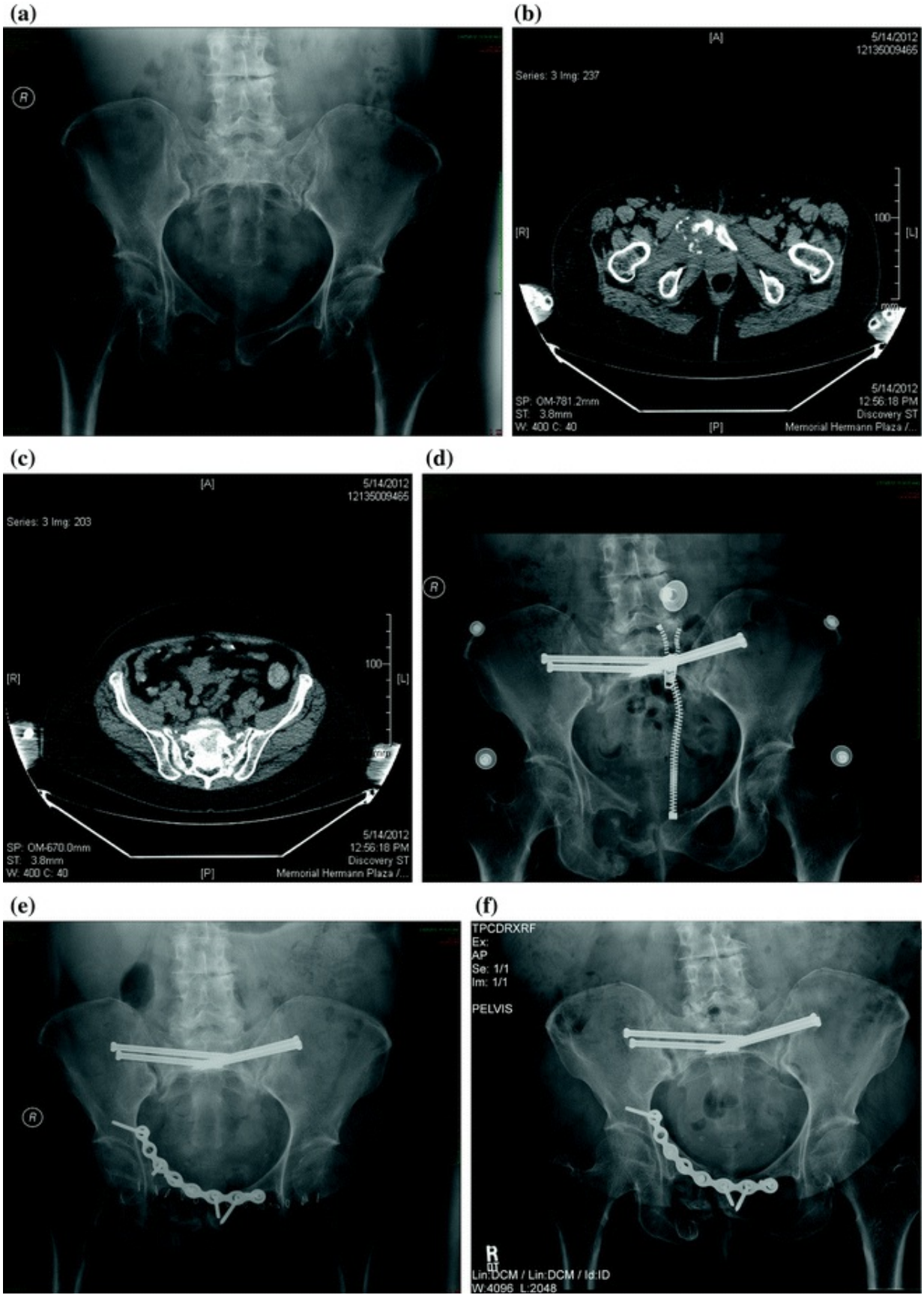

(a)
(b)
[A]
5/14/2012
12135009465
Series: 3 Img: 237
[R]
100
[L]
SP: OM-781.2mm
ST: 3.8mm
W: 400 C: 40
[P]
5/14/2012
12:56:18 PM
Discovery ST
Memorial Hermann Plaza /...
(c)
(d)
[A]
5/14/2012
12135009465
Series: 3 Img: 203
[R]
100
[L]
SP: OM-670.0mm
ST: 3.8mm
W: 400 C: 40
[P]
5/14/2012
12:56:18 PM
Discovery ST
Memorial Hermann Plaza /...
(e)
(f)
TPCDRXRF
Ex:
AP
Se: 1/1
Im: 1/1
PELVIS
Lin:DCM / Lin:DCM / Id:ID
W:4096 L:2048

***Fig. 8.4*** **a** Anteroposterior (AP) X-ray after a bladder sling (no trauma but an insufficiency fracture). **b** Axial CT scan showing the bilateral sacral fractures. **c** Axial CT scan of the rami nonunion. **d** Postoperative AP X-ray showing the percutaneous placement of bilateral iliosacral screws. **e** Postoperative AP pelvis 8 weeks out showing all fixation. **f** AP pelvis at 17-month follow-up

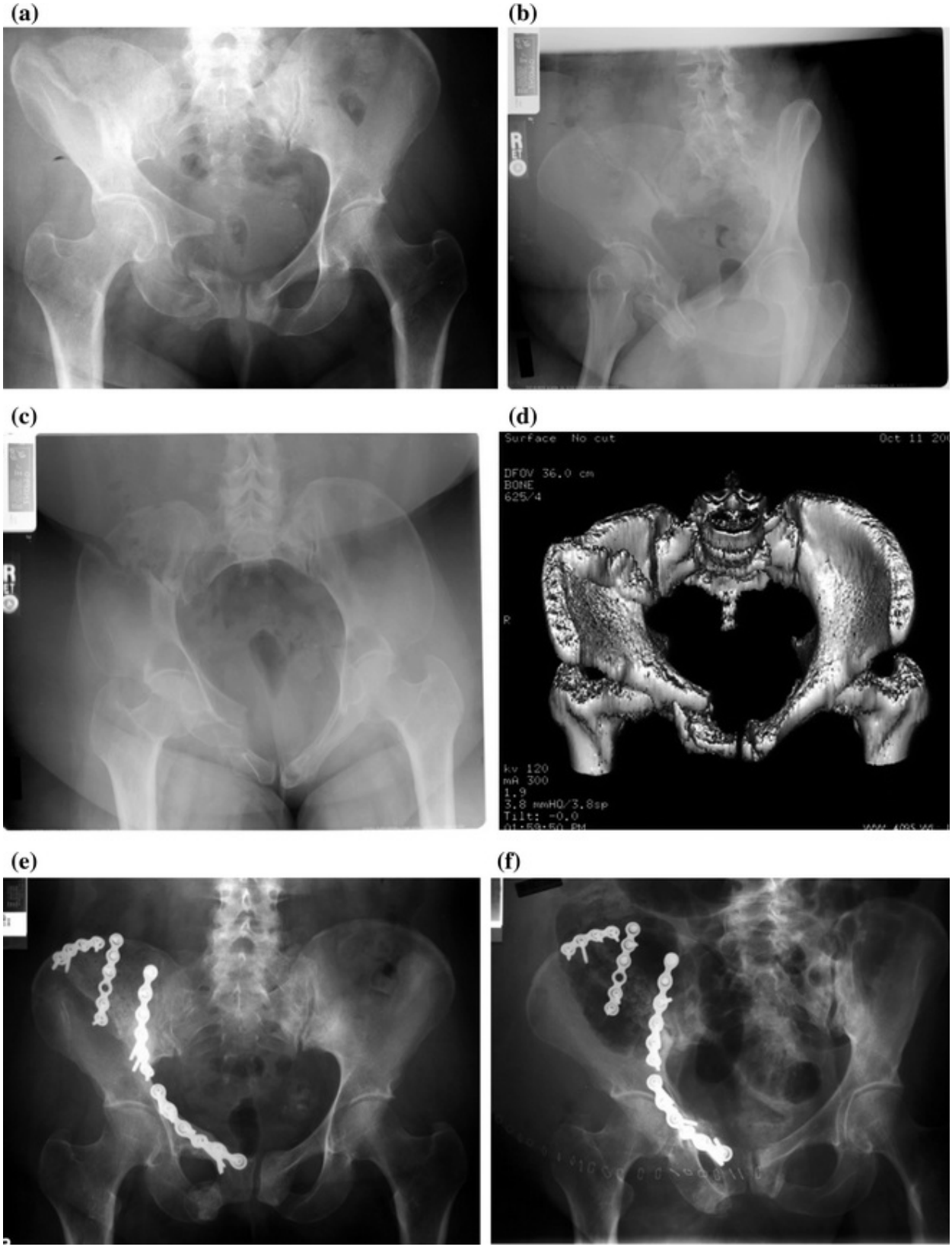



***Fig. 8.5*** **a–c** Anteroposterior (AP), iliac oblique, and inlet X-rays of pelvis demonstrating the pelvic

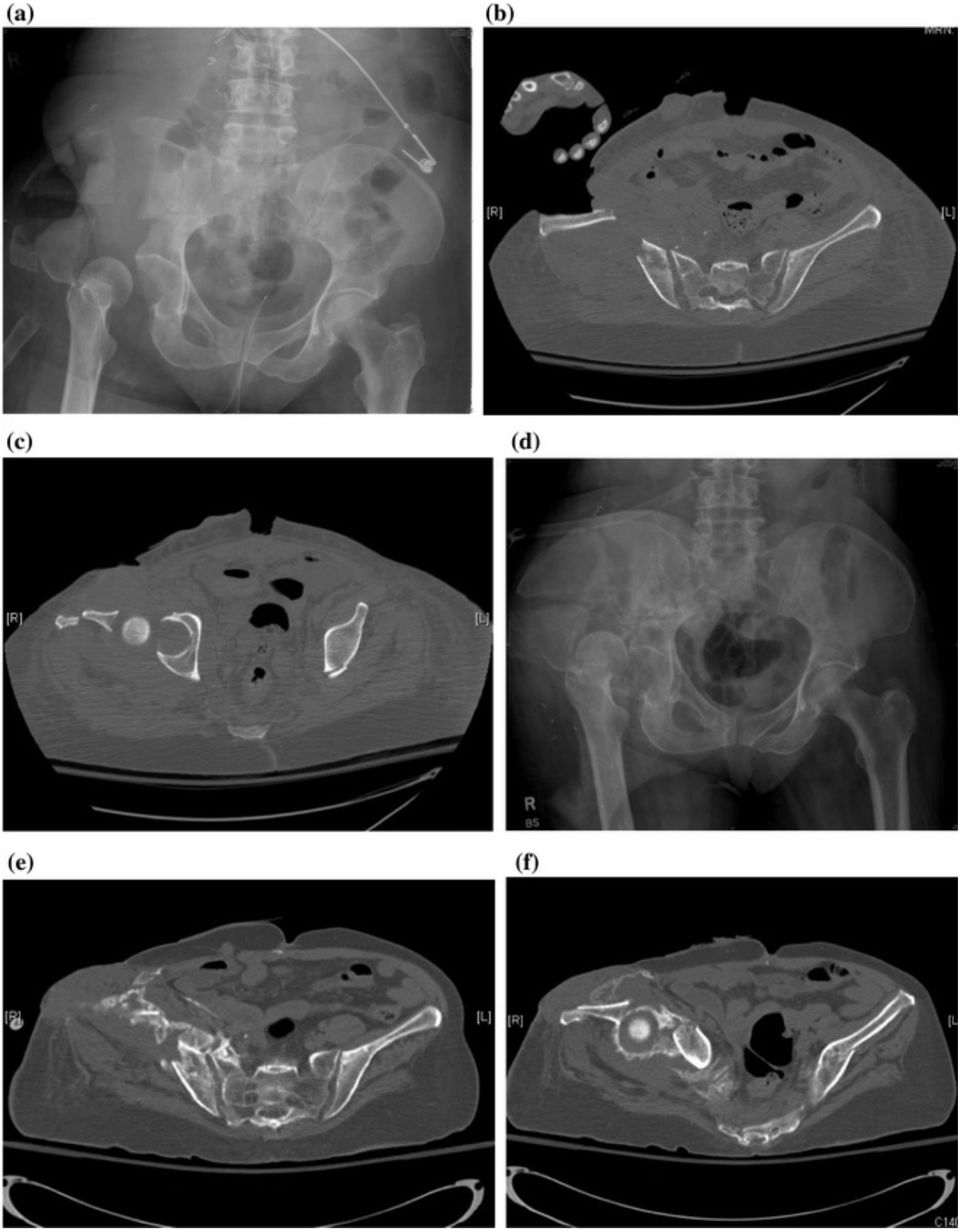
ring nonunion with translation, flexion, and internal rotation deformities. **d** Three-dimensional CT reconstruction of the pelvis. **e**, **f** Postoperative AP and iliac oblique X-rays of the pelvis

Pain secondary to nonunion of the pelvis is often present during weight bearing and improves with rest. Because weight is transmitted posteriorly through the pelvis, pain is more commonly associated with the sacrum or SI nonunions (see Fig. 8.1). Nonunions of the anterior pelvic ring are rarely painful because less than 10% of the body's weight is transmitted through the anterior part of the pelvis [4]. When the rare case of a painful nonunion of the anterior pelvic ring does present, it is often following a protracted course and multiple consultations with medical specialists (gynecologists, general surgeons, urologists, rheumatologists, etc.) (see Figs. 8.2 and 8.3). The patient may also experience low back pain secondary to the pelvic deformity, or neurogenic pain that radiates to the ankle secondary to compression or distraction of the nerves at the level of the roots or the lumbosacral plexus. Scarring within the nerve is a common cause of chronic pain.

Patients may also complain of pain while sitting or lying down. The two major causes for this are pelvic malalignment that causes sitting or lying imbalance and ischial nonunions that result in painful motion of the fracture upon sitting. The pain with nonunion is due to the micromotion that is occurring at the nonunion site.

### 8.1.1 Deformity

Pelvic deformity is responsible for complaints in many clinical areas—pain, gait abnormalities, genitourinary system, etc.—and is beyond the scope of this chapter. Occasionally, failure to heal or a nonunion will eventually displace, resulting in an associated malalignment (see Fig. 8.3). Additionally, initial displacement or deformity can separate the bone enough to cause a nonunion (Fig. 8.6).


(a)
(b)
[R]
[L]
(c)
[R]
[L]
(d)
R
(e)
[R]
[L]
(f)
[R]

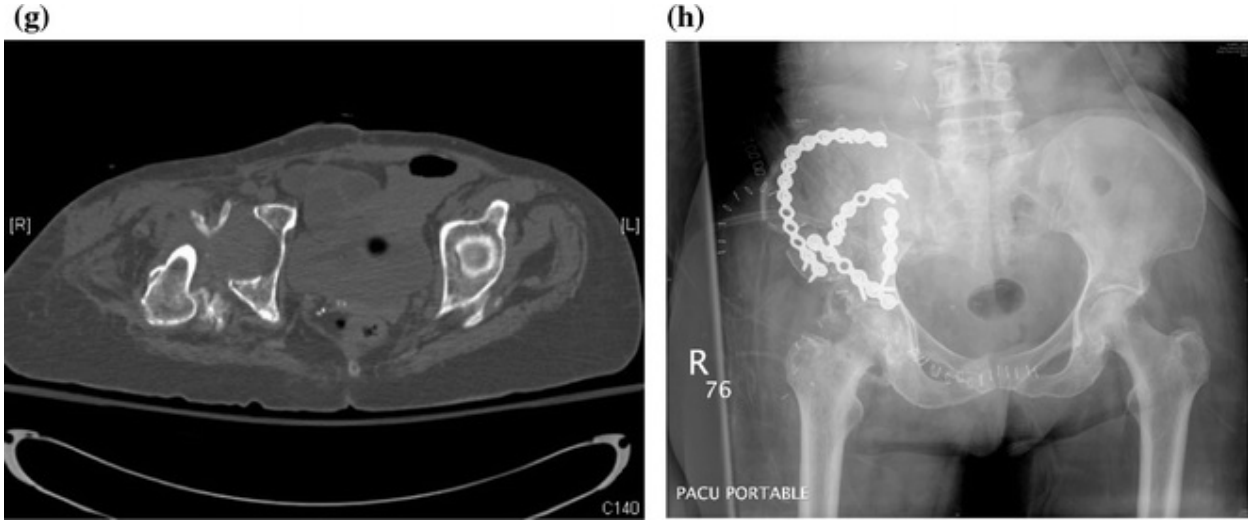



***Fig. 8.6*** **a** Anteroposterior (AP) X-ray of the pelvis demonstrates significant lateral displacement of the femoral head. **b**, **c** Axial CT scan images demonstrating displacement of iliac wing and dislocation of femoral head. **d** AP X-ray of the pelvis 3 months postinjury. **e–g** Axial CT scan images demonstrate extensive callous around the fracture site. **h** AP X-ray of the pelvis following repair of the nonunion

## 8.1.2 Neurologic Injuries

Permanent nerve damage is a common cause of disability following pelvic injuries. A nerve injury occurs in 46% of the patients with an unstable vertical pelvis [5]. The most commonly affected nerve roots are L5 and S1, but any root from L2 to S4 may be damaged. In Huittinen's [5] study of 40 nerve injuries, 21 (52.5%) were traction injuries, 15 (37.5%) were complete disruptions, and 4 (10%) were compression injuries. Interestingly, the lumbosacral trunk and superior gluteal nerve sustained traction injuries, while most of the disruptions occurred in the roots of the cauda equina. Compression injuries occurred in the upper three sacral nerve foramina in patients with fractures of the sacrum. Furthermore, the traction and nerve disruption injuries occurred in the vertically unstable pelvic injuries, while the compressive nerve injuries occurred following lateral compression of the pelvis. Lateral compression injuries of the pelvis often impact portions of the sacral bone into the foramen, resulting in compression of the nerve, and may require decompression if neurologic examination worsens.

A thorough neurologic examination is necessary to determine any preoperative deficits, and for intra-operative as well as postoperative nerve monitoring. Disruption of peripheral nerves should be evaluated by nerve conduction/electromyography tests. Peripheral disruptions may be repaired

with possible salvage of some function or more consistent return of protective sensation. Myelograms and magnetic resonance imaging (MRI) are used to rule out spinal nerve avulsions.

Our studies on malunions and nonunions show that 57% of the patients had a preoperative nerve injury and only 16% were resolving postoperatively [2, 3]. Only one patient in our studies would not have the nonunion/malunion surgery again, and this was due to a postoperative nerve complication. The patient underwent two operations on a 16-year-old nonunion that was extremely mobile. An L5 nerve root injury occurred from likely reducing a vertical malreduced hemipelvis almost 5 cm. The patient required reoperation for persistent nonunion. At the time of the second operation, the posterior fixation was changed. The complaints of deformity were completely resolved, but the patient still suffered from pain in the L5 nerve distribution, despite having a stable pelvis.

### 8.1.3 Patient Expectations

An important aspect of the preoperative assessment is to discover a patient's understanding and expectations regarding their clinical problem. Significant discussion is necessary prior to making a decision for surgery. The patient must make the final decision based upon realistic goals and an understanding of the risk of complications. Specific symptoms of deformity, such as limb shortening, sitting imbalance, vaginal impingement, and cosmetic deformity, are expected to be reliably addressed by surgery. The patient must be cautioned, however, that while the majority of the deformity can be corrected, the actual anatomic result is usually less than perfect. In our series of pelvic malunions, only 76% of our reductions had less than 1 cm of residual deformity [2, 3].

Posterior pelvic pain in the absence of a demonstrable nonunion or instability is often difficult to explain and may not completely or reliably improve with the correction of the pelvic deformity. Ninety-five percent of patients with malunion of the pelvis report improvement of their pain; however, only 21% have complete relief of their posterior pain [2, 3]. Radiographic evidence of SI joint arthrosis is not a reliable indication of the cause of posterior pelvic pain. However, in patients with a pelvic nonunion, a significant reduction in pain is seen (see Figs. 8.1, 8.3, 8.4, and 8.5).

### 8.1.4 Radiographic Assessment

Radiographic assessment includes five standard pelvis X-ray views (AP, both 45° oblique views, 40° caudad, and 40° cephalad), a weight-bearing AP X-ray, computed tomography (CT) scan, and a three-dimensional CT. The CT scan can be used to make a three-dimensional pelvic model. This model helps the surgeon to understand the deformity and plan preoperatively. The displacement and rotation of all fragments need to be understood so that appropriate release and reduction of fragments can be obtained. An obturator oblique clearly shows the SI joint on the ipsilateral side, while a single-leg weight-bearing AP determines stability of the nonunions. Technetium bone scans may be helpful in identifying the activity of the nonunion (atrophic or hypertrophic) but are not routinely ordered. Together, these multiple plain films and CT scans are used to assess nonunions and deformities of the pelvis. The displacements are often complex and include rotational and translational displacements around a three ordinate axis.

Plain X-rays will often show the anterior rami nonunions (see Figs. 8.2, 8.3, 8.4 and 8.5). A CT scan with sagittal and coronal reconstructions are required to define the posterior pelvic nonunions (see Figs. 8.1 and 8.4). When viewing the CT, all of the slices need to be studied. Musculoskeletal radiologists sometimes call a nonunion because a particular slice may not have any apparent boney bridging, but following the individual pieces will often show some slices with boney bridging, eliminating the diagnosis of nonunion.

Evaluating the nonunion site radiographically can determine whether the nonunion is hypertrophic (needs stability), oligotrophic (may need stability and biology), or atrophic (needs biology). Atrophic nonunion s, especially in the pelvis, should alert the surgeon to the possibility of a hormonal, nutritional, or medical problem. Working with a bone endocrinologist is helpful in these difficult cases (see Figs. 8.4 and 8.5). Infection is always a concern in any nonunion, so C-reactive protein (CRP) , erythrocyte sedimentation rate (ESR) , and white blood count (WBC) are routinely ordered. In infected nonunions (see Figs. 8.1 and 8.4), these parameters are important to follow; the use of these labs on and off antibiotics can help determine when the infection is controlled enough to fix the nonunion.

### 8.1.5 Treatment

The best treatment is prevention [1, 6–9]. But even after adequate treatment, nonunions can occur (see Figs. 8.1, 8.2, 8.3 and 8.4). The problem of malunions and nonunions appears most commonly after inadequate initial treatment of displaced fractures and unstable pelvic ring injuries (see Fig. 8.5) [10]. In the acetabulum, nonunions can occur in transverse fractures when these fractures are treated nonoperatively, with inadequate fixation (e.g., one-third tubular plates without lag screws instead of reconstruction plates and lag screws), or there are medical problems with the patient (e.g., malnutrition, vitamin D deficiency, rickets). In pelvic nonunions, the same possible causes exist, nonoperative or inadequate fixation and medical comorbidities. Because most of the weight is transferred through the posterior pelvis, anterior rami nonunions rarely exist and, if present, are rarely symptomatic. However, if not treated, rami nonunions in elderly patients can lead to additional pathological fractures (see Fig. 8.4). More commonly nonunions occur posteriorly and are due to unrecognized instability of the pelvis with vertical instability that is treated nonoperatively or with an external fixator. These nonunions progress to malunions. From the technical standpoint, late correction is very difficult; the anatomy is altered and less recognizable, and the potential complications are increased. Osteotomies can easily damage the structures that lie on the opposite side of the bone. Scarring around nerves prevents the fragments from moving freely without causing nerve palsy.

Indications for surgery include pain, pelvic ring instability, and clinical problems relating to the pelvic deformity (gait abnormalities, sitting problems, limb shortening, genitourinary symptoms, vaginal wall impingement, etc.). A thorough knowledge of pelvic anatomy is required to understand the three-dimensional deformity. Furthermore, extensive preoperative planning is needed to determine the proper order of exposures for release, reduction, and fixation. Because each patient is different, it behooves the surgeon to individualize the treatment.

Previous literature focused on simple nonunions. These patients often do not require extensive anterior and posterior ring releases and reduction, and respond to in situ fusion only (see Fig. 8.4). Pennal and Massiah showed that patients treated with surgery are significantly better than those treated conservatively [11]. In their study, 11 out of 18 surgery patients returned to preinjury occupation versus five out of 24 conservatively treated patients. In nonunion cases with significant displacement, in situ fusions are unrewarding

and leave the patient with complaints related to deformity as well as significant pain (see Fig. 8.3).

Surgical technique in the presence of significant deformity can be complex and is beyond the scope of this chapter, but one must be familiar with correction of deformities along with the management of the nonunion. This chapter will focus on healing the bones that generally require only one stage versus the more complex three-stage reconstruction as described by Letournel [2, 3, 7].

A radiolucent table with image intensification is commonly used for the procedure. The Judet table is also useful for positioning the leg anywhere in space to help with the approaches and exposure. Somatosensory-evoked potentials and motor-evoked potentials have been used on some patients intra-operatively that require significant correction of vertical displacement and but are not routinely used.

### 8.1.6 Pelvic Nonunions

Painful nonunions without deformity can be treated with stabilization, bone graft, or both. A technetium bone scan can indicate activity of the nonunion (atrophic [requires bone graft] or hypertrophic [requires stabilization]). In most cases, it is not necessary, and surgery involves both bone graft and stabilization.

The preparation of the bone is the same no matter where the nonunion occurs. Cultures are always taken regardless of how the nonunion looks. We have seen a number of culture-positive less virulent organisms that do not form pus but infect and prevent bones from healing. The two sides of the bone are exposed with as minimal stripping as possible. It is important to remember that the blood supply comes predominantly from the soft tissue. The surgeon burrs both sides of the bone until cortical bleeding is seen. The surgeon then either burrs or drills inside the nonunion site to penetrate any cortical cap and get bleeding from the cancellous bone. The surgeon then lays cancellous strips from bleeding to bleeding bone. This is covered with good bleeding muscle. The fixation is either intra-medullary (i.e., iliosacral screws) or plate fixation next to the cancellous graft not covering it.

Nonunions of rami fractures are rare. If they occur, they are often located in the medial aspect of the pubis bone or in the symphyseal region. Because more than 90% of weight bearing is posterior, many nonunions of the anterior pelvic ring are asymptomatic. Because these are so rare, they are often not

part of the differential diagnosis, and some patients can be evaluated by several specialists (obstetrics and gynecology, general surgery, etc.) before an X-ray identifies a painful nonunion (see Fig. 8.2). Often, treatment of symptomatic superior rami nonunion will heal the inferior rami nonunion, especially if the nonunion is hypertrophic or the underlying medical comorbidity has been corrected (see Fig. 8.5). However, there are cases where plating both the superior and the inferior rami is required, especially in cases of atrophic nonunions (see Fig. 8.2).

A Foley catheter is always placed preoperatively. A Pfannenstiel incision is made 2 cm cephalad from the symphysis. The decussation of the fascia fibers of the rectus abdominis marks the division between the two heads of the rectus. The two heads are split, with extreme care being taken to avoid entering the bladder. The surgeon then inspects the bladder to detect any perforations. The Foley should be palpated to ensure that the urethra is intact. A malleable retractor or lap sponge is then used to hold the bladder away from the symphysis pubis. Two Hohmann retractors are used to retract the two heads of the rectus from the superior surface of the symphysis pubis. The superior surface of the superior rami is cleaned for the plate, but the anterior insertion of the rectus remains intact. A large Weber clamp or pelvic reduction clamp can be used anteriorly to hold the symphysis together or rami fracture together. Usually, a six-hole 3.5 mm curved reconstruction plate is then implanted. Clinical research supports the implantation of this device [12]. Rarely, when a patient has a very small symptomatic nonunion of the pubis or late painful instability of the symphysis, a symphyseal fusion is indicated. When a fusion of the symphysis is needed, an additional four-hole plate is used anterior to the symphysis with cancellous bone graft. Additionally, when fusion of the symphysis is indicated, an eight to ten-hole plate is used rather than a six-hole plate superiorly. Through the Pfannenstiel approach, the SI joints can be visualized and the quadrilateral surface exposed via the modified Stoppa approach [13]. Therefore, a plate can be placed from the symphysis to the SI joint along the brim superiorly bilaterally. Furthermore, a plate can be placed within the pelvis from the symphysis along the quadrilateral plate to the SI joint. Plates or screws can be used on the inferior rami (see Fig. 8.2) via a direct approach, with the patient in the lithotomy position. This position allows the surgeon to also perform a Pfannenstiel incision as well. Rarely, in already osteoporotic bone, rami nonunion can cause enough instability to cause insufficiency fractures

posteriorly (see Fig. 8.4). Elderly simple rami fractures need to be worked up for osteoporosis to prevent this disastrous complication. The initial treatment of these insufficiency fractures includes touchdown weight bearing with no range of motion or exercises to that side for eight weeks. If the patient continues to be symptomatic, the pelvis is stabilized with posterior iliosacral screws (for posterior insufficiency fractures) and/or platting of rami fractures with another eight weeks of touchdown weight bearing. These patients are aggressively treated (parathyroid hormone, vitamin D, calcium, etc.) by a bone endocrinologist. Furthermore, the failure of rami to heal properly may cause further displacement and eventual pelvic malunion (see Fig. 8.3).

For iliac wing nonunions, the lateral window of the ilioinguinal approach is performed (see Fig. 8.5). Most of these have occurred with nonoperative treatment or in a patient with a significant bone-healing problem. The L5 nerve runs 2 cm medial to the SI joint and must be protected. If vertical translation has occurred, mobilization of the nerve is required to reduce the hemipelvis without causing nerve palsy. Bicortical 3.5-mm screws are used in a reconstruction plate. Anterior plating of the SI joint with two three-hole plates positioned 90° to each other (first plate along the brim with one screw in the sacrum and 2 screws in the ilium) can give excellent fixation of the SI joint. Since the joint from anterior has about a 15° medial slope, angling the drill in this direction will provide longer screws. The use of a long oscillating drill, which allows tactile feel of the second cortex and prevents over penetration with the drill, is recommended because of its flexibility and safety. Alternatively, percutaneous iliosacral screws can be placed. Iliac wing nonunions usually require plate fixation only without involvement of the SI joint. Depending on the reduction required, the iliac portion of the ilioinguinal and the Pfannenstiel may be adequate for the combination iliac wing and rami nonunion. However, in chronic nonunions of both the pelvis and the acetabulum, a full ilioinguinal is usually required (see Figs. 8.4 and 8.5).

Sacral nonunions (see Fig. 8.1), due to limited visualization from an anterior approach, almost always are operated on through a posterior approach. A longitudinal approach two centimeters lateral to the posterior superior iliac spine is made [10]. The gluteus maximus is raised off of the iliac crest, lumbodorsal fascia, and paraspinal muscles, exposing the posterior SI joint and ligaments. The bone is prepared as above, being very careful of the sacral nerve roots. Fixation is usually obtained with two 6.5 mm, 16-mm-

thread-length iliosacral screws. Again, the use of an oscillating drill is recommended for safety and so that three cortices are entered but not the fourth. Additional stability can be achieved by placing one or two posterior reconstruction plates from one iliac wing to the other iliac wing. These plates act as a tension band and are less prominent if placed caudad to the posterior superior iliac spine. These tension band plates (typically 12–14 holes) have three screws in both iliac wings, with one screw from shooting between the inner and outer cortical tables and the second and third shorter screws transversing the two tables. Iliosacral bars are also an option; however, they are usually prominent and were not used in our series [2, 3]. More recently, trans-sacral screw fixation has been described [14, 15], but in our series of sacral injures, these were rarely required [10]. There is theoretical risk of compression of the nerve roots in comminuted sacral fractures using a lag screw. However, using fully threaded screws risks maintaining a gap at the fracture site, potentially increasing the risk of nonunion. In the author's experience of greater than 1000 lag iliosacral screws, with only 2 cases of nerve palsy that eventually resolved, use of a fully threaded screw is rarely used. Furthermore, the pelvic nonunions referred to our institution, if operated on, usually have a malreduced pelvis, and a fully threaded screw possibly increases the risk of nonunion.

Patients are touchdown weight bearing or wheelchair transfer for only 12 weeks postoperatively. After adequate healing, range of motion and strengthening exercises are instigated.

## 8.1.7 Malunions and Displaced Nonunions of the Pelvis

To treat symptoms related to deformity of the pelvis, a reduction of the pelvis is required because a simple in situ fusion will be unrewarding and not completely relieve the pain (see Fig. 8.3). If a nonunion leads to pelvic deformity, osteotomy, and release of the hemipelvis is required to reduce the deformity and alleviate symptoms (see Fig. 8.3) [16]. Correction of the deformity is beyond the scope of this chapter.

## 8.1.8 Results

In our series that included both nonunions and malunions, the time frame from injury to operation in our series averaged 42 months (range from

4 months to 14 years) [2, 3]. In our initial series, 9% (3/34) had pure iliac wing nonunions, but the combination fracture dislocation of the SI joint produced 35% (12/34) nonunions. By far the most common nonunions were in the sacrum, which were 56% (19/34) of the nonunions. Most rami fractures associated with the nonunions had healed, but there were some cases of continued symphyseal instability. Sixty-six percent (23/35) were initially treated nonoperatively, and seven of these had a previous attempt to fix their nonunion/malunion. The cases that were operated were inadequately stabilized with an external fixator or screw and plate fixation. The more recent trend is that of more cases being operated on, but there is inadequate fixation with an external fixator, or adequate reduction is not achieved with iliosacral screws. Operative time averaged 7 h (range 1.5–10.4 h). Operative blood loss averaged 1977 cc (range 200–7200 cc).

At follow-up (average, 3 years, 11 months; range, 9 months to 11 years), all but one patient had a stable union of their pelvic ring. Ninety-five percent of the patients were satisfied with the operation, and 100% of the patients were satisfied with the improvement of their preoperative deformity. As mentioned earlier, the unsatisfied patient continues to have L5 nerve palsy. Now, with the experience of over a hundred pelvic nonunion and malunion patients, prevention is still the key.

Complications included loss of reduction, neurologic injury, and vascular injury (external iliac vein). There were no postoperative surgical infections, although there were some preoperative infections that had to be treated with a staged approach (see below). Although residual low back pain was present in most of the patients preoperatively, 95% reported less pain following surgery.

## 8.1.9 Pelvic Nonunion Case Discussions

**Case 1. Nonunion of the sacrum** (see Fig. 8.1)

A 62-year-old was involved in a head-on motorcycle collision with complicated urologic injuries and history of infections in both the symphysis and the sacrum. Patient initially treated with iliosacral screws and symphyseal plating. Patient with persistent sacral (posterior) pain at 2 years. Radiographic imaging revealed sacral nonunion. Patient underwent bone grafting and revision fixation of the sacrum with a successful union.

**Case 2. Painful nonunion of the anterior pelvic ring** (see Fig. 8.2)

A 67-year-old presented 1 year postinjury after multiple consultations with medical specialists. Patient found to have nonunion of the anterior pelvic ring confirmed by radiographic imaging (plain and CT). Patient underwent stabilization of the superior and inferior rami with complete healing and resolution of pain. Patient stopped using narcotics 1 week postop after being on narcotics for over 1 year. Patient continues to be pain free >2 years postoperatively.

**Case 3. Patient with a nonunion of the inferior ramus with associated deformity of left hemipelvis** (see Fig. 8.3)

A 58-year-old presented 6 months postinjury with complaints of groin pain and impotence. Patient found to have nonunion of inferior rami left side, which was fixed but associated malunion was not addressed at first surgery. Patient's groin pain resolved and impotence improved. Patient now had increase in posterior SI pain and abnormal gait in intoeing of left side. After evaluation and determination of a left hemipelvis internal rotation deformity, surgical osteotomy of the sacrum with correction of the deformity was performed. Pain diminished and patient returned to work as a train conductor.

**Case 4. Rami nonunion leading to insufficiency bilateral sacral fractures after placement of a vaginal bladder sling** (see Fig. 8.4)

A 53-year-old with a 4-month history of groin pain and a 1-month history of being unable to ambulate. Patient had a trans-vaginal bladder mesh placed 5 months previously and woke up (no history of any trauma) 1 month later with groin pain. The patient was eventually worked up after finding a right rami fracture with an MRI and CT scan, which were negative for any other lesion. She was found to be vitamin D deficient and was started on appropriate therapy. 3 months later or 7 months after the onset of symptoms without any trauma, the patient presented to our clinic with increasing bilateral posterior pain eventually diagnosed as bilateral insufficiency sacral fractures. Patient underwent stabilization with iliosacral screws and anterior plating, cancellous bone graft, and placement of BMP-2 (Infuse® Medtronics) of the rami nonunion. The right superior ramus had debridement of granulation tissue suspicious for a chronic burnt-out infection that had negative cultures. Patient treated with 6 weeks of broad-spectrum intravenous antibiotics. Patient was now able to ambulate (after 10 months of nonambulating) with some pain.

**Case 5. Nonunion of the iliac wing and superior and inferior rami with no history of trauma and orthopedic diagnosed malabsorption syndrome** (see Fig. 8.5)

A 49-year-old presented 1 year postpelvic fracture after being referred to multiple medical specialists before X-rays were taken. Patient was unable to ambulate for the previous 9 months. Patient was found to have a pelvic nonunion with significant associated deformity. Patient underwent single-stage repair of the nonunion through an ilioinguinal approach. Patient is ambulating without pain 5 years postop.

---

## 8.2 Nonunion of the Acetabulum: Clinical Assessment and Pain

Pain associated with an acetabular nonunion usually occurs either at the nonunion site or in the hip joint. An acetabular nonunion may have a slight malreduction at the joint causing pain because of increased intra-articular pressure during weight bearing. This is due to articular incongruity reducing the contact area between the head and the acetabulum, wear of the head rolling over a malreduced fracture line, avascular necrosis, or osteoarthritis of the acetabulum. Symptoms include increasing severity of pain with hip motion, limp, and restriction of hip motion. Radiographic studies are used (as described later) to determine whether a nonunion exists and the extent and location of the damage in the hip. Critical to the preoperative assessment of acetabular nonunion is the condition of the femoral head. Evaluation of the hip joint is also important to determine how much cartilage remains. Attempts to compensate for loss of substance of the femoral head or the cartilage have not been successful. The osteoarthritis rarely improves, and at best the deterioration is halted. Before attempting reconstruction of an acetabular nonunion, the following must be understood:

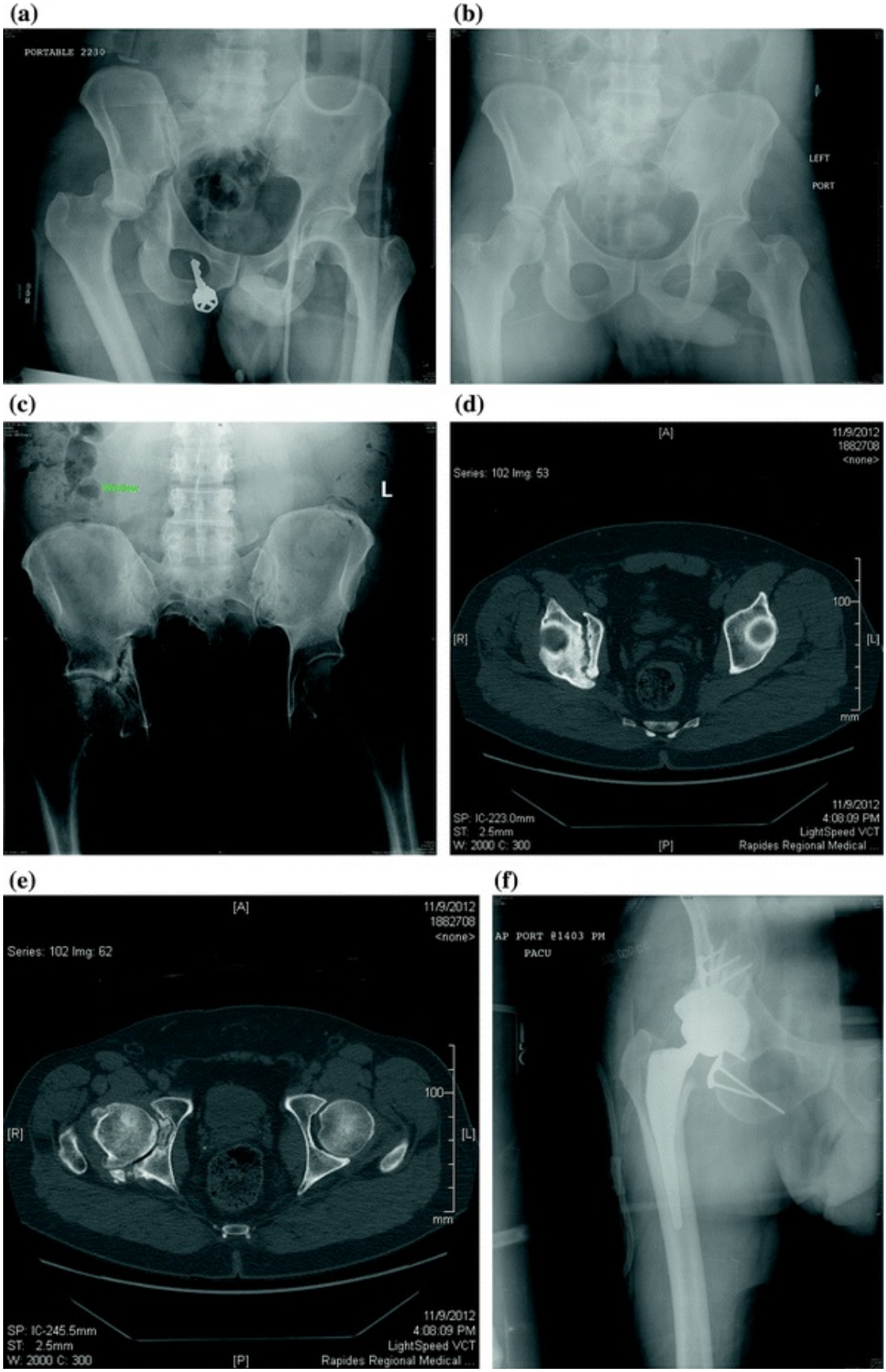
1. The location and condition of the different articular fragments and the bony columns supporting them,

2. The extent and location of wear on the femoral head,

3. The presence, location, and extent of osteoarthritis, and

4. The presence, location, and extent of avascular necrosis [7].

In all cases, a total hip arthroplasty (THA) is considered an option (see Fig. 8.6). If there is complete cartilage loss involving more than 50% of the dome, a THA is probably required (Fig. 8.7). Depending on the associated deformity, the THA may need to be performed in conjunction with reduction of the columns (Fig. 8.8).


(a)
PORTABLE 2230
(b)
LEFT
PORT
(c)
L
(d)
[A]
11/9/2012
1882708
<none>
Series: 102 Img: 53
100
[R]
[L]
mm
SP: IC-223.0mm
ST: 2.5mm
W: 2000 C: 300
[P]
11/9/2012
4:08:09 PM
LightSpeed VCT
Rapides Regional Medical ...
(e)
[A]
11/9/2012
1882708
<none>
Series: 102 Img: 62
100
[R]
[L]
mm
SP: IC-245.5mm
ST: 2.5mm
W: 2000 C: 300
[P]
11/9/2012
4:08:09 PM
LightSpeed VCT
Rapides Regional Medical ...
(f)
AP PORT @1403 PM
PACU

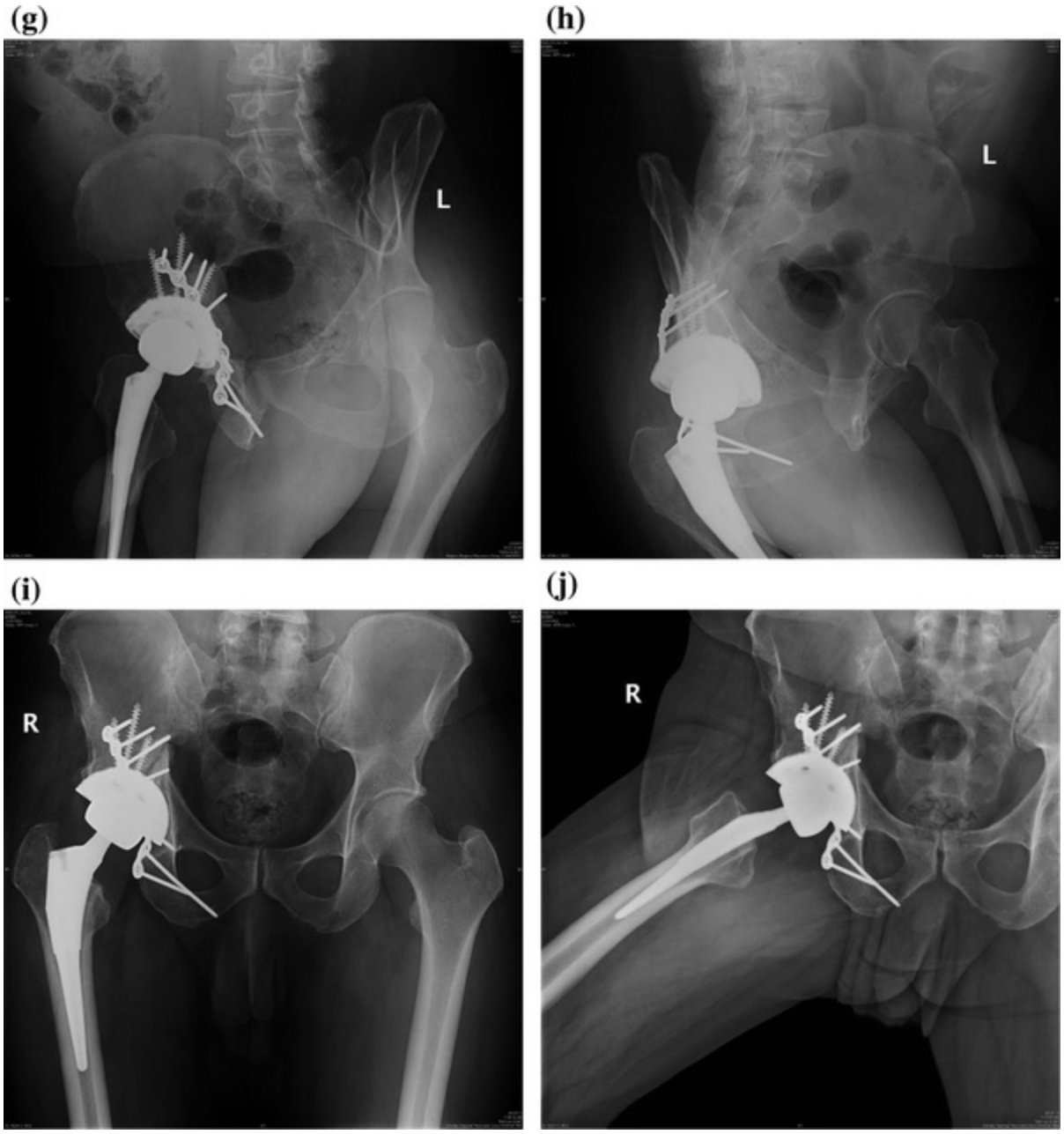
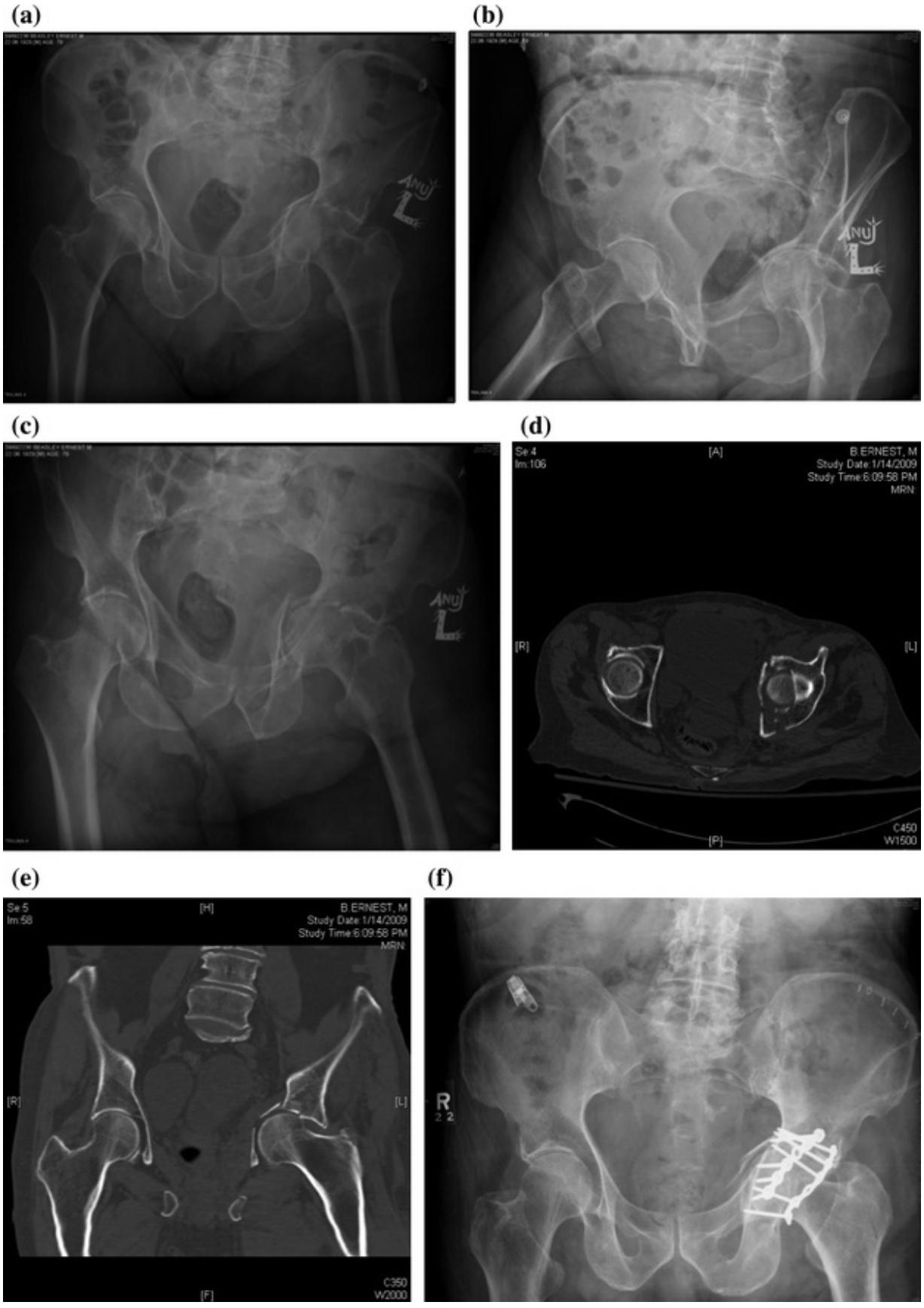



***Fig. 8.7*** **a**, **b** Anteroposterior (AP) of the pelvis at the time of the accident 5 years before presentation pre- and postreduction of the hip dislocation. **c** AP pelvis at presentation with a complete nonunion and end-stage osteoarthritis. **d**, **e** Axial CT showing a complete nonunion of the transverse fracture without any of the cuts showing bridging and the end-stage osteoarthritis. **f** AP pelvis postsingle-stage open reduction internal fixation and hip arthroplasty through a Kocher-Langenbeck approach. **g–j** Six-month follow-up with both 45° oblique views, and AP and lateral of *right hip* showing solid fixation and patient without pain


(a)
(b)
(c)
(d)
Se:4
Im:106
[A]
B.ERNEST, M
Study Date:1/14/2009
Study Time:6:09:58 PM
MRN
[R]
[L]
[P]
C450
W1500
(e)
Se:5
Im:58
[H]
B.ERNEST, M
Study Date:1/14/2009
Study Time:6:09:58 PM
MRN
[R]
[L]
[F]
C350
W2000
(f)
R

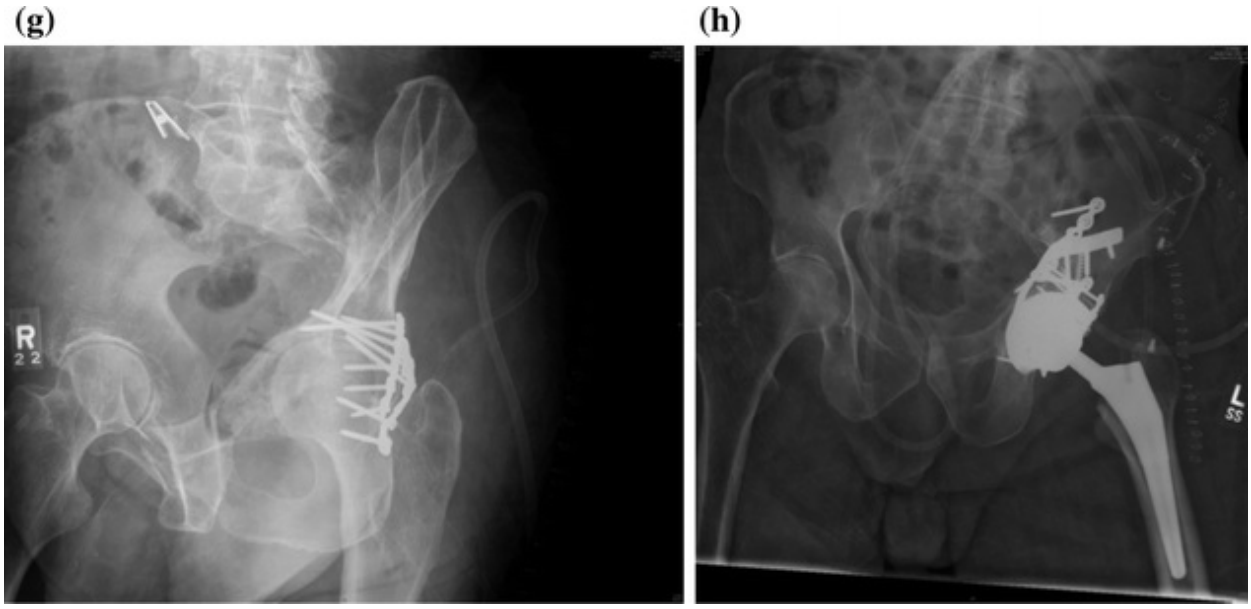



***Fig. 8.8*** **a–c** Anteroposterior (AP) obturator oblique and iliac oblique X-ray, respectively, following a fall from a standing position. **d** Axial CT showing the medial dome impaction and central migration of the head. **e** Coronal CT reconstruction showing the medial dome impaction and central migration of the head. **f**, **g** AP and obturator oblique X-ray, respectively, showing the medial subluxation of the femoral head due to a nonunion. **h** Postoperative AP X-ray after repair of nonunion and arthoplasty

## 8.2.1 Deformity

Acetabular deformity and/or hip protrusio causes symptoms similar to what is seen in the pelvis: gait abnormalities, sitting imbalance, and limb length discrepancy (i.e., shortening of a transverse fracture). Furthermore, protrusion of the femoral head centrally will cause a significant decrease in motion (see Fig. 8.8). The difference between pelvic nonunions and acetabular nonunions is that nonunions of the acetabulum require early diagnosis to prevent the development of severe arthritis after which the hip will no longer be salvageable (see Figs. 8.7 and 8.8). Radiographic analysis is critical (see below) to determine the type of the fracture present and the amount and direction of displacement.

## 8.2.2 Genitourinary System

Genitourinary symptoms in acetabular nonunions can present similar to what is seen in pelvic nonunions. Besides the symptoms listed in the pelvic section, a separate cyst of urine can be found where a rami fracture has

perforated the bladder.

### 8.2.3 Neurologic Injuries in Acetabular Nonunions

The neurologic injuries associated with acetabular fractures are somewhat different than neurologic injuries in pelvic fractures and dislocations. The predominant nerve injury in acetabular fractures is the common peroneal tract of the sciatic nerve causing a foot drop. Additional nerve injuries include the superior gluteal nerve (abductor weakness) and obturator nerve (adductor weakness and numbness of the inner thigh). Rarely, the femoral nerve may be injured. A preoperative examination will often identify partial or complete muscle weakness. In acetabular nonunions, complete knowledge of the anatomy is required to prevent a retractor iatrogenic nerve injury. Mayo et al. described postoperative nerve palsies following correction of acetabular malunion in six percent of their cases (three percent superior gluteal and three percent sciatic) [17].

### 8.2.4 Patient Expectations

For acetabular nonunion patients, the results are not as good as those seen in pelvic nonunions, and the degree of difficulty and the need for precise anatomic reduction is an order of magnitude greater than that discussed for the pelvis. Nothing less then a perfect reduction of the acetabulum is acceptable, and even in experienced hands, in patients with nonunions of the acetabulum, 58% of the patients go on to develop arthritis [17]. Timing is also an important factor, with 57% good to excellent results if operated on within three weeks of the injury, and 29% good to excellent results if the delay exceeded 12 weeks from the time of injury. Once again, significant discussion is necessary prior to making a decision for surgery. The patient must have realistic goals and an understanding of the risks and benefits of surgery. The patient needs to understand preoperatively that success is limited and that THA is likely in the intermediate or long term.

### 8.2.5 Radiographic Assessment

The radiographic analysis of acetabular nonunions uses the same radiographs as in the pelvic section with the addition of MRI to look for cartilage damage and avascular necrosis. Often, these injuries have areas of malunions and

nonunions in the same fracture line; that is, in a transverse fracture, the fracture heals supero-medially and has a nonunion postero-inferiorly. To make the diagnosis of a nonunion (see Fig. 8.7), no bridging bone is found anywhere along the fracture line. The patients with partial unions typically do not have pain from the nonunion site but due to hip arthritis. Like the pelvis, the patterns of displacement of certain fracture types have been determined. The way a both column or T-type fracture displaces is somewhat consistent. The anterior and posterior columns open up like “saloon doors” as the head pushes medially (see Fig. 8.8). Drawing the fracture on a model is mandatory to determine the rotation of the broken pieces that either need to be released (nonunions) or osteotomized and released (malunions) in order to obtain anatomic reduction. For instance, transverse fractures have two axes of deformity. The inferior piece rotates around an axis that travels down the symphysis pubis with greater displacement posterior versus anterior. The inferior transverse fracture rotates around a second axis from that lies on a line from the symphysis pubis to the fracture site through the posterior column as the head pushes medially (see Fig. 8.7).

The edges of nonunions are typically seen radiographically as hypertrophied bone. Narrowing of greater than 50% of the dome articular surface is an indication for THA. Wear in other areas of the joint may be well tolerated. Interestingly, some both-column fractures detach the whole articular surface. The femoral head remains congruent with the dome despite widening medially between the two columns and medial translation of the entire joint. Medial widening up to one centimeter may be well tolerated; therefore, treating these malunions conservatively may be the best option. As mentioned, transverse fractures seem to have the highest incidence of nonunions whether they are operated on or not (see Fig. 8.7). As mentioned, Letournel reported on a 0.7% nonunion rate in nonoperatively treated acetabulum fractures with most of these being transverse fractures or the transverse component of a transverse posterior wall acetabular fracture (7 total: 2 posterior column, 3 transverse, 1 transverse posterior wall, and 1 posterior column of a both-column) [18]. Even those that heal can still have the inferior part of the posterior column fracture line remain unhealed. Letournel described this as a nonunion/malunion and had 14 of these, including 3 posterior column fractures, 1 anterior column, 2 T-shaped, 2 transverse and posterior wall, and 6 both-column fractures [18]. The key to these is the resection of the boney bridge to be able to correct the deformity.

This may be due to the synovial fluid tracking into that area, preventing the bone from healing inferiorly. There are not enough cases to say definitively whether operatively treated acetabulum fractures have more or less percentage of nonunions. Treatment of trans-tectal transverse and transverse posterior wall without surgery reliably leads to healing. Sometimes a transverse nonunion is associated with the psoas tendon being caught in the transverse fracture line in the psoas grove. In the handful of cases that were operated on by the author or were referred, a few similarities existed. The patients were not compliant with touchdown weight bearing for 8 weeks, had a transverse or "T"-type fracture, had a slight deformity, and eventually had THA (see Figs. 8.7 and 8.8).

## 8.2.6 Treatment

The indications for surgery include displaced acetabular nonunions that meet the indications for acute surgery (i.e., incongruence at the femoral head or > 2-mm step off in the weight-bearing dome). Pain without deformity is also an indication for surgery [19]. Age has a limited role in the decision pathway. If the patient has physiologically good bone and is active, it is prudent to try to save his hip. As mentioned, all of the patients seen in our institution have a combination nonunion with some deformity and require resection of fibrous tissue and bone to achieve anatomic reduction. The key is the quality of the cartilage. One can reconstruct the hip if there is at least 50% of the dome cartilage intact. If there is already complete loss of articular cartilage, the surgeon must ask him/herself whether a successful total hip can be performed with or without an osteotomy of the acetabulum to correct the deformity. In the case of nonunion, the nonunion always has to be stabilized first prior to the THA or there will be a significant increase in cup loosening (see Figs. 8.7 and 8.8). If the hip is out of the socket either medially or laterally (see Fig. 8.6), or the patient has not started weight bearing, usually the cartilage is preserved and the joint can be salvaged.

The treatment of the nonunion site is similar to the treatment described in the pelvic nonunion section. Preoperative laboratory work includes possible diagnosis of other contributing diseases such as nutritional, endocrine, or infectious abnormalities. WBC, ESR, and CRP are routinely ordered. Elevation of these tests may prompt a two-stage revision; the first stage involves an open debridement with multiple site biopsies for cultures and alpha defensin, removal of previously placed hardware, and antibiotic

polymethyl methacrylate (PMMA) beads (typically, use one bag of tobramycin [1 g] cement with an additional 3 vials of vancomycin [1 g/vial, 3 g total] and 2 vials of tobramycin [1.2 g/vial, 3.4 g total]). Culturing the nonunion site; burring to bleeding cortical bone; drilling through potential end caps, stimulating cancellous bleeding; bridging the nonunion site with cancellous bone; and covering with healthy bleeding muscle are the key concepts for obtaining union. Importantly, all callous or healed bone that is preventing anatomic reduction is removed.

### 8.2.7 Acetabular Nonunions

Simple acetabular nonunions are treated in a similar manner to pelvic nonunions. Unfortunately, the more common scenario is that by the time a diagnosis of acetabular nonunion is made, the patient already has complete loss of the articular surface (see Figs. 8.7 and 8.8). It then becomes imperative to treat and stabilize the nonunion prior to doing a THA; this can be done at the same setting. If THA is performed without stabilization of the nonunion, >80% of these cases will have loosening of the acetabular component. Anatomic reduction of the joint is required if salvaging the acetabulum (see Fig. 8.6). If there is significant protrusio of the femoral head within the pelvis, even in the face of doing a THA, osteotomies and reduction of the two columns to keep the THA out of the pelvis are performed.

The choice of approach in nonunions is similar to the acute setting: The Kocher-Langenbeck (KL) for nonunions of the posterior column and wall, the ilioinguinal for the anterior wall and columns, and the extended iliofemoral (EIF) for all other fractures that require the surgeon to be on both sides of the acetabulum. Sequential approaches can be used for these more complex patterns in the acute setting but do not work well in established nonunions if some reduction will be required. Both sides need to be freed up prior to reduction, so simultaneous approaches is an option, but EIF is preferred to get to both sides of the bone, particularly in transverse and “T”-type fractures. The EIF is used to salvage the hip joint but is not used if a THA will be performed. Either a KL [20] or an anterior Smith-Peterson approach can give enough exposure for plating the nonunion prior to performing a THA (see Figs. 8.7 and 8.8). In all cases, the fibrous tissue is removed from the fracture site, including intra-articularly through a capsulotomy. The edges of the nonunion can be sclerotic and need to be freshened up so that there is bleeding from both ends, and cancellous bone

graft is packed into the gap. Intra-operative traction with subluxation/dislocation of the hip allows the intra-articular nonunion to be corrected, and stabilization is performed with standard compression plate techniques [21]. Intra-articular osteotomies are reserved for when the acetabulum is to be salvaged (see Fig. 8.6). Displaced nonunions require mobilization of the fragments similar to acetabular malunions, with direct intra-articular visualization. If greater than 50% of the dome has osteoarthritis, a THA is performed, usually without mobilizing the fractured fragments but treating the nonunion site and stabilizing the nonunion with a plate without attempting an anatomic reduction prior to performing THA. The exception to this, as mentioned, is where a protrusion of the femoral head into the pelvis requires lateralization of the head and reduction of the two columns medially with plate stabilization prior to doing a THA.

## 8.2.8 Malunions and Displaced Nonunions of the Acetabulum

Most of the acetabular nonunions have some displacement that requires reduction if salvage is desirable, and are complicated by the fragments being healed or scarred down in the wrong position (see Fig. 8.6). Complete release of the bone and associated soft tissue is required for anatomic reduction of the joint. Interestingly, bone healing is much more rapid than cartilage healing, so osteotomies through old malunions can be visualized more easily intra-articularly than extra-articularly. Also, reduction can be visualized intra-articularly to ensure congruency. Discussion of acetabular malunions is beyond the preview of this chapter.

## 8.2.9 Acetabular Nonunion Case Discussions

**Case 1. Nonunion of the acetabulum** (see Fig. 8.6)

A 65-year-old initially presented with an open anterior column acetabular fracture. Surgery was delayed until 3 months postinjury due to the condition of the soft tissues. Patient had developed a nonunion of the acetabulum, which underwent single-stage open reduction internal fixation through an ilioinguinal approach. Patient ambulating without pain and no joint narrowing one year postop.

**Case 2. Nonunion of a juxtatectal transverse acetabulum leading to degenerative hip arthritis** (see Fig. 8.7)

A 49-year-old presented 5 years after a transverse acetabulum fracture that was treated conservatively. Patient was never compliant with the previous touchdown weight-bearing recommendations. Patient was found to have a symptomatic nonunion and end-stage osteoarthritis based on radiographic examination. Patient underwent single-stage repair of the nonunion and THA. Patient did well and his pain resolved. Unfortunately, the patient was trying to outrun the police on a motorcycle, and at the time of this writing was in prison.

**Case 3. Nonunion of a transtectal "T"-type acetabular fracture** (see Fig. 8.8)

A 79-year-old fell from standing position suffering a "T"-type acetabulum with medial dome impaction and central migration of the femoral head. Patient underwent operative repair of acetabular fracture, but there was a failure of reduction of the quadrilateral surface. Patient with pain and discomfort in left hip with established nonunion. Patient underwent an anterior Smith-Peterson approach, fixation of the anterior and posterior columns of the "T", and an anterior THA. A buttress plate over the brim onto the quadrilateral plate was placed to prevent protrusion (custom-made lone star plate) and buttress plate along the pelvic brim buttressing the lone star plate using the femoral head as a medial bone graft and performing a THA. Patient 2 years out walking without a walking aid, no groin or thigh pain, but with some trochanteric pain.

---

## 8.3 Conclusion

Stabilization of nondisplaced pelvic nonunions, especially posteriorly, has been proven to be successful in returning patients to their preinjury status [11]. Treatment of acetabular nonunions is not as successful due to pain coming from the hip joint in the form of osteoarthritis or avascular necrosis. Operative correction of nonunions of the acetabulum can give excellent results if the joint does not already have significant damage. The results of surgery in the setting of nonunion are not as good as those of acute treatment of acetabular fractures or pelvic ring injuries. Once the nonunion has established itself and chronic symptoms develop, the probability of surgical

reconstruction returning the patient to his or her preinjury status is decreased. Also, the rate of complications is higher for late surgical treatment [3, 17]. Prevention by acute anatomic reduction and internal fixation of unstable pelvic injuries and anatomic articular reductions in acetabular fractures is the best treatment for pelvic and acetabular nonunions.

---

# References


1. Semba RT, Yasukawa K, Gustilo RB. Critical analysis of results of 53 Malgaigne fractures of the pelvis. J Trauma. 1983;23(6):535–7.
[Crossref][PubMed]

2. Dickson KF, Matta JM. Surgical reduction and stabilization of pelvic nonunions and malunions. Paper presented at: The 63rd Annual Meeting of the American Academy of Orthopaedic Surgeons; 1996; Atlanta, Georgia.

3. Matta JM, Dickson KF, Markovich GD. Surgical treatment of pelvic nonunions and malunions. Clin Orthop Relat Res. 1996;329:199–206.
[Crossref]

4. Tile M, editor. Fractures of the pelvis and acetabulum. Baltimore: Williams and Wilkins; 1984.

5. Huittinen VM, Slatis P. Nerve injury in double vertical pelvic fractures. Acta Chir Scand. 1972;138(6):571–5.
[PubMed]

6. Hundley J. Ununited unstable fractures of the pelvis. Proceedings of the 33rd Annual Meeting of the American Academy of Orthopaedic Surgeons. J Bone Joint Surg Am. 1966;46A.

7. Letournel E. Diagnosis and treatment of nonunions and malunions of acetabular fractures. Orthop Clin North Am. 1990;21(4):769–88.
[PubMed]

8. Matta JM, Saucedo T. Internal fixation of pelvic ring fractures. Clin Orthop Relat Res. 1989;242:83–97.

9. Kellam JF. The role of external fixation in pelvic disruptions. Clin Orthop Relat Res. 1989;241:66–82.

10. Hsu JR, Bear RR, Dickson KF. Open reduction of displaced sacral fractures: techniques and results. Sacral fractures. Orthopedics. 2010;33(10):730.
[PubMed]

11. Pennal GF, Massiah KA. Nonunion and delayed union of fractures of the pelvis. Clin Orthop Relat Res. 1980;151:124–9.

12. Matta JM, Tornetta P 3rd. Internal fixation of unstable pelvic ring injuries. Clin Orthop Relat Res.

1996;329:129–40.
[Crossref]

13. Cole JD, Bolhofner BR. Acetabular fracture fixation via a modified Stoppa limited intrapelvic approach. Description of operative technique and preliminary treatment results. Clin Orthop Relat Res. 1994;305:112–23.
[Crossref]

14. Beaule PE, Antoniades J, Matta JM. Trans-sacral fixation for failed posterior fixation of the pelvic ring. Arch Orthop Trauma Surg. 2006;126(1):49–52.
[Crossref][PubMed]

15. Griffin DR, Starr AJ, Reinert CM, Jones AL, Whitlock S. Vertically unstable pelvic fractures fixed with percutaneous iliosacral screws: does posterior injury pattern predict fixation failure? J Orthop Trauma. 2006;17(6):399–405.
[Crossref]

16. Frigon VA, Dickson KF. Open reduction internal fixation of a pelvic malunion through an anterior approach. J Orthop Trauma. 2001;15(7):519–24.
[Crossref][PubMed]

17. Mayo KA, Letournel E, Matta JM, Mast JW, Johnson EE, Martimbeau CL. Surgical revision of malreduced acetabular fractures. Clin Orthop Relat Res. 1994;305:47–52.

18. Letournel E, Judet R, editors. Fractures of the acetabulum. 2nd ed. Berlin: Springer; 1993.

19. Zura RD, Kahler DM. A transverse acetabular nonunion treated with computer-assisted percutaneous internal fixation. A case report. J Bone Joint Surg Am. 2000;82(2):219–24.

20. Weber M, Berry DJ, Harmsen WS. Total hip arthroplasty after operative treatment of and acetabular fracture. J Bone Joint Surg. 1998;80-A(9):1295–1305.

21. Mohanty K, Taha W, Powell JN. Non-union of acetabular fractures. Injury. 2004;35(8):787–90.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_9

# 9. Proximal Femur Nonunions

Roman A. Hayda[1]

(1) Rhode Island Hospital, Brown University Warren Alpert School of Medicine, 2 Dudley St Suite 200, Providence, RI 02905, USA

**Roman A. Hayda**
**Email:** roman_hayda@brown.edu

**Keywords** Hip – Intertrochanteric – Subtrochanteric fracture – Nonunion – Valgus osteotomy – Neglected hip fracture

Given the frequency of fractures of the proximal femur, the active fracture surgeon is likely to encounter patients with nonunion. Fractures of the proximal femur, to include the femoral neck, intertrochanteric, and subtrochanteric regions, are common, particularly in the elderly, with over 310,000 occurring annually in the USA alone [1]. In adults, almost all fractures in this region are treated with surgery. While nonunion is reported in all patterns of proximal femur fracture, it is most common in the femoral neck.

In the younger patient, proximal femur fractures occur with high-energy mechanisms, while in the elderly, low-energy falls are sufficient to cause displaced fractures with comminution. With the exception of the elderly patient in which arthroplasty is performed for displaced femoral neck fractures, internal fixation of proximal femur fractures is performed with intramedullary devices, sliding screw and side plate, or cannulated screws depending on the site of the fracture and surgeon preference. When proximal femur fractures are appropriately fixed, union typically results in resolution of pain with restoration of ambulatory capacity.

Nonunion or impending nonunion should be suspected when there is loss of reduction or there is continued pain. Any hardware failure or loss of reduction should prompt the clinician to initiate evaluation and possible surgical intervention. Physical examination and careful evaluation of radiographs with comparison to initial postoperative films provide valuable clues to early detection of nonunion. Defining the timing and adequacy of union may be difficult in relying on radiographic and clinical signs [2]. Scoring of progression of union on anteroposterior and lateral films in serial films using the RUSH (Radiographic Union Score for Hip) can be predictive of union and provides an organized method of assessment. Bhandari et al. demonstrated in a series of femoral neck and intertrochanteric fractures higher interobserver reliability in assessing fracture union [3, 4]. In select cases, the use of a CT scan can define the presence of bridging bone particularly when hardware obstructs osseous anatomy. In most cases, bridging bone in the presence of hardware leads to continued consolidation. Magnetic resonance imaging and other modalities can help detect avascular necrosis, infection, and other pathology .

When the diagnosis of nonunion is made, the clinician should perform an organized evaluation the patient. The radiographs and operative report should be evaluated for mechanical factors that may have contributed to the development of nonunion. These mechanical factors are specific to the region being treated and are related to the type of fixation device. The clinician should assess whether the device was appropriate for the type of fracture and whether it was appropriately implanted. Secondary questions then arise whether the surgical revision should employ a similar device or alternate implants are required. Additionally, the surgeon must consider if special extraction devices or techniques are required especially in the face of broken hardware. In select cases, the surgeon may elect to leave some of the previous implant in place when extraction would pose risks without additional benefit.

Biologic factors cannot be ignored when evaluating a patient with a nonunion [5, 6]. Failure to take these into consideration may lead to continued failure after considerable effort on the part of the patient and the surgeon. Biologic factors may be local as well as systemic. Local factors that contribute to nonunion are devascularization of the bone whether secondary to the injury or surgical insult, infection, and even bone necrosis due to irradiation (Fig. 9.1). Systemic factors include malnutrition, smoking , and diseases such as diabetes and chronic illness particularly those treated with

corticosteroids and other anabolic suppressants. Endocrine and vitamin deficiencies have also been implicated as contributors to nonunion and should be assessed[5]. Whether the causes are local or systemic, the surgeon and the patient should make an effort to mitigate the biologic factors that contribute to nonunion .


***Fig. 9.1*** Subtrochanteric nonunion in irradiated bone with failed hardware

A careful history that evaluates systemic conditions and medications must be performed. The use of nonsteroidal anti-inflammatory drugs has been implicated in nonunion [7]. The inhibition of the cyclooxygenase pathway by nonsteroidals impairs the recruitment of cellular elements critical for the initiation of bone healing. Although intermittent use does not seem to impair healing, consistent dosing has been shown to delay union in animal models and in clinical series [8]. Screening for infection with a complete blood count, erythrocyte sedimentation rate, and C reactive protein may raise the index suspicion for infection although intraoperative culture is the optimal test. Nutritional status can be evaluated with albumin level and total lymphocyte count. Screening for vitamin D deficiency and other endocrine abnormalities should be considered [9]. Alternatively, referral to an internist or endocrinologist may assist in evaluation of metabolic contributors to

nonunion.

Bisphophonates have been credited with reducing the risk of osteoporosis-related fractures and are commonly prescribed in the elderly population at risk these low-energy fractures. However, they have been implicated in atypical fractures of the femur, especially in the subtrochanteric area [10]. Their mechanism of action for reducing osteoporosis is by limiting osteoclastic resorbtion of bone. However, in so doing they may limit the critical function of bone remodeling contributing to the formation of atypical stress fractures of the femur often after 5 or more years of treatment, a time frame where continued use offers no added benefit. At present, there is no evidence that it impairs fracture union. However, authors have noted that these atypical fractures are prone to delayed union [10]. The role of anabolic agents such as synthetic parathyroid hormone to promote union especially in a patient with osteoporosis is not yet defined [11].

The surgical decision-making is specific for the femoral neck, intertrochanteric, and subtrochanteric regions. However, in developing the treatment plan for a nonunion, the surgeon must take into account not only mechanical factors but also biologic factors, local and systemic, as well as the patient's functional needs. Balancing these issues with the potential risks of surgery will lead to a reasonable result. For instance, although preservation of native anatomy especially the femoral head is preferred, endoprosthetic replacement for failed treatment of femoral neck and intertrochanteric fractures may be the optimal treatment even in a patient under 65 after careful consideration of the expected treatment course and potential risks. In this discussion, the surgeon should not only consider the quickest or most straightforward treatment option but also keep in mind the consequences of failure of the chosen course. With prosthetic replacement, the potential for dislocation, periprosthetic fracture, and the devastating effect of infection must be taken into account. The patient and surgeon must maintain awareness of the potential for such outcomes. In select cases, Girdlestone resection arthroplasty with its attendant limitations may remain the best treatment option for problematic proximal femur nonunion especially in the face of recalcitrant infection or a patient with significant comorbidities or low functional demand [12].

## 9.1 Femoral Neck

Among fractures of the proximal femur, the femoral neck is the most prone to nonunion. Although one series reported a nonunion rate of 59% and avascular necrosis rate of 86% [13], most authors have reported nonunion rates as of 0–30% in young patients [14–16]. Union is expected within three months of fixation although failure can be seen very early following fixation particularly in inadequately fixed fractures or noncompliant patients. These early failures should be considered as a nonunion even though 3 or 6 months have not passed from the time of fixation. Even surgically repaired nondisplaced fractures have been noted to proceed to nonunion in up to 8% of cases indicating the multifactorial nature of nonunion [17, 18]. The risk of fixation failure in elderly patients with displaced fracture has led to the recommendation for arthroplasty to avoid multiple procedures and prolonged disability [19]. The propensity for nonunion can be attributed to biologic and mechanical factors. The femoral neck is intracapsular and lacks periosteum, an important contributor to fracture healing. The blood supply enters through the circumflex vessels, which may be disrupted by the injury and surgery [20, 21]. Not only may vascular disruption impair fracture healing, but it may also contribute to avascular necrosis.

In femoral neck fractures, the development of a nonunion is usually heralded by loss of reduction especially varus collapse. Varus is particularly unstable, leading to continued collapse and ultimately failure. In this instance, the surgeon should consider early revision. Shortening along the axis of the cannulated screws or screw and side plate is often seen and may result in union. However, progressive shortening without evidence of stabilization is a sign of nonunion. Ultimately, the fixation device may end up within the hip joint and failure requiring revision (Fig. 9.2). Although any varus collapse should alert the surgeon for potential revision, Alho et al. [22] suggested specific radiographic criteria of failure. High rates of revision were associated with a change in reduction of 10 mm, change in screw position by 5%, or screws backing out by 20 mm .

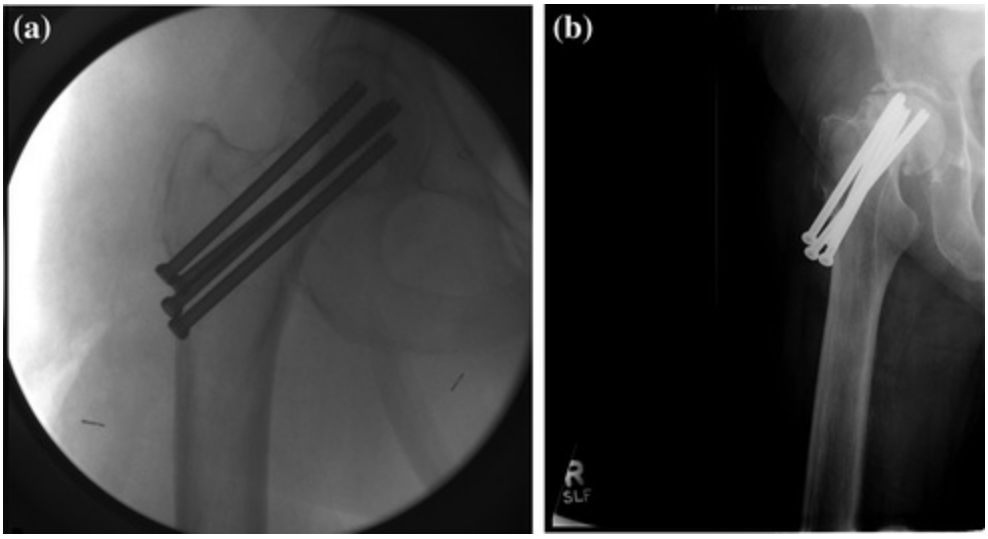



***Fig. 9.2*** Progressive failure of femoral neck "valgus impacted" fracture with hardware encroaching the joint. Note failure into varus and screws backing out **(a, b)**

Mechanically, the femoral neck is subject to substantial forces while depending on its trabecular structure for mechanical strength [21]. Certain patterns of injury, namely the vertically oriented fracture, are prone to nonunion because of poorly controlled shear forces [23]. The Pauwels classification scheme based on the angle of the fracture line to the horizontal defines 3 types: low, < 30 degrees; intermediate, between 30 and 60 degrees, and high, greater than 60 degrees (Fig. 9.3). Regardless of fracture pattern, fixation strategies that do not engage subchondral bone will have limited ability to control this short bone segment. The optimal treatment device has not been definitively established with proponents of sliding screw and side plates and those favoring cannulated screws [24]. At present, data suggest that sliding screw and side plate devices may be less prone to failure but may have a higher rate of avascular necrosis. In the case of cannulated screws, placing the device adjacent to the cortex improves fracture stability by increasing screw spread and by taking advantage of cortical bone. Placing cannulated screws more centrally has been compared to placing them in an "empty can" in the elderly patient contributing to fixation failure [25] .

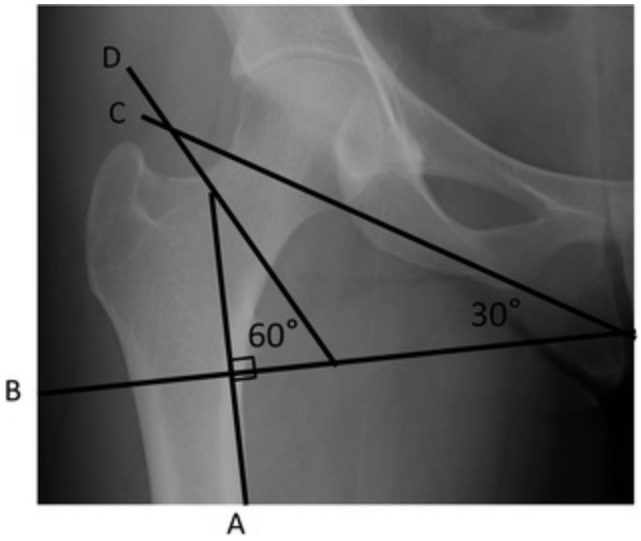



***Fig. 9.3*** Pauwels classification: *Line A* is drawn in line with the femoral shaft; *line B* is drawn perpendicular to *line A*. *Lines C* and *D* represent possible femoral neck fracture lines. The angle created by *line C* or *D* to the perpendicular determines the Pauwels type: Pauwels 1, <30 (*line C*); Pauwels 2, 30–60; Pauwels 3, >60 (*line D*)

The quality of reduction is a determinant of stability [14]. Fractures with poor reduction, especially varus, have impaired mechanics and are prone to failure. In the young patient where retention of the hip is critical, the surgeon should strive for an anatomic reduction. Therefore, open reduction is advocated since closed reduction may not consistently achieve this goal. The exception to this rule is a valgus impacted fracture, usually seen in the elderly patient. This fracture pattern is considered stable and is fixed in situ.

## 9.1.1 Surgical Options

Options for revision in femoral neck fractures nonunion are arthroplasty or revision fixation with or without osteotomy. In the older patient, arthroplasty is the primary form of treatment, while a younger patient should be considered for revision fixation. There is no particular age at which revision fixation or arthroplasty should be done. Marti successfully performed osteotomies up to age 70, while arthroplasty surgeons have advocated replacement at age 60–65, especially as arthroplasty implants have improved and patients are less tolerant of lengthy rehabilitation [16, 19]. Careful consideration of expected clinical course and potential complications should be used in choosing treatment (Table 9.1) [26–34]. Revision fixation requires a healing period prior to the resumption of activities, which may last 3–

6 months and does not guarantee success. However, it allows for preservation of the native hip joint. Even the presence of low-grade avascular necrosis is not a contraindication to hip preservation [15, 26, 31]. In the uncommon instance of biologic failure, the healing environment can be augmented by autogenous or vascularized bone graft. The Meyers graft [35, 36] which utilizes the bony insertion of the quadratus femoris placed into the posterior femoral neck or a vascularized fibula as advocated by LeCroy et al. [31] can provide the stimulus to achieve union.

***Table 9.1*** Comparative table of treatment of femoral neck nonunion

| Study | Number | Age | Avg time from index surgery (mos) | Technique | Follow-up | % Union | Complications | Outcome |
|---|---|---|---|---|---|---|---|---|
| Marti et al. [26] | 50 | 19–76 | 9 (2−60) | Osteotomy; 120° double-angle blade plate, local bone graft | 7.1 (3–13) years | 86 | 3 UTI, 3 DVT, 1 deep infection (ankylosis) 14% THR (3 nonunion, 3 AVN, 1 failed hardware (Etoh abuse) | Harris score 91, Merle D'Aubigne 78% good to excellent; AVN in 22 pts 3 requiring THR, |
| Anglen et al. [27] | 13 | 34 (18–59) | 21 (4–54) | Osteotomy; 120 deg double-angle plate, local bone graft | 2 years (0.7–3.5) | 100% | AVN in 2 pts-THR | Harris 93, no pain 7/8 retur to work, avg LLD 1.5 cm |
| Shoenfeld et al. [28] | 4 | 35 (24–42) | 6 | Opening/closing wedge osteotomy; cannulated 130–140° sideplate, local bone graft | 1.25 (0.25–2.3) years | 100% | None | Merle D'Aubigne 16.5; cane 1 p no LLD but sl Trendelenburg 2/4 |
| Hartford et al. [29] | 8 | 46 (30–65) | 10 | Closing wedge, cannulated 135–155 sideplate, local bone graft | 2 years | 88% | Unrelated death at 4 months | Harris hip score 73, cane 1 pt. Trendelenburg 4/7 |
| Said et al. [30] | 17[a] | 37 (18–49) | | 130 single-angle blade, closing wedge | 3.5 (1–5) | 97%[a] | AVN 5 cases 1 THR for nonunion[a] | 61% pain free LLD < 0.5 cm |
| Lecroy et | 22 (all with | 28.7 | 18.3 | Free fibula, | 7 years | 90% | 2 nonunion (1 | 60% |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| al. [31] | AVN at presentation) | | | cannulated screws | | | iliac crest graft, 1 Meyers graft); 4 hardware removal for articular penetration | progression of AVN but 90% retained native hip, Harris score 78.9 |
| Jun et al. [32] | 26 | 41 (22–60) | 12 | Free fibular in anterior trough | 3.5 (1–5) years | 100% | 1 infection, 1 AVN (THR) | Harris score 8 |
| Elgafy et al. [33] | 17 | 46 (24–58) | 8 (4–22) | 13 autograft non-vascularized fibula; cannulated screws | | 69% | 4 nonunion-arthroplasty, 2 ankle pain | |
| | | | | 6 allograft fibula; cannulated screws | | 33% | 3 arthroplasty, 1 autograft fibula | |
| Wu et al. [34] | 26 | 38 (17–60) | 1.4 (0.8–2) | 17 osteotomy with sliding screw | >2 years (2–6) | 100% | 2 AVN, 1 nonunion osteotomy | Osteotomy indicated if shortening was >1.5 cm |
| | | | | 9 sliding screw only | | 100% | None | |

*AVN* avascular necrosis; *UTI* urinary tract infection; *THR* total hip replacement; *LLD* limb length discrepancy

[a]Series included 19 acute fractures not reported separately

## 9.1.2 Revision Fixation

Mechanical failure is most common with failure of fixation and development of a deformity on radiographic examination. In femoral neck nonunion attributed to mechanical failure, the principal decision is whether osteotomy will improve the mechanical environment. On analysis of the nonunion, the new construct must assure adequate stability in the face of physiologic forces to allow for healing. In the instance of a poorly placed initial construct, revision fixation alone may be considered. Cannulated screws in the most optimal position or a screw and side plate device with screw augmentation may allow for healing. Wu, in a mixed series with and without osteotomy, reported 100% healing in the group treated with a screw and side plate alone [34].

### 9.1.3 Valgus Osteotomy

In high angle fractures often seen in the younger patient, an osteotomy is indicated when a nonunion develops. These nonunions are often associated with varus and shortening. Properly executed osteotomies result in union rate of 70–100% [26–29, 34]. As described by Pauwels, the osteotomy converts a vertically oriented fracture with shear to a more horizontal orientation creating compressive forces promoting union. In considering an osteotomy, the duration of healing along with alterations of anatomy must be considered. These alterations of shortening and a lesser abductor moment are generally well tolerated but should be discussed with the patient.

In this procedure, the nonunion is not exposed directly. A blade plate is most often used to compress the osteotomy, but a screw and side plate may also be used [28, 29, 34]. A blade plate is technically more demanding but offers proven stability while requiring only a narrow corridor of bone.

The critical step in performing the intertrochanteric osteotomy is preoperative planning (Fig. 9.4). The concept is to convert the angle of the fracture to less than 30° by performing an intertrochanteric osteotomy. Good anteroposterior pelvis and lateral hip views are required. The angle of the fracture to the horizontal is measured. Due to leg rotation, the precise angle may be hard to measure, but may be facilitated by measuring it to a line perpendicular to the femoral shaft [27]. A closing wedge osteotomy is planned to correct the angle of the fracture to less than 30°. In the case of a fracture angle of 70°, a wedge of 40° is planned.

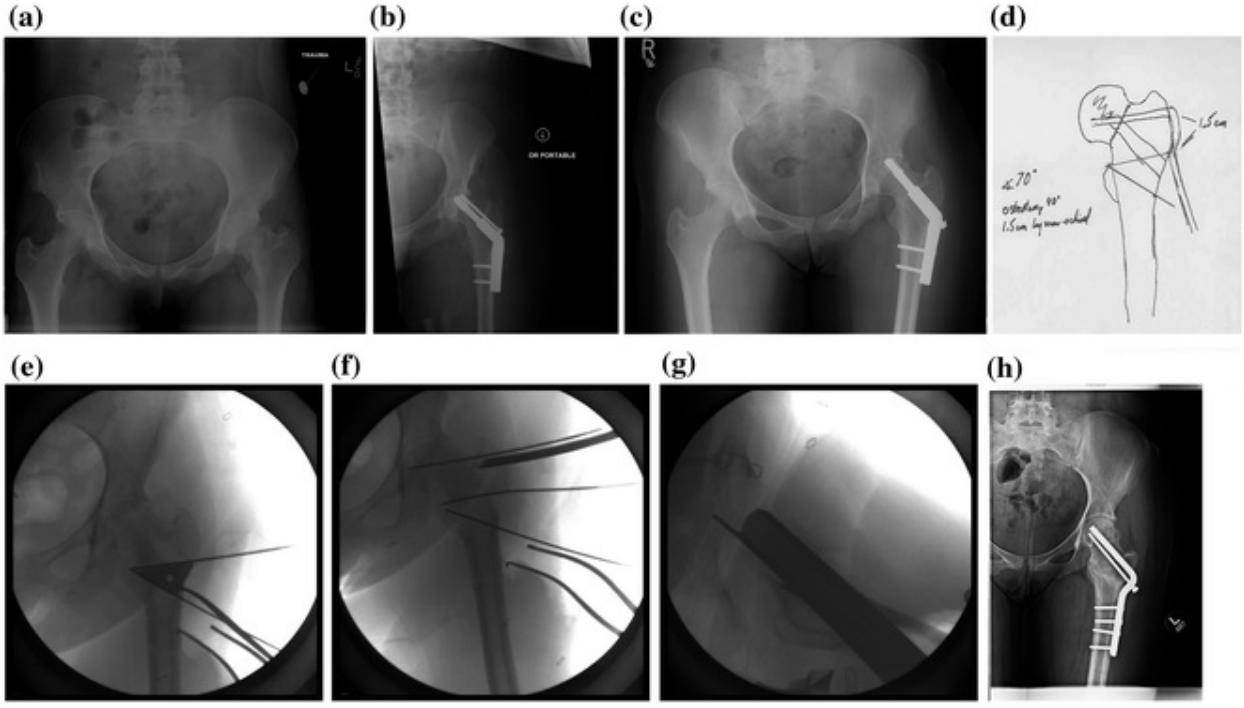



***Fig. 9.4*** Corrective osteotomy of femoral neck nonunion: **a** injury radiograph note vertical angle; **b** initial fixation; **c** nonunion, note varus, and shortening; **d** surgical template—fracture angle is 70°, a 40° correction will result in an angle of 30°, and osteotomy is planned 1.5 cm distal to projected blade insertion site; **e** intraoperative templating; **f** placement of chisel anteroposterior view; **g** placement of chisel lateral view; **h** healed nonunion and osteotomy

The position of the blade plate that allows for adequate fixation is templated. Inevitably, the biomechanics of the hip are altered with some shortening of the neck and medialization of the shaft. Some modifications of the osteotomy have been proposed that can minimize the anatomic alterations. A combination of opening and closing wedge osteotomy or translating the shaft laterally along the osteotomy are methods used to limit biomechanical alterations but may carry a theoretic risk of nonunion.

The intertrochanteric osteotomy is executed on a radiolucent table. The surgeon must assure adequate radiographic visualization of the hip prior to create the sterile field. The proximal femur is exposed through a lateral approach, but the nonunion site is not opened. The previous hardware is removed. The projected osteotomy site is marked as well as the position of the fixation device. The pathway for proximal fixation should be established prior to the osteotomy since it is very difficult to place a blade plate after the osteotomy is completed. For a blade plate, a guide wire and drill bits establish the pathway for the chisel. The pathway starts on the greater trochanter skirts

the piriformis fossa, across the nonunion and terminates in the inferior medial femoral head. The chisel is used to create a pathway for the blade plate. Frequent fluoroscopic views in both the anteroposterior and lateral views assure that correct placement.

The planned closing wedge osteotomy is marked with Kirschner wires. The proximal cut is parallel with the blade plate track and at least 1.5–2 cm distal to provide for a bone bridge. The osteotomy is then performed with a saw while cooling the blade with irrigation making sure not to cause thermal necrosis. It is also important to keep the osteotomy perpendicular to the shaft to avoid flexion or extension. The selected blade plate is seated, and then, the osteotomy is compressed with a compression device placed distally on the shaft. On the anteroposterior view, care is taken to avoid excessive medialization along the osteotomy as it contributes to not only to limb shortening but also to loss of abduction and valgus alignment of the knee. A longer blade and lateralization of the shaft limits this problem. Close attention is paid on the lateral view and rotation to assure that these are maintained in final fixation. Displacement in the lateral view will render subsequent arthroplasty if needed much more difficult. The harvested wedge is morselized and placed around the osteotomy site. Postoperatively, touch weight bearing and then protected weight bearing are allowed until healing is seen, usually in 3 months. Patients are cautioned that persistent limp and mild leg length difference are common while preserving the native hip.

Results of valgus intertrochantgeric osteotomy have been reported by a number of authors consistently reporting union from 70 to 100% of patients with avascular necrosis occurring around 20% [26–29, 34]. Marti et al. in a series of 50 patients with a mean age of 53 achieved a union rate of 86% [26]. Of 22 patients that had evidence of osteonecrosis without collapse only 3 progressed requiring arthroplasty. An additional 3 patients required arthroplasty for continued nonunion and one for periprosthetic fracture for an arthroplasty rate of 14% with a mean follow-up of 7 years. In this series, the Harris hip score was 91, while 78% were good or excellent on the Merle d'Aubigné score. Anglen had 100% union rate in 13 patients although 2 required arthroplasty by 2 years for osteonecrosis [27]. Ballmer et al. reported that in 17 patients, 71% healed following the index procedure while an additional 3 healed following additional bone grafting for an overall union rate of 88% [37]. Three, however, required subsequent arthroplasty for avascular necrosis. In Hartford et al.'s series of 8 patients treated with a

sliding hip screw union was achieved in all cases and Harris hip score improved from 24 to 73 [29]. Another more recent series reported the use of a single-angled 130 degree plate [30]. In 36 patients (mean age 37), union was achieved in 97%, while 5 developed avascular necrosis. 61% reported no pain, and the average leg length deficit of 2.5 cm was corrected to 0.5 cm. The authors of this series felt that the single-angle plate limited medialization while optimizing length. Other authors, using the double-angle plate, have been able to control this problem by laterally translating the femur. Unfortunately, more detailed outcomes measures are not available to judge functional status or compare methods of treatment.

### 9.1.4 Vascularized Graft

A vascularized fibular strut or vascular iliac crest has been proposed to enhance vascularity while correcting deformity and enhancing stability [31, 38, 39]. The fibular technique has been particularly advocated in nonunions with associated avascular necrosis provided the acetabulum is intact, but this is technically demanding and requires microvascular anastomosis.

The surgical technique described involves removal of hardware and revision fixation. Through a Watson-Jones approach, a channel is created in the lateral femur into the neck and head to accommodate the harvested fibula. The necrotic bone in the head if present is removed with special burrs. Correction of the femoral neck deformity is obtained from the mobility gained when the channel is created. The defect in the head is filled with cancellous bone from the greater trochanter. The fibula is placed into the head and neck and stabilized with a K-wire after the fibula is inserted. The critical vascular anastomosis is then performed. Further stability is obtained by placing cannulated screws parallel to the free fibula. Postoperatively the patient is nonweight bearing for 6 weeks and then gradually progressed thereafter. This technique is clearly technically demanding but is useful in the face of avascular necrosis.

The series reported by LeCroy et al. describes 22 patients (mean age 29) who underwent a vascularized fibula repair of a nonunion at a mean of 18 months following initial fixation [31]. Union was achieved in all patients at an average of 9 months but 2 required additional surgeries to achieve union. Osteonecrosis was present in all 22 cases at reconstruction surgery (predominantly Steinberg Stage II, 12 cases, but also Stage I, 4 cases; Stage III, 2 cases; Stage IV, 3 cases; even Stage V, 1 case). Despite osteonecrosis

progressing in 13 patients, the native hip was retained in 20 patients with an average Harris hip score of 78.9. Although only 5 reported being able to participate in vigorous physical activity that included running, 16 were able to perform moderate activity.

Recently, an alternate method of free vascularized fibula was reported by Jun and colleagues [32]. In the reported series of 26 cases, the free fibula was inserted via an anterior approach into a trough facilitating placement and simplifying vascular re-anastomosis. Twenty-four healed at an average of 5 months. One case of postperative osteonecrosis was observed. Unlike the series of Lecroy where all had osteonecrosis, in this series only 1 patient had radiographic findings of osteonecrosis preoperatively. Outcomes were reported in terms of the Harris hip score, which improved on average to 87.9.

The use of nonvascular fibular grafts has been reported by Elgafy and associates in 19 cases with minimal varus malalignment [33]. In that small series, nonvascular fibular sutografts achieved union in 9 of 13 cases (69%). The same authors reported union in only 2 of 6 cases in which a fibular allograft was used. Despite the relative technical ease of such a technique, there is no advantage in using a nonvascular fibular strut in the treatment of femoral neck nonunion.

### 9.1.5 Arthroplasty

Arthoplasty particularly with modern bearing surfaces should be considered in femoral neck nonunion not only in the face of acetabular destruction or avascular necrosis. In the patient over 60, arthroplasty is likely to provide a reliable method to return the patient to activity but does not match the excellent results achieved with primary arthroplasty for osteoarthritis. Although in the series of Marti et al. osteotomy was generally preferred in patients up to age 70 [26] and by Hitt to age 60 [19], modern techniques and implants have lowered the age in which arthroplasty is considered. Particularly in the younger patient, considerations should include the potential for infection, dislocation, and periprosthetic fracture. Although the incidence for each of these complications is low, for the patient with an infection, for instance, the outcomes are greatly diminished. It is well established that arthroplasty in the face of previous surgery raises the risk of infection [40].

The surgeon planning the arthroplasty should also consider the influence of disuse and the previous hardware on fixation of the stem (Fig. 9.5). In the

elderly patient in which arthroplasty is the preferred treatment for nonunion, osteopenia of both the proximal femur and the acetabulum may be present [41–45]. Press fit components should be used with caution particularly when compared to the sclerotic bone present in patients with osteoarthrtitis [46]. Relative osteopenia of the acetabulum may present problems with preparation and fixation. Sharp reamers in nonsclerotic bone may lead to excess medializaton and even iatrogenic protrusion. Cup preparation should be deliberate to avoid this problem. Screw augmentation of cup fixation should be also considered to assure stable fixation for ingrowth.

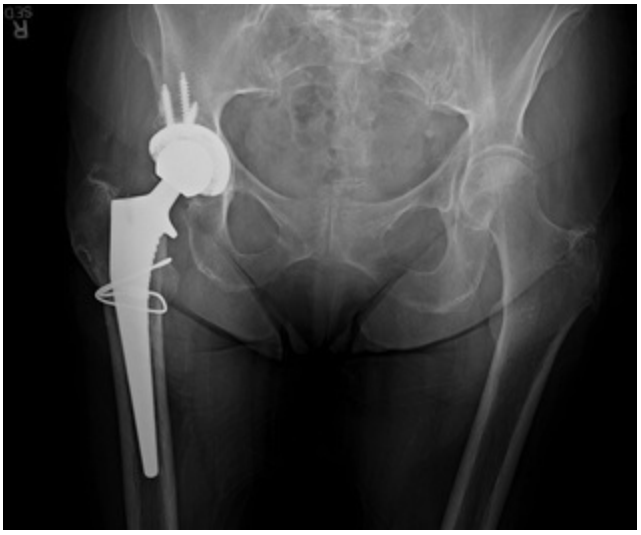


***Fig. 9.5*** Total hip arthroplasty for femoral neck nonunion performed for case in Fig. 9.2. Note screw augmentation of cup and cable of proximal femur

Secure fixation of the stem presents another challenge. In the younger patient with good bone stock, a press fit stem is preferred. However, the screw holes may predispose to fracture. Prophylactic cabling should be considered to avoid iatrogenic fracture. If cementing is selected, the surgeon should take measures to avoid extrusion of cement by plugging the holes with bone, cement, or hardware. Intraoperatively, the surgical site should be inspected and excess cement removed. The approach is dictated by previous incisions, hardware, and surgeon preference although the anterior approach may have lower dislocation rates [46]. Postoperatively, hip position restrictions are dictated by the approach, but full weight bearing is usually allowed immediately.

Outcomes following salvage arthroplasty have been described in a limited

degree but are consistently not as good as primary arthroplasty for osteoarthritis. McKinley and Robinson concluded that cemented total arthroplasty was the preferred treatment to salvage of failed femoral neck fixation with arthroplasty while noting results were inferior to primary arthroplasty for fracture [47]. In a matched group of 107 displaced femoral neck fractures, primary arthroplasty cases compared to 107 salvage patients with failed fixation, the primary arthoplasty group had fewer dislocations (8 vs. 21%) and fewer infections. The salvage group also had worse function and lower implant survivorship at 5 and 10 years. A series by Mabry et al. describes longer-term outcome of Charnley arthroplasty for femoral neck nonunion [48]. With a mean age of 68 years at time of surgery and a mean of 12 years follow-up, 10-year survivorship was 93 and 76% at 20 years. Dislocation was seen in 9% of cases. Revision surgery was performed in 12 of 99 cases: 10 for aseptic loosening, 1 for dislocation, and 1 Girdlestone arthroplasty for infection. In terms of function, 96% had no or mild pain, while 33% required no ambulatory aids and 36% used a single cane or crutch for support; 11% could not walk.

### 9.1.6 Arthroplasty for Failed Osteotomy

In the event of a failed intertrochanteric osteotomy, arthroplasty offers a means of restoring function. The altered anatomy may present challenges to the surgeon in placing the stem. Ferguson and colleagues noted a high rate of complications in his series of 305 cases although 79% were noted to have good to excellent results [49]. There were technical challenges in 23% of cases with perioperative complication in 12 and 3.2% infection rate. Revision was required in 10 years for 18% of cases with stem loosening predominating. In contrast, Boos et al. reported a case–control series of 74 arthroplasty cases following osteotomy with 74 primary arthroplasties finding few significant differences [50]. Ten-year survival was slightly less at 82% as opposed to 90%, but perioperative complications were equal at 11%. Revisions for sepsis were also slightly higher at 8% compared to 3%.

### 9.1.7 Neglected Femoral Neck Fracture

Although uncommon in the developed world, missed or delayed diagnosis of femoral neck fracture may present for treatment weeks or months after injury. Such an instance may be considered a “neglected” femoral neck fracture,

which presents with pain and limited ambulatory capacity [51–54]. These may present to the surgeon with or without signs of avascular necrosis. Contrary to popular belief, these patients are not condemned to developing avascular necrosis or poor results although these cases are challenging. Successful repair with salvage of the native femoral head can be achieved with careful planning and execution of surgery. In addition to plain radiographs, CT scan provides valuable information regarding fracture orientation and bone loss. Evaluation of options and careful operative planning should proceed with evaluation of the degree of bony erosion at the fracture site. In the young patient, hip preservation with appropriate fixation should be performed (Fig. 9.6).


***Fig. 9.6*** Delayed diagnosis of femoral neck fracture: **a** at presentation, **b** CT axial note apex anterior deformity, **c** CT coronal note varus, **d** healed after fixation. Combined Smith Peterson and lateral

approach was utilized. Iliac crest graft was utilized to fill the defect. A universal distractor placed supra-acetabular provided distraction intraoperatively

Although some shortening of the femoral neck is common, anteversion should be restored along with critical coronal plane alignment. Fixation constructs such as those used in fresh fractures may be used if fracture orientation is not vertical and reduction can be obtained. Cancellous bone graft should be considered. However, a valgus intertrochanteric osteotomy should strongly be considered at the outset in most cases even to include intermediate Pauwels angles.

Lifeso and Younge in their series of 31 neglected femoral neck fractures (ages 16–90) treated 19 with arthroplasty, 3 with internal fixation, and 6 with valgus intertrochanteric osteotomy at an average of 34 months post-injury [55]. Three patients with nondisplaced fractures underwent fixation alone; however, only 1 had a good result. The 6 patients with displaced neglected fractures undergoing valgus osteotomy were all under 40 years of age. Of these, 3 were considered to have a good result achieving union, while 2 had fixation failure and one avascular necrosis. More detailed outcome data is not provided but demonstrates that successful salvage is possible in this young patient population with neglected fractures. A more recent series of 32 patients in India achieved union in 91% of cases [56]. The surgeons performed a valgus intertrochanteric osteotomy at an average of 6 months post-injury. The average patient age was 42. Follow-up at an average of 5 years revealed a Harris hip score of 82. Three developed nonunions, and six had stage 3 or 4 avascular necrosis. The authors noted that fracture resorption and excessive valgus correlated with poor outcome, while the presence of avascular necrosis did not. Although there is limited data on treatment of this uncommon presentation of the neglected femoral neck, successful salvage is achievable in the young patient where arthroplasty is not desired.

## 9.2 Intertrochanteric Fracture Nonunion

Intertrochanteric fractures unlike femoral neck fractures almost always heal although malunion is not uncommon. The metaphyseal area with its good blood supply promotes healing. Nonetheless, nonunion must be in the differential if the patient has persistent pain after fixation or if the hardware fails. Visualization of the nonunion may be difficult due to hardware obscuring the fracture line, which also may be out of the plane of standard

radiographs. Computerized tomography is invaluable in differentiating nonunion from malunion. The lesser trochanter may remain ununited, but this is usually not problematic. Other areas without bony bridging may be seen but likely do not constitute a nonunion or delayed union unless 50% or more is not bridged. No precise definition exists here making clinician judgment critical [2, 4].

Once the diagnosis of intertrochanteric nonunion is established, diagnostic workup based on history and physical is performed with appropriate laboratory and radiographic evaluation as outlined earlier in the chapter. Good quality anteroposterior pelvis and full femur films are important to devise the surgical plan. Aside from patient age and functional status, decision-making depends on the status of previous hardware, the quality of the femoral head and neck, and critically whether the acetabulum is intact. In the younger, more active patient achieving union is preferred, while in the older, lower demand patient arthroplasty should be considered.

In the presence of satisfactory alignment with intact hardware and hip articulation, the option for simple bone grafting with or without hardware revision can be successful. Otherwise, the hardware should be revised. An intramedullary device offers a biomechanical advantage while also providing bone graft from reaming. The intramedullary device lies in the axis of force of the proximal femur, while a side plate is more lateral, subjecting it to cantilevering when the side plate is loaded with weight bearing. With a variety of angles available for the lag screw, an intramedullary implant can be placed to replace a variety of previous devices. A blade plate offers alternate fixation of the proximal segment entering very proximally laterally while taking advantage of the often-pristine area of the inferior head and neck. It can also allow for compression axially while correcting the varus deformity. The use of proximal femur-locking plates may be successful in select cases but has been associated with a number of reported failures [57, 58].

Varus deformity and lucency around the implant are signs of nonunion. Hardware failure may be seen in the form of pull out or breakage in an intertrochanteric nonunion. Intramedullary devices may fracture at the junction of the lag screw and the rod. Broken screws are removed with conventional techniques. A broken rod may be more difficult. The use of an extraction hook can capture the distal end allowing for its removal.

Given the biomechanical advantages of intramedullary devices and new designs, revision fixation is most commonly used by the author although no

series is available to support this trend (Fig. 9.7). However, blade plate is an appropriate option if a deformity needs to be corrected, if alternate proximal fixation is required, or failure of exchange reamed nailing.

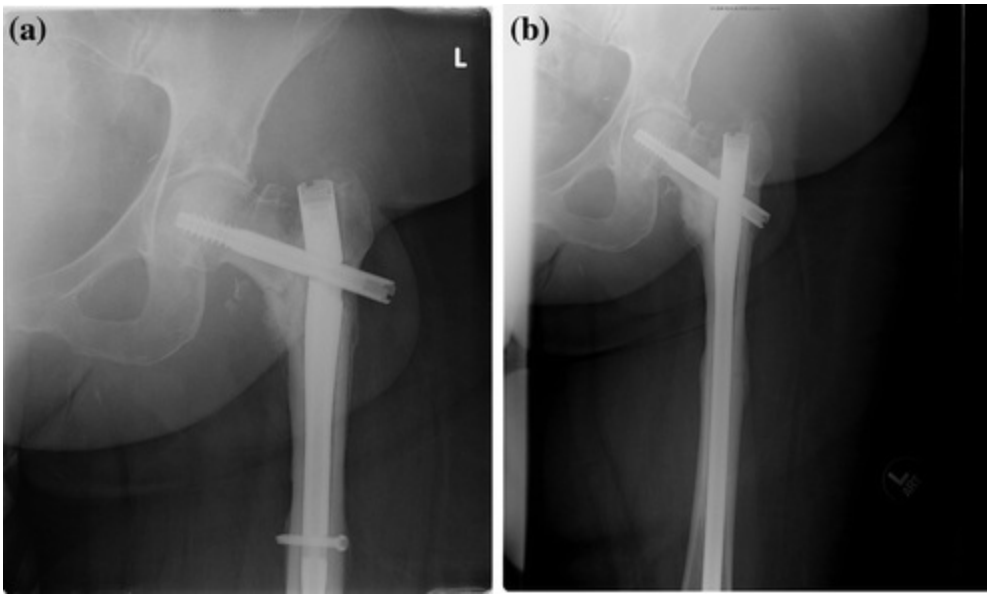



***Fig. 9.7*** Nonunion of intertrochanteric fracture: **a** broken nail, **b** healed with revision with larger nail

In the elderly patient or when the hip articulation is damaged, arthroplasty offers a reliable method for restoring mobility (Fig. 9.8). A hemiarthroplasty can be used if the acetabulum is intact. A unipolar head allows for improved stability compared to a total arthroplasty at the risk of acetabular wear and arthritis. A calcar-replacing stem can be used, or conventional or revision arthroplasty stems can be used, provided that distal fixation is achieved. When planning for the arthroplasty, restoration of normal biomechanics with restoration of length and offset will provide optimal results. However, changes from the surgery and subsequent deformation may lead to contracted soft tissues and weak abductors. The most critical factor is to restore stability of the hip by retaining critical soft tissue and establishing correct length. Modular components assist in achieving this goal.


***Fig. 9.8*** Total hip arthroplasty for intertrochanteric nonunion with fixation of trochanter

There are few reported series of treatment of intertrochanteric nonunions and all predate the widespread use of intramedullary implants. Haidukewych and Berry reported on 20 cases that underwent revision, achieving union in all but one with resolution of pain [59]. The patients were older with an average age of 58 (range 21–86) presenting at a mean of 12 months from initial injury. The previous implant was a plate device in all cases (14 sliding screw and side plate) while only one was a cephalomedullary nail. Two cases of infected nonunion underwent staged treatment with removal, interval antibiotics, and subsequent revision. Revision implants were most commonly low angle blade plates (11 cases), and autologous bone graft was used in 17 cases. Other devices included dynamic hip and dynamic condylar screws. Only 1 older generation intramedullary device was used. In 4 cases, an osteotomy was performed to correct the deformity. The one failure was converted to a cemented arthroplasty. No patient under 60 required ambulatory supports, 8 required a single cane, and 4 used a walker. The authors commented that effective salvage is possible in the physiologically younger patient with good bone with the ability to achieve stable fixation and an intact hip articulation.

Other reported series consisted of 11 or fewer patients in a similar age group [60–62]. The Sarathy et al. series [61] consisted of previously unoperated cases treated with a medial displacement osteotomy, a technique predating the widespread use of internal fixation. Similar fixation strategy was used in all 3 series; however, they capitalized on fixation in the inferomedial femoral neck and utilized autologous bone graft. The union success rate was uniformly over 90%. Another small series reported on 4 cases of varus nonunion and malunion treated with an intertrochanteric osteotomy with a 120° blade plate similar to a femoral neck nonunion [63]. All cases healed while regaining an average of 2 cm of leg length and the Harris hip score improved from 73 to 92.

The experience with arthroplasty is similarly limited [64–67] as a treatment for intertrochanteric nonunion. The principles and potential complications mirror the experience with arthroplasty treatment for femoral neck nonunion. A series of 60 patients with an average age of 78 years from the Mayo clinic were treated with arthroplasty [64], 28 with hemiarthroplasty, and the remainder with predominantly cemented total hip arthroplasty. Calcar-replacing stems were used in 51 cases. Of 44 survivors not lost to follow-up, 39 walked with minimal pain. Two patients sustained femur fractures during surgery managed by cerclage wiring. One patient had 2 episodes of dislocation each treated with closed reduction. Survivorship was 100% at 7 years and 87.5% at 10 years indicating excellent durability. The two revisions were at 8 and 10 years for aseptic loosening and severe osteolysis. The authors noted that careful surgical technique can lead to very functional results with few complications. Hemiarthroplasty in cases of lower demand and well-preserved articular cartilage did not appear to result in symptomatic wear in this series.

Hammad et al. reported on a younger cohort of 32 patients with an average age of 64 [65]. Again, most underwent cemented arthroplasty but 5 were uncemented and 7 hybrid. Perioperative complications were low with one fracture, one dislocation, and 3 deep venous thromboses. On follow-up at a mean of 5 years, Harris hip scores were markedly improved from a preoperative score of 24–84 postoperatively. Community ambulation was noted in 88% although half required a single cane or crutch.

## 9.3 Subtrochanteric Nonunion

Subtrochanteric fractures have historically presented greater difficulty in achieving satisfactory union [68]. The high biomechanical loads and deforming forces present challenges to the surgeon and the implant. However, using modern implants with the emphasis on preserving the local blood supply to bone, high union rates are achieved. Kinast and colleagues achieved union rates of 100% using a blade plate and indirect reduction techniques [69]. Other modern series particularly those using modern standard and cephalomedullary nails noted union rates of 92–100% [70–73].

The radiographic evaluation of this region is more straightforward that the femoral neck and the intertrochanteric region in assessing union. Nonetheless, computerized tomography may be useful to evaluate the location and extent of the nonunion. Clinical and radiographic assessment of length, angulation, and rotation is also important in creating the surgical plan and informing the patient of expected outcomes. Flexion and varus deformity are often encountered and should be corrected to the maximal extent possible.

In the surgical plan, the surgeon must take into account the status of the existing bone and the hardware present. In many cases of nonunion with a preexisting nail, exchange reamed nailing allows for use of previous approaches while the reaming provides bone graft when no significant deformity is present. Both reconstruction nails and third-generation cephalomedullary nails exist in larger diameters of 13 and even 15 mm upon request from the manufacturers enhancing not only the strength of the nail but also the fill within the canal. The use of dynamic interlocking can further enhance compression at the fracture. Alternatively, bone graft in situ can be considered when the fracture implant, nail, or plate is intact. However, the surgeon must be satisfied that the construct provides appropriate stability and no significant deformity is present.

A blade plate or a 95° condylar plate placed centrally into the inferior femoral head is a useful alternative device particularly when alternate control of the proximal segment is felt to be necessary (Fig. 9.9). With both of these devices, additional screws can be placed through the plate into the proximal segment enhancing its control. Locking proximal femur plates offer another alternative here but have been associated with failure [57, 58].

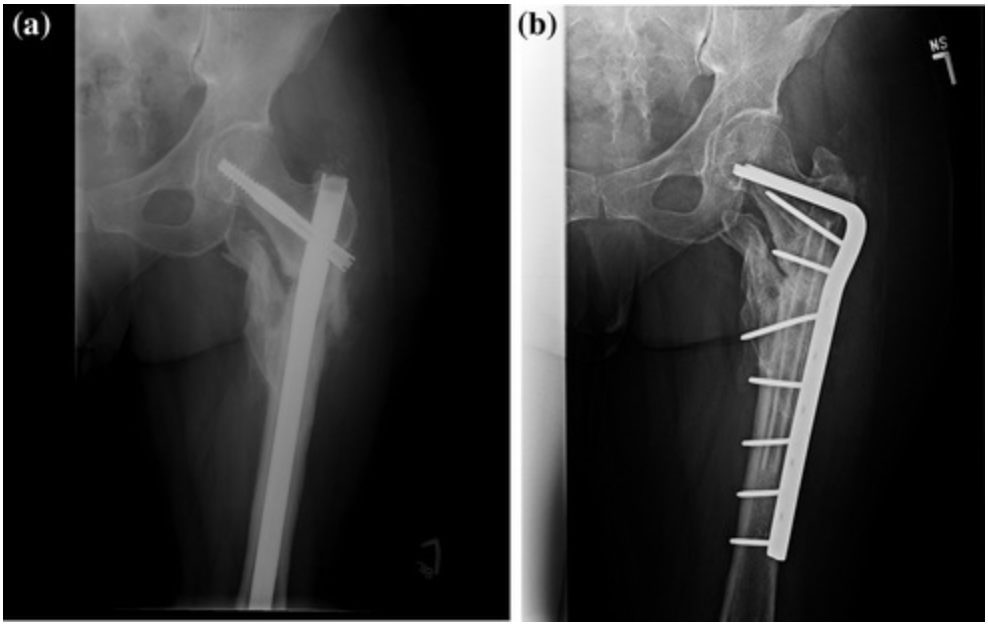



***Fig. 9.9*** Subtrochanteric nonunion: **a** nonunion after first revision, **b** revision with intramedullary allograft fibula, autogenous bone graft and blade plate

Several retrospective clinical series report results of nonunion treatment. Haidukewych and Berry reported a 95% union rate in a series of 24 cases in patients with an age of 55 years (range 16–88) presenting 21 months after injury [74]. The majority had initial treatment with plate devices. Nonunion surgery consisted of hardware removal and revision fixation. Fixation constructs included: 15 nails (7 reconstruction, 1 cephalomedullary, 7 standard antegrade); and 7 plates (5 blade plates, 1 screw and side plate, 1 dynamic condylar screw, and one dual plating). Autograft was used in 8 patients although 1 patient with four previous failed nailing attempts underwent vascularized fibular grafting in addition to nail stabilization. Eleven of 21 walked without ambulatory aids. When shortening is noted, restoration of native length can be achieved in one stage with use of bone graft and an intramedullary nail. A series reported 100% union and restoration of length in 21 cases of aseptic nonunion with shortening of 2–5 cm at presentation [75].

Another series of 29 patients treated exclusively with a cephalomedullary nail achieved successful union in 88% of cases following the initial surgery and 96% with a single additional procedure [76]. This group was slightly older (average age 63) but also included young patients with high-energy injuries, with 11 patients between the age of 26 and 50. Similar to the Mayo

series [74], 66% had been previously treated with plate devices. Although they reported that 81% returned to their preinjury status, outcome data does not otherwise allow comparison of techniques to determine whether intramedullary devices or plating is superior. Decision-making should be based on the ability to correct deformity if present and maintain fixation in the proximal segment to achieve union.

In cases where the biologic environment for healing is severely compromised due to the presence of segmental bone loss or from devascularization from multiple surgical procedures, infection, or irradiation, a vascularized fibula strut may allow for bypassing of the affected segment into an environment that allows for healing. In addition to the technical challenge of the vascular anastomosis, a stable construct must be created for both the fibular strut and the femur as whole. The fibula may be inset into the medullary canal or into a trough anteriorly or even medially. A plate may then stabilize the femur. Union must be achieved of the fibula to the proximal and distal femur segments. The segment may then gradually hypertrophy in response to physiologic stress of loading the femur. Weight bearing and activity must be limited and gradually advance in the course of the healing process.

Duffy reported on 4 patients who sustained proximal femur fractures following an average of 5500 gray irradiation for bony and soft tissue malignancy in a mixed series of 17 patients with radiation associated fractures in various locations [77]. The fracture occurred at an average of 111 months after irradiation. The age of these patients at the time of free fibular transfer was between 13 and 82 years of age and had an established nonunion for 15 months on average. In addition to the free fibula, the femur was stabilized with an intramedullary rod. Three of four had excellent results, while the fair result was associated with infection requiring debridement and had continued pain. It should be noted that one of the excellent results sustained a second fracture distal to the vascularized fibula requiring a second vascularized fibula. The authors advise that fractures in irradiated bone should undergo vascularized fibular grafting when nonvascular fibula graft with an appropriate fixation fails to achieve union in 6 months.

In a very unusual circumstance, proximal femoral replacement can be considered as a reconstructive option. Stability of the hip joint and the function of the hip abductors are a challenge to restore.

Another scenario for delayed or nonunion of the proximal femur occurs in

association with the so-called atypical fractures femur in association with prolonged bisphosphonate use (Fig. 9.10) [10]. These are often transverse fractures in the proximal femur with an associated thickening of the cortex from the chronic stress. Trivial injury can complete the fracture, or the patient may simply present with chronic pain in the femur with radiographic changes of a stress fracture. These atypical fractures occur usually after 5 years of bisphosphonate use.

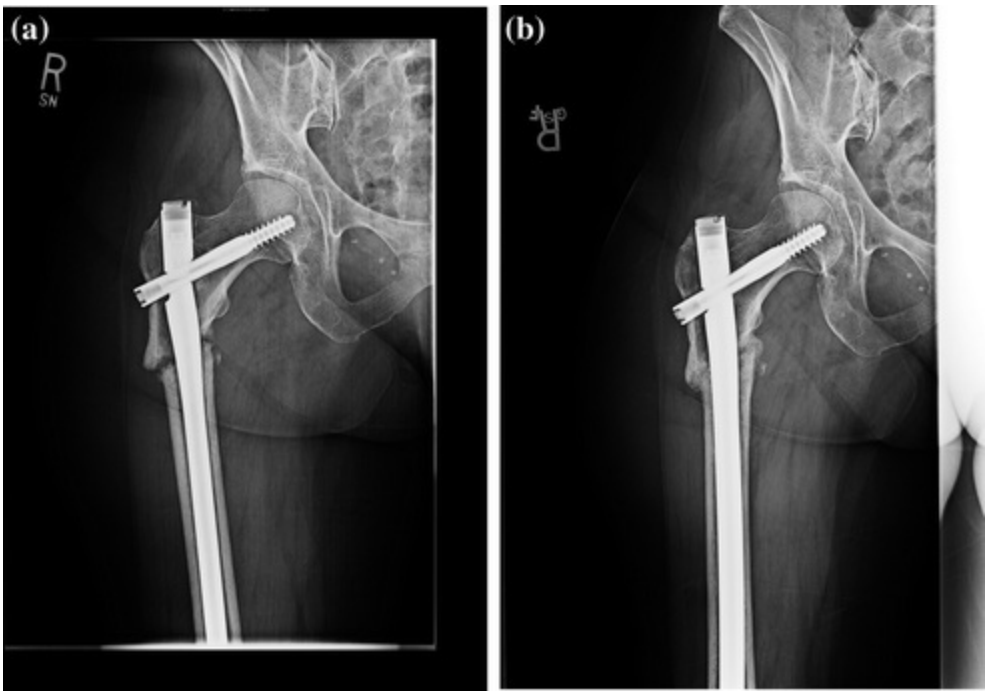



***Fig. 9.10*** Atypical fracture of the proximal femur: **a** nonunion with broken nail (initially fixed in slight varus), **b** healed after revision fixation with larger nail

At the time of initial treatment, bisphosphonate treatment should be terminated. The use of dynamic interlock allows for compression with weight bearing. In the event of nonunion, exchange reamed nailing will typically result in success. However, if initial treatment resulted in varus alignment, the use of a blade plate or dynamic condylar plate can correct the deformity and compress across the fracture.

## 9.4 Summary

Nonunion of the proximal femur is most common in the femoral neck although it may also be seen in the intertrochanteric and subtrochanteric

region. Often the nonunion is evidenced by persistent fracture gap, increasing deformity, or broken hardware. It is imperative that the surgeon perform not only a careful radiographic analysis of the nonunion but also investigate all potential contributing factors to the nonunion. Repair of the nonunion then depends on correction of the deformity, and an improved biomechanical construct and where appropriate augmentation of the biologic potential for healing with the use of bone graft. Successful union can be achieved with a careful evaluation, thoughtful planning, and skillful execution of the surgical plan in most cases. In some cases depending on the patient's age functional status and the status of the hip articulation, arthroplasty offers the best option to functional restoration. Careful discussion with the patient is required for them to understand the expected postoperative course along with the potential for complications.

---

# References


1. Cornwall R, Gilbert MS, Koval KJ, Strauss E, Siu AL. Functional outcomes and mortality vary among different types of hip fractures: a function of patient characteristics. Clin Orthop Relat Res. 2004;425:64–71.
[Crossref]

2. Morshed S. Current options for determining fracture union. Adv Med. 2014, Article ID 708574. doi:9.1155/2014/708574.

3. Bhandari M, Chiavaras M, Ayeni O, Chakraverrty R, Parasu N, Choudur H, et al. Assessment group for radiographic evaluation and evidence (AGREE) Study Group (AGREE Investigators Writing Committee). Assessment of radiographic fracture healing in patients with operatively treated femoral neck fractures. J Orthop Trauma. 2013;27(9):e213–9.
[Crossref][PubMed]

4. Bhandari M, Chiavaras MM, Parasu N, Choudur H, Ayeni O, Chakravertty R, et al. Radiographic union score for hip substantially improves agreement between surgeons and radiologists. BMC Musculoskelet Disord. 2013;14:70.
[Crossref][PubMed][PubMedCentral]

5. Brinker MR, O'Connor D, Monla Y, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trauma. 2007;21(8):557–70.
[Crossref][PubMed]

6. Koval KJ, Maurer SG, Su ET, Aharonoff GB, Zuckerman JD. The effects of nutritional status on outcome after hip fracture. J Orthop Trauma. 1999;13(3):164–9.
[Crossref][PubMed]

7. Dodwell ER, Latorre JG, Parisini E, Zwettler E, Chandra D, Mulpuri K, Snyder B. NSAID

exposure and risk of nonunion: a meta-analysis of case–control and cohort studies. Calcif Tissue Int. 2010;87(3):193–202.
[Crossref][PubMed]

8. Gerstenfeld LC, Thiede M, Seibert K, Mielke C, Phippard D, Svagr B, et al. Differential inhibition of fracture healing by non-selective and cyclooxygenase-2 selective non-steroidal anti-inflammatory drugs. J Orthop Res. 2003;21(4):670–5.
[Crossref][PubMed]

9. Matthews V, Cabanela ME. Femoral neck nonunion treatment. Clin Orthop. 2004;419:57–64.
[Crossref]

10. Blood T, Feller RJ, Cohen E, Born CT, Hayda R. Atypical fractures of the femur: evaluation and treatment. JBJS Reviews. 2015;3(3):e1–8.
[Crossref]

11. Nardi A, Ventura L, Cozzi L, Tonini G, Zennaro R, Celi M, Ramazzina E. The bone anabolic therapy. Aging Clin Exp Res. 2013;25(Suppl 1):S121–4.
[Crossref][PubMed]

12. Rubin LE, Murgo KT, Ritterman SA, McClure PK. Hip resection arthroplasty. JBJS Rev. 2014;2(5):e3.
[Crossref]

13. Protzman RR, Burkhalter WE. Femoral-neck fractures in young adults. J Bone Joint Surg Am. 1976;58(5):689–95.
[Crossref][PubMed]

14. Swiontkowski MF, Winquist RA, Hansen ST Jr. Fractures of the femoral neck in patients between the ages of twelve and forty-nine years. J Bone Joint Surg Am. 1984;66(6):837–46.
[Crossref][PubMed]

15. Angelini M, McKee MD, Waddell JP, Haidukewych G, Schemitsch EH. Salvage of failed hip fracture fixation. J Orthop Trauma. 2009;23(6):471–8.
[Crossref][PubMed]

16. Haidukewych GJ, Rothwell WS, Jacofsky DJ, Torchia ME, Berry DJ. Operative treatment of femoral neck fractures in patients between the ages of fifteen and fifty years. J Bone Joint Surg Am. 2004;86-A(8):1711–1716.

17. Parker MJ, Raghavan R, Gurusamy K. Incidence of fracture-healing complications after femoral neck fractures. Clin Orthop Relat Res. 2007;458:175–9.
[PubMed]

18. Conn KS, Parker MJ. Undisplaced intracapsular hip fractures: results of internal fixation in 375 patients. Clin Orthop Relat Res. 2004;421:249–54.
[Crossref]

19. Hitt K. Femoral neck nonunion: osteotomy or arthroplasty. Tech Orthop. 2001;17(4):434–42.
[Crossref]

20.

Trueta J, Harrison MH. The normal vascular anatomy of the femoral head in adult man. J Bone Joint Surg Br. 1953;35-B(3):442–461.

21. Hughes PE, Hsu JC, Matava MJ. Hip anatomy and biomechanics in the athlete. Sports Med Arthrosc Rev. 2002;10(2):103–14.
[Crossref]

22. Alho A, Benterud JG, Solovieva S. Internally fixed femoral neck fractures: early preduction of failure in 203 elderly patients with displaced fractures. Acta Orthop Scand. 1999;70(2):141–4.
[Crossref][PubMed]

23. Bartonícek J. Pauwels' classification of femoral neck fractures: correct interpretation of the original. J Orthop Trauma. 2001;15(5):358–360. Review.

24. Goldstein C, Petrisor BA, Ferguson T, Bhandari M. Implants for fixation of femoral neck fractures. Tech Orthop. 2008;23(4):301–8.
[Crossref]

25. Gurusamy, Parker MJ, Rowlands TK. The complications of displaced intracapsular fractures of the hip the effect of screw positioning and angulation on fracture healing. J Bone Joint Surg Br. 2005;87(5):632–634.

26. Marti RK, Schüller HM, Raaymakers EL. Intertrochanteric osteotomy for non-union of the femoral neck. J Bone Joint Surg Br. 1989;71(5):782–7.
[Crossref][PubMed]

27. Anglen JO. Intertrochanteric osteotomy for failed internal fixation of femoral neck fracture. Clin Orthop. 1997;341:175–82.
[Crossref]

28. Schoenfeld AJ, Vrabec GA. Valgus osteotomy of the proximal femur with sliding hip screw for the treatment of femoral neck nonunions: the technique, a case series, and literature review. J Orthop Trauma. 2006;20(7):485–91.
[Crossref][PubMed]

29. Hartford JM, Patel A, Powell J. Intertrochanteric osteotomy using a dynamichip screw for femoral neck nonunion. J Orthop Trauma. 2005;19(5):329–33.
[PubMed]

30. Said GZ, Farouk O, Said HG. Valgus intertrochanteric osteotomy with single-angled 130° plate fixation for fractures and non-unions of the femoral neck. Int Orthop. 2010;34(8):1291–5.
[Crossref][PubMed]

31. LeCroy CM, Rizzo M, Gunneson EE, Urbaniak JR. Free vascularized fibular bone grafting in the management of femoral neck nonunion in patients younger than fifty years. J Orthop Trauma. 2002;16(7):464–72.
[Crossref][PubMed]

32. Jun X, Chang-Qing Z, Kai-Gang Z, Hong-Shuai L, Jia-Gen S. Modified free vascularized fibular grafting for the treatment of femoral neck nonunion. J Orthop Trauma. 2010;24(4):230–5.
[Crossref][PubMed]

33. Elgafy H, Ebraheim NA, Bach HG. Revision internal fixation and nonvascular fibular graft for femoral neck nonunion. J Trauma. 2011;70(1):169–73.
[Crossref][PubMed]

34. Wu CC, Shih CH, Chen WJ, Tai CL. Treatment of femoral neck nonunions with a sliding compression screw: comparison with and without subtrochanteric valgus osteotomy. J Trauma. 1999;46(2):312–7.
[Crossref][PubMed]

35. Meyers MH, Harvey JP Jr, Moore TM. The muscle pedicle bone graft in the treatment of displaced fractures of the femoral neck: indications, operative technique and results. Orthop Clin North Am. 1974;5(4):779–92.
[PubMed]

36. Baksi DP. Internal fixation of ununited femoral neck fractures combined with muscle-pedicle bone grafting. J Bone Joint Surg Br. 1986;68(2):239–45.
[PubMed]

37. Ballmer FT, Ballmer PM, Baumgaertel F, Ganz R, Mast JW. Pauwels osteotomy for nonunions of the femoral neck. Orthop Clin North Am. 1990;21(4):759–67.
[PubMed]

38. Hou SM, Hang YS, Liu TK. Ununited femoral neck fractures by open reduction and vascularized iliac bone graft. Clin Orthop Relat Res. 1993;294:176–80.

39. Leung PC, Shen WY. Fracture of the femoral neck in younger adults: a new method of treatment of delayed and nonunions. Clin Orthop Relat Res. 1993;295:156–60.

40. Cordero-Ampuero J, de Dios M. What are the risk factors for infection in hemiarthroplasties and total hip arthroplasties? Clin Orthop Relat Res. 2010;468(12):3268–77.
[Crossref][PubMed][PubMedCentral]

41. Franzén H, Nilsson LT, Strömqvist B, Johnsson R, Herrlin K. Secondary total hip replacement after fractures of the femoral neck. J Bone Joint Surg Br. 1990;72(5):784–7.
[Crossref][PubMed]

42. Hägglund G, Nordström B, Lidgren L. Total hip replacement after nailing failure in femoral neck fractures. Arch OrthopTrauma Surg. 1984;103(2):125–7.
[Crossref]

43. Nilsson LT, Jalovaara P, Franzén H, Niinimäki T, Strömqvist B. Function after primary hemiarthroplasty and secondary total hip arthroplasty in femoral neck fracture. J Arthroplasty. 1994;9(4):369–73.
[Crossref][PubMed]

44. Tabsh I, Waddell JP, Morton J. Total hip arthroplasty for complications of proximal femoral fractures. J Orthop Trauma. 1997;11(3):166–9.
[Crossref][PubMed]

45. Turner A, Wroblewski BM. Charnley low-friction arthroplasty for the treatment of hips with late complications of femoral neck fractures. Clin Orthop. 1984;185:126–30.

46. Haidukewych G, Berry DJ. Salvage of failed treatment of hip fractures. J Am Acad Orthop Surg. 2005;13(2):101–9.
[Crossref][PubMed]

47. McKinley JC, Robinson CM. Treatment of displaced intracapsular hip fractures with total hip arthroplasty: comparison of primary arthroplasty with early salvage arthroplasty after failed internal fixation. J Bone Joint Surg Am. 2002; 84-A(11):2010–5.

48. Mabry TM, Prpa B, Haidukewych GJ, Harmsen WS, Berry DJ. Long-term results of total hip arthroplasty for femoral neck fracture nonunion. J Bone Joint Surg Am. 2004;86-A(10):2263–2267.

49. Ferguson GS, Cabanela ME, Ilstrup DM. Total hip arthoplasty after failed intertrochanteric osteotomy. J Bone Joint Surg Br. 199;76(2): 252–257.

50. Boos N, Krushell R, Ganz R, Müller ME. Total hip arthroplasty after previous proximal femoral osteotomy. J Bone Joint Surg Br. 1997;79(2):247–53.
[Crossref][PubMed]

51. Sandhu HS, Sandhu PS, Kapoor A. Neglected fractured neck of the femura predictive classification and treatment by osteosynthesis. Clin Orthop Relat Res. 2005;431:14–20.
[Crossref]

52. Nagi ON, Dhillon MS, Goni VG. Open reduction, internal fixation and fibular autografting for neglected fracture of the femoral neck. J Bone Joint Surg Br. 1998;80(5):798–804.
[Crossref][PubMed]

53. Huang HC. Treatment of neglected femoral neck fractures in young adults. Clin Orthop Relat Res. 1986;206(5):119–126.

54. Roshan A, Ram S. Early return to function in young adults with neglected femoral neck fractures. Clin Orthop Relat Res. 2006;447(6):152–7.
[Crossref][PubMed]

55. Lifeso R, Younge D. The neglected hip fracture. J Orthop Trauma. 1990;4(3):287–92.
[Crossref][PubMed]

56. Verghese VD, Boopalan PR, Titus V, Oomen VT, Jepegnanam TS. Indices affecting outcome following neglected femoral neck fracture after valus intertrochanteric osteotomy. J Orthop Trauma. 2014;28(7):410–6.
[Crossref]

57. Glassner PJ, Tejwani NC. Failure of proximal femoral locking compression plate: a case series. J Orthop Trauma. 2011;25(2):7683.
[Crossref]

58. Johnson B, Stevenson B, Chamma R, Patel A, Rhee S, Lever C, et al. Short-term follow-up of pertrochanteric fractures treated using the proximal femoral locking plate. J Orthop Trauma. 2014;28(5):283–7.
[Crossref][PubMed]

59. Haidukewych GJ, Berry DJ. Salvage of failed internal fixation of intertrochanteric hip fracture.

Clin Orthop Relat Res. 2003;412:184–8.
[Crossref]

60. Mariani EM, Rand JA. Nonunion of intertrochanteric fractures of the femur following open reduction and internal fixation: Results of second attempts to gain union. Clin Orthop Relat Res. 1987;218:81–9.

61. Sarathy MP, Madhavan P, Ravichandran KM. Nonunion of intertrochanteric fractures of the femur. J Bone Joint Surg Br. 1995;77(1):90–2.
[PubMed]

62. Wu CC, Shih CH, Chen WJ, Tai CL. Treatment of cutout of a lag screw of a dynamic hip screw in an intertrochanteric fracture. Arch Orthop Trauma Surg. 1998;117(4–5):193–6.
[Crossref][PubMed]

63. Bartonícek J, Skála-Rosenbaum J, Dousa P. Valgus intertrochanteric osteotomy for malunion and nonunion of trochanteric fractures. J Orthop Trauma. 2003;17(6):606–12.
[Crossref][PubMed]

64. Haidukewych GJ, Berry DJ. Hip arthroplasty for salvage of failed treatment of intertrochanteric hip fractures. J Bone Joint Surg Am. 2003;85-A(5):899–904.

65. Hammad A, Abdel-aal A, Said HG, Bakr H. Total hip arthroplasty following failure of dynamic hip screw fixation of fractures of the proximal femur. Acta Orthop Belg. 2008;74(6):788–92.
[PubMed]

66. Laffosse JM, Molinier F, Tricoire JL, Bonnevialle N, Chiron P, Puget J. Salvage hip arthroplasty for trochanteric osteosynthesis failure in the elderly. Acta Orthop Belg. 2007;73(6):729–36.
[PubMed]

67. Tabsh I, Waddell JP, Morton J. Total hip arthroplasty forcomplications of proximal femoral fractures. J Orthop Trauma. 1997;11(3):166–9.
[Crossref][PubMed]

68. Lundy DW. Subtrochanteric femoral fractures. J Am Acad Orthop Surg. 2007;15(11):663–71.
[Crossref][PubMed]

69. Kinast C, Bolhofner BR, Mast JW, Ganz R. Subtrochanteric fractures of the femur: Results of treatment with the 95 degrees condylar blade-plate. Clin Orthop Relat Res. 1989;238:122–30.

70. Wiss DA, Brien WW. Subtrochanteric fractures of the femur: results of treatment by interlocking nailing. Clin Orthop Relat Res. 1992;283:231–6.

71. Kang S, McAndrew MP, Johnson KD. The reconstruction locked nail for complex fractures of the proximal femur. J Orthop Trauma. 1995;9(6):453–63.
[Crossref][PubMed]

72. Pai CH. Dynamic condylar screw for subtrochanteric femur fractures with greater trochanteric extension. J Orthop Trauma. 1996;10(5):317–22.
[Crossref][PubMed]

73. French BG, Tornetta P 3rd. Use of an interlocked cephalomedullary nail for subtrochanteric

fracture stabilization. Clin Orthop Relat Res. 1998;348:95–100.
[Crossref]

74. Haidukewych GJ, Berry DJ. Nonunion of fractures of the subtrochanteric region of the femur. Clin Orthop Relat Res. 2004;419:185–8.
[Crossref]

75. Wu C. Locked nailing for shortened subtrochanteric nonunions: a one-stage treatment. Clin Orthop Relat Res. 2009;467(1):254–9.
[Crossref][PubMed]

76. Barquet A, Mayora G, Fregeiro J, López L, Rienzi D, Francescoli L. The treatment of subtrochanteric nonunions with the long gamma nail twenty-six patients with a minimum 2-year follow-up. J Orthop Trauma. 2004;18(6):346–53.
[Crossref][PubMed]

77. Duffy GP, Wood MB, Rock MG, Sim FH. Vascularized free fibular transfer combined with autografting for the management of fracture nonunions associated with radiation therapy. J Bone Joint Surg Am. 2000;82(4):544–54.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_10

# 10. Femoral Shaft Nonunions

Gil R. Ortega[1] and Brian P. Cunningham[2]

(1) Department of Orthopedic Surgery, Mayo Clinic Arizona Orthopedic Residency Program, Mayo Clinic Arizona, HonorHealth Scottsdale Osborn Trauma Center, Sonoran Orthopedic Trauma Surgeons, 3126 N. Civic Center Plaza, Scottsdale, AZ 85251, USA
(2) Department of Orthopedic Surgery, University of Minnesota, Regions Hospital, St. Paul, MN 55101, USA

**Gil R. Ortega**
**Email:** gortega@sotsmd.com

**Keywords** Femoral nonunion – Nonunion – Femur

## 10.1 Introduction and Background

In 1939, Dr. Küntscher introduced intramedullary (IM) nails and subsequently revolutionized the treatment of long bone fractures [1]. The transition to intramedullary nails for the treatment of femoral shaft fractures has resulted union rates ranging between 90 and 100% [2–4]. These fractures are typically a high-energy injury resulting in significant mortality risks, and they are often encountered with other injuries. Improved trauma care has provided decreased mortality for severely injured patients, and it has been suggested that this may result in an increasing number of complex femoral shaft fractures and subsequently increase the number of femoral nonunion [5–8]. Femoral shaft nonunions can result from a number of etiologies including inadequate fixation stability, insufficient blood supply, bone loss, or infection. Despite the success of treating femoral shaft fractures as well as

improved surgical techniques, improved implants design, and the development of biologic adjuvants, nonunions still occur and represent a challenging diagnostic and clinical problem. Femoral nonunion also represent a serious socioeconomic challenge resulting in prolonged patient morbidity, gait abnormality, multiple operations as well as high resource utilization, emotional impairment and ultimately a prolonged inability to return to work.

A systematic approach to the evaluation and treatment of femoral nonunions is required for a successful outcome because of the multifactorial nature of the problem. The origin of the process requires the diligent investigation of the fracture, fixation strategy previously employed, signs of infection, and critical evaluation of the characteristics of the nonunion. Once the fracture has been diagnosed, the nonunion must be characterized in order to identify the factors that prevented fracture union. When the personality of the nonunion has become clear, a surgical plan can be developed. A large number of tools are currently available in the orthopedic armamentarium including dynamization, plate osteosynthesis , external fixation, exchange nailing, adjuvant such as electrical or ultrasound stimulation, bone grafting with autogenous or allogenic bone, and biologic agents.

Despite advances in technology and technique, current union rates range from 50 to 100% of patients, which is significantly lower than previously suggested [9, 10]. Surgeons must counsel patients accurately and make every effort to select the optimal intervention for each unique situation. This chapter will discuss the epidemiology of femoral shaft nonunions and examine briefly the socioeconomic impact, as well as briefly discuss the etiology of nonunions with special attention to those factors, which are unique to femoral shaft injuries. The diagnosis of femoral shaft fractures including history, physical examination, and laboratory and imaging work up will be addressed. Finally, the treatment option will be discussed as well as a brief note on adjuvant treatments including biologic agents. The last section will contain a few case examples that illustrate some of the challenges faced when treating femoral shaft nonunions.

## 10.2 Epidemiology

The annual incidence of midshaft femur fractures is approximately 10–37 per 100,000 person per years with the incidence peaking among the young and then again in the elderly [11, 12]. Current estimates place femoral shaft

nonunion rates above humeral shaft nonunions, but lower than tibial shaft nonunions [13]. Extrapolated from those statistics using a 1–9% nonunion rate and the current population in the USA of 313 million then each year between 310 and 2790, patients in the USA will go onto a femoral shaft nonunion [14].

Fracture nonunions represent a difficult challenge for the surgeon, the patient, and thus also to the health system and the social services supporting them. Their average treatment management requires large assets and long-lasting therapies. Recent estimates have placed treatment of femoral nonunions at $28,970 utilizing exchange nailing without biologic adjuvant [13]. This provides a starting point; however, other surgical techniques, infectious etiology, and reoperation would all drastically increase the overall costs. Moreover, it is estimated that the indirect costs for musculoskeletal conditions represent about 80% of the total costs of these conditions and can be highly variable [15]. Utilizing this data, the cost of treating femoral shaft nonunions ranges from 9 to 80. One million dollars annually with this likely being at the low end of the spectrum.

## 10.3 Etiology

Various predisposing and contributing factors have been identified that predispose a patient to nonunion. Identification of these factors is a critical element of the evaluation of femoral shaft nonunions in order to create a surgical plan that corrects the offending variables. Female gender and age have been linked to decreased healing and potential increase in nonunion rate secondary to deceased estrogen levels and biologic activity, respectively [16]. Metabolic and endocrine abnormalities are well-established etiologies of nonunions. A recent series of patients with unexplained nonunions found 83% of patients were found to have a previously undiagnosed metabolic or endocrine abnormality after being evaluated by an endocrinologist; however, interestingly, the rate was 100% in the subset of patients with femoral shaft nonunions underscoring the importance of investigating this group of patients [17]. Additionally, cigarette use, medical comorbidities, and NSAIDs use have all been correlated to and increased risk of nonunion [18–21].

Fracture-specific characteristics can also increase the risk of nonunion in femoral shaft fractures. Disruption of the soft tissue envelope through either an open fracture [22] or open reduction during intramedullary nailing [21]

has been shown to increase the risk of nonunion. The degree of fracture comminution has also been shown to increase the risk of nonunion in open femur fractures with the assumption that a greater degree of periosteal and soft tissue damage has occurred [23] resulting in impaired physiology at the fracture site [24]. The presence of a fracture gap has also been indicated to increase the nonunion risk [25] with the caveat that this variable has to be taken in context with the fracture type (simple > comminuted) and fixation strategy (compression or nail > bridge or external fixator). Infection has been well documented to increase the risk of nonunion in all fracture types and is most applicable in femoral shaft fractures after open fractures and prolonged open reductions.

The surgical techniques utilized also play a role in femoral shaft fracture healing. Although there are many techniques to stabilize femoral shaft fractures, the literature is most clear in addressing femoral shaft fracture healing when these fractures are treated with a femoral nail. For example, in regard to femoral shaft fracture nailing technique, the femoral shaft fracture union rates are similar with union rates near equal at 100% for antegrade nailing and 98% for retrograde nailing [26]. According to a systematic literature review, reamed femoral nailing has a higher union rate when compared to unreamed femoral nailing [27].

## 10.4 Diagnosis

As with all clinical problems, the starting point for evaluation is a thorough history. Target areas include a detailed discussion of the original injury with notes on the mechanism, time to treatment, postoperative course, and potential signs of infection such as wound healing issues or fevers. A concerted effort to gather records from previous surgeries can be extremely valuable in understanding the original injury and fixation strategy. The most important component of the history is pain with ambulation, with careful attention paid to the temporal association, character, and duration. Demographic information must also be considered as well as medical comorbidities because both major factors can increase the prevalence of nonunions.

The physical exam should focus on four main components, the soft tissue envelope, the motion at the fracture site, the deformity, and the signs of infection. Evaluation of the soft tissue envelope should provide information

regarding both the original injury (open vs. closed), previous soft tissue reconstruction, reduction and fixation strategy (open vs. closed reduction), and a careful exam of the vascular status of the extremity. Motion at the fracture site will most often result in pain with ambulation, and this is uniquely sensitive for femoral shaft nonunion. In the developing world, union is often defined by a simple clinical test called “squat and smile” as a surrogate for radiographic union [28]. Evaluation of the deformity of the extremity enables the surgeon to create a successful operative plan. Deformity is often rotational, but can be varus/valgus or a length discrepancy. Finally, the surgeon should have a high degree of suspicion for femoral shaft nonunions to be infected with careful inspection to assess warmth, induration, sinus tracts, fluctuate, and tenderness to palpation.

Radiographic evaluation should include orthogonal views of the femur with additional views to establish the mechanical axis of the entire extremity (hip to ankle standing, tibia AP, and Lateral). On occasion, computed tomography (CT) is useful to analyzed the presence and amount of bridging callus as well as quantifying rotational deformities. While data is limited, CT has been shown to improve diagnostic accuracy in tibial nonunions [29].

Laboratory evaluation for patients with femoral shaft nonunions has two main purposes. The exclusion of infection is a multifactorial process; however, preoperative laboratory analysis should include an erythrocyte sedimentation rate, C-reactive protein level, and complete blood count with differential. While not routinely utilized, the literature has suggested some value exists utilizing radionuclide and indium 111–labeled leukocyte scans in equivocal cases [30]. The gold standard to diagnosis infection continues to be tissue culture and all antibiotics should be discontinued 7–14 days prior to collection [31].

## 10.5 Classification

Nonunions are classified according to the criteria described by Müller [32] and Weber [33]. This classification divides nonunions into viable or nonviable subtypes. Viable nonunions are defined as having biologic evidence of healing such as bone bleeding, bony soft tissues attachments, and nonsclerotic bony edges.

Hypertrophic nonunion s are well vascularized and show evidence of an attempt to heal the fracture site. Radionuclide scans will be hot secondary to

callus formation. This type of nonunion typically results from a fixation strategy that allows gross motion at the fracture site. Hypertrophic nonunions are subdivided into:

1. ‘Elephant foot’ nonunions: These nonunions lay down abundant callus.

2. ‘Horse hoof’ nonunions: Moderate callus and possibly signs of bone sclerosis.

3. Oligotrophic nonunions : Osteogenic activity is minimal at the fracture site, minimal to no callus formation, and differ from atrophic subcategory based on intact blood supply.

Atrophic nonunion s show little or no evidence to heal the fracture site typically without any callus formation. The fracture site forms sclerotic mottled bone ends and biologic activity is minimal. These types of nonunions require both an improved fixation strategy as well as biologic augmentation. Radionuclide scans demonstrate cold secondary to osteologic ischemia. Atrophic nonunion s are subdivided into:

1. Torsion wedge nonunions: Fracture fragment with reduced blood supply and no evidence of callus formation.

2. Comminuted nonunions: typically seen with at least one necrotic, with no evidence of callus formation.

3. Defect nonunions: Segmental bone loss makes fracture healing nearly impossible. May be viable or nonviable at the bone ends.

4. Atrophic nonunion s: Fibrous tissue is formed between atrophic bone at the fracture site.

---

## 10.6 Treatment Options

The overall goal of treating femoral shaft nonunions is to achieve bony

healing, however there are other considerations as well including, but not limited, to curing infection, correction of malalignment, rehabilitating the patient, and return to previous functional level. The treatment strategy should consider all the goals for the patient and maybe altered secondarily to one component. The patient factors must be accounted for and every effort should be made to optimize medical comorbidities and nutritional status. In addition to optimizing the host status, any substance that interferes with osteogenic activity should be stopped (NSAIDs, cigarette use). A number of treatment options exist and will briefly be discussed including dynamization, conversion of a plate to nail, exchange nailing, plate augmentation, plate osteosynthesis , external fixation, and adjuvant alternatives.

### 10.6.1 Dynamization

Dynamization is the removal of those interlocking screws creating a length unstable nail and theoretically allowing compression at the fracture site with weight bearing [34]. Typically, a screw is left in an oblong whole on the dynamized end of the nail to provide rotational control of the segment. While tibial shaft fractures have seen improved outcomes with this technique [35], outcomes in femoral shaft nonunions is mixed [36, 37]. The literature reports a union rate of approximately 50% [38], although numerous complications have been seen with this technique. Shortening greater than 2 cm has been reported in up to 20% of patients underscoring the importance of a careful analysis of fracture pattern and identification of length stable injuries [34, 36, 37]. These patients require close follow-up, and this technique provides the best results with early utilization.

### 10.6.2 Plate Conversion to a Nail

Revising a compression or bridge plate to a reamed intramedullary nail has been reported with excellent outcomes. The advantages include open access to the fractures site for correction and removal of any fibrous tissue as well as biologic activity of the reamings and relative stability of the intramedullary nail. Outcomes have reported as union rates ranging from 91 to 100% with an average time to union of 6 months [39–42]. The addition of autologous bone graft did not improve outcomes further demonstrating the beneficial biologic activity of intramedullary reamings [42]. This technique has limited utility in the USA where the vast majority of adult femoral shaft fractures are treated

primarily with intramedullary nails.

### 10.6.3 Exchange Nailing

Exchange nailing refers to the technique of removing the previous intramedullary implant, reaming the medullary canal to a larger diameter and implanting a second intramedullary nail with a significantly larger diameter. The benefits of this technique include improved biomechanics at the fracture site with a larger nail ($r^4$) and improved endosteal contact through isthmic elongation, augmentative biologic activity of the intramedullary reamings, preservation of the soft tissue envelope, and early weight bearing. The amount of reaming deposited at the fractures site has been speculated to be limited, and therefore, a role for open bone grafting in some situations may exist [43].

Outcomes from exchange nailing have been mixed with union rates reported from 53 to 100% [44–46]. A recent systematic review demonstrated 343 patients treated with exchange nailing in 11 studies averaged a union rate of 73% ($n$ = 251) at an average of 7 months [47]. Efforts should be made to increase the diameter of the new nail by 2 mm typically necessitating reaming the canal 1.5 mm larger than the anticipated implant. While exchange nailing is clearly not the previously described panacea for all femoral shaft nonunions, it does provide some clear advantages to more invasive treatments. This technique is recommended for viable nonunion in particular because adequate biologic activity will lead to union with improved biomechanical environment.

### 10.6.4 Augmentative Plate Fixation

Femoral shaft nonunions that result from fracture patterns with extensive comminution and large segmental defects or metaphyseal–diaphyseal location have had poor outcomes with exchange nailing [44, 48]. Recently, the concept of plate augmentation has been introduced for nonunions [49, 50]. The benefits of this technique capitalize on the load-sharing properties of the nail while adding the compression and rotational control created by addition of the plate as well direct access to the fracture site. The development of locked plates has simplified the surgeon’s ability to utilize the technique of augmentative plating through reliable unicortical screw fixation.

Outcomes in the literature from augmentative plate fixation for femoral nonunions are limited. A recent systemic review of femoral shaft nonunions demonstrates an average union rate of 96% with an average time to union of 6 months [47, 50, 51]. Challenges with this technique are chiefly the disruption of the soft tissue envelope and the inability to correct any pre-existing deformity.

### 10.6.5 Plate Fixation

Weber and Čech advocate debridement with the use of plates for "mechanical rest and massive cancellous autograft" [52]. Muller and Rosen first described the use of the plate compression principle in the treatment of femoral nonunions [32, 53]. Plate osteosynthesis provides advantages over other techniques, specifically compression and direct access to the nonunion site where bone grafting and/or biologic agents can also be supplemented to the plate fixation. In these situations, the surgeon should give consideration to plate osteosynthesis. As previously discussed, plating can be particularly helpful in metadiaphyseal nonunions of the femur where direct endosteal contact cannot be attained. Plate fixation has also been shown to have improved biomechanical profile compared to a retrograde nail with regard to axial and torsional stability [54]. Without question, an infected nonunion can be amenable to debridement, open reduction, and compression with plate fixation and has been reported with successful outcomes in a single-staged procedure [55].

Outcomes from plate fixation have historically been excellent. In one large retrospective series of femoral shaft nonunions utilizing a wave plate, union rates were 98% after a single procedure [56]. A more recent prospective series utilized a subvastus approach and indirect reduction techniques with a AO 95° blade plate and reported 91% union rate at 3 months [57]. While excellent outcomes have been achieved with plate osteosynthesis, there are some considerable downsides. Compared with closed medullary nailing, plate osteosynthesis has a higher risk for infection, greater blood loss, and further devascularization to soft tissues [58, 59]. A further challenge with plate fixation is patient compliance with restricted postoperative weight bearing which can also lead to decreased functional rehabilitation and stiffness postoperatively [60].

### 10.6.6 External Fixation

The circular external fixators (Ilizarov) have also been used for the treatment of femoral nonunions While small case series have been reported with good results in aseptic nonunions [61], the principal role of this type of fixation is primarily for infected nonunions [62, 63]. Cyclic compression and distraction has been retrospectively investigated using Ilizarov techniques. Union was achieved in an average of 5.8 months; however, the authors noted that pain control and pin-related complications were a major limitation of this technique [64]. Advantages of the Ilizarov technique include percutaneous application, minimal blood loss, correction of the deformity in three planes, and leg length discrepancies, while allowing the patient early weight bearing. Disadvantages of this technique include emotional challenges for psychological impaired patients, pin tract infections, and risks of neurovascular injury at the time of wire insertion. The technical challenge and economic costs estimated at $50,607 per patient relegates the utility of this technique primarily to tertiary referral centers [65].

### 10.6.7 Masquelet Technique

The clinical management of segmental bone defects in femoral shaft nonunions is a particularly challenging situation. Current treatment options include the use of autografts for defects <5 cm; however, techniques for larger defects require treatments with high morbidity such as vascularized bone grafting or intercalary bone transport [66–69]. Masquelet and colleagues [70, 71] reported successful repair of wide diaphyseal defects >25 cm with concurrent severe soft tissue loss in patients utilizing a technique of induced membrane and autologous cancellous bone grafting in a two-step surgical procedure. This technique was initially described in 1986 for the reconstruction of extensive diaphyseal bone loss up to 25 cm in length with an associated severe soft tissue injury [70]. This technique allows the reconstruction of large diaphyseal defects even in the face of irradiated or infected recipient site [72]. The first stage consists of complete surgical debridement of all necrotic tissue followed by the insertion of a polymethylmethacrylate cement spacer into the defect, which leads to a mild foreign body reaction and the formation of a membrane. After the soft tissue has stabilized and there are no signs of infection, the second stage involves removing the spacer and bone grafting. This stage can be preformed at a

variable time from 2 to 6 months after the first stage. Careful incision of the membrane and removal of the cement spacer is then followed by bone grafting. Masquelet initially described cancellous autograft for bone grafting; however, many strategies have been advocated including intramedullary reamings, allograft, and combinations of the previously listed sources [73]. The graft is carefully placed into the membrane and the membrane is then sutured closed to create a confined compartment. The application of this technique has been reported for femoral shaft nonunions with a union rate of 88% in patients with segmental defects ranging from 1 to 25 cm [74].

## 10.7 Biologic Augmentation

### 10.7.1 Adjuvant Treatments

In addition to nonunion fixation strategies, adjuvant therapy can promote union and improve biologic activity of nonviable nonunions. Adjuvant modalities include electrical stimulation, bone grafting and bone graft substitutes, and bone morphogenic proteins.

While these modalities can be employed in isolation, they are almost exclusively utilized to complement a previously described technique. Electrical stimulation in isolation has shown improved union rates; however, the data is limited in femoral shaft fractures [75, 76]. Autogenous bone, allograft bone, bone marrow aspirate, and BMPs, independently or in combination to nonunion sites, can improve the biology of the local environment. These adjuvants are typically utilized in nonviable nonunions or in cases with bone loss in combination with a revised fixation strategy. While isolated bone grafting has been reported in the literature, this technique is rarely utilized at our institution [77] and has not shown a benefit when converting fixation from a plate to intramedullary nail for femoral shaft nonunions [78]. BMP has been studied in both animal models and clinical trials, and while the indications remain controversial, it has been evaluated in the treatment of femoral shaft nonunions. A retrospective review of 30 patients who had atrophic aseptic femoral shaft nonunions and reported union rate of 87% at median period of 6 months [79].

## 10.8 Summary

Femoral shaft nonunions are a debilitating clinical problem for patients as well as a diagnostic and technical challenge for orthopedic surgeons. Patients are burdened with gait abnormality, inability to return to work, re-operations, and psychological impairment; while care for these patients take a tremendous amount of resources and burdens hospitals systems with tremendous costs. A systematic approach to the evaluation and treatment of femoral nonunions is required for a successful outcome because of the complexity of the problem.

Diligent investigation of the original fracture, previous fixation strategy, and critical evaluation of the characteristics of the nonunion are paramount to develop a successful treatment algorithm. At this time, there is no golden standard for femoral nonunions and each patient must be approached as a unique situation requiring a unique solution.

## 10.9 Case Discussions

**Case 1**

59-year-old female that suffered an initial Type IIIA open segmental femur fracture that was treated with a variety of techniques including initial external fixation, Masquelet technique, plating and bone grafting, intramedullary nail fixation, and bone grafting. Femur healed after 12 months of treatments that required intramedullary nail fixation and bone grafting as final treatment (Figs. 10.1, 10.2, 10.3, 10.4, 10.5 and 10.6).

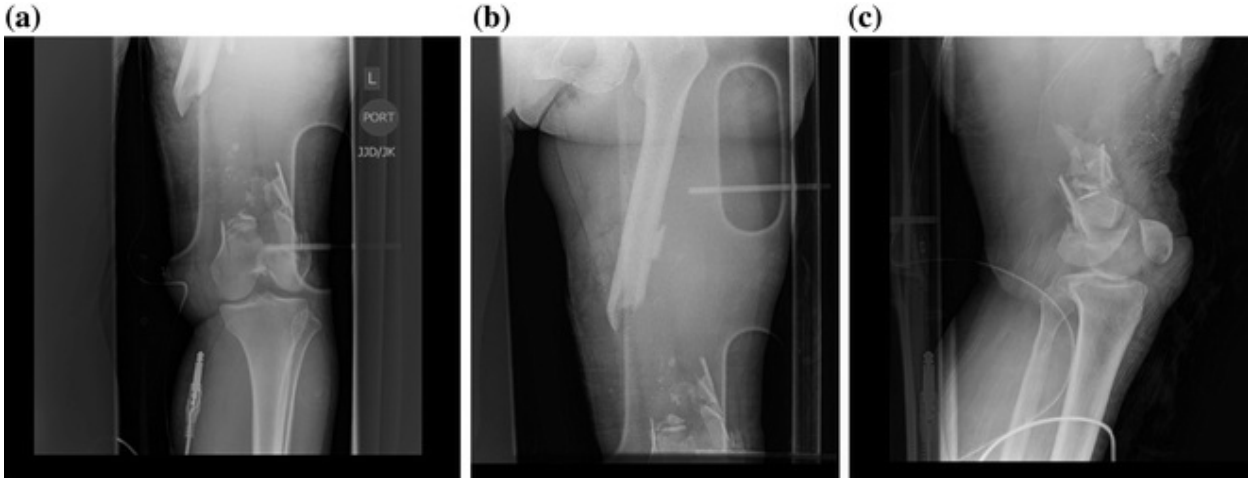



***Fig. 10.1*** Initial injury X-rays **(a–c)**

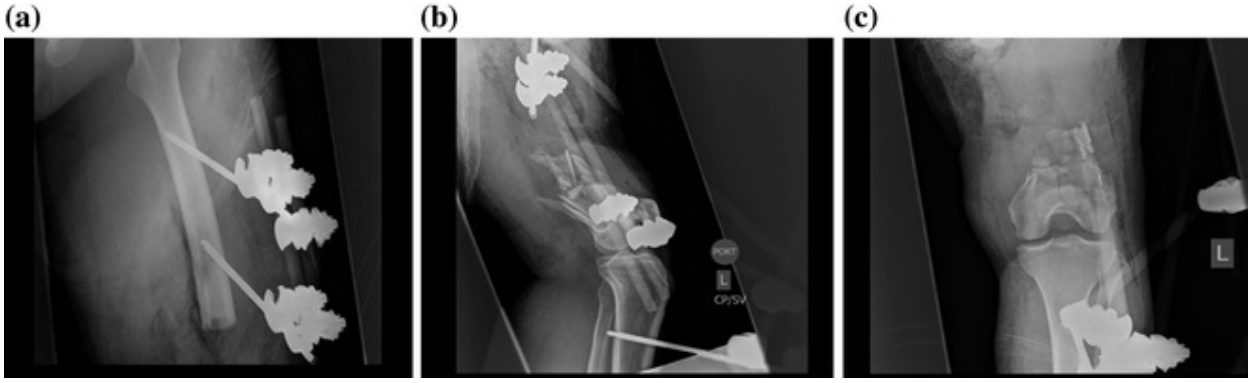



***Fig. 10.2*** Post-initial debridement and external fixation (**a**–**c**)

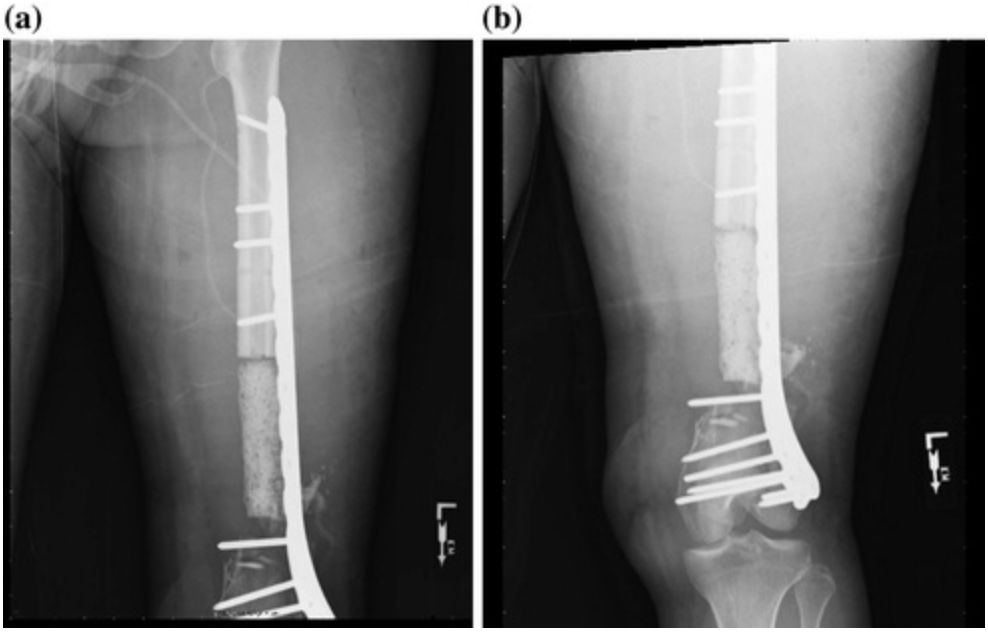



***Fig. 10.3*** Initial Masquelet technique after debridements (**a**, **b**)

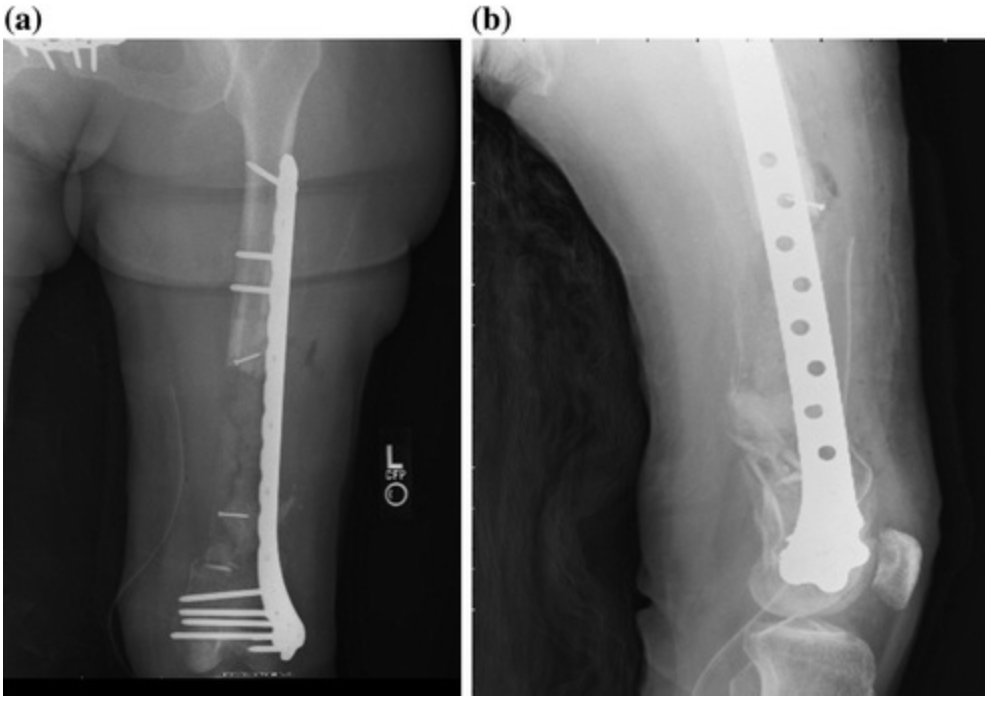



***Fig. 10.4*** Massive bone grafting using the RIA or reamer-irrigator-aspirator (DePuy Synthes, Warsaw IN, USA) and bone morphogenetic protein-2 (off label) (Medtronic, Memphis TN, USA) (**a**, **b**)

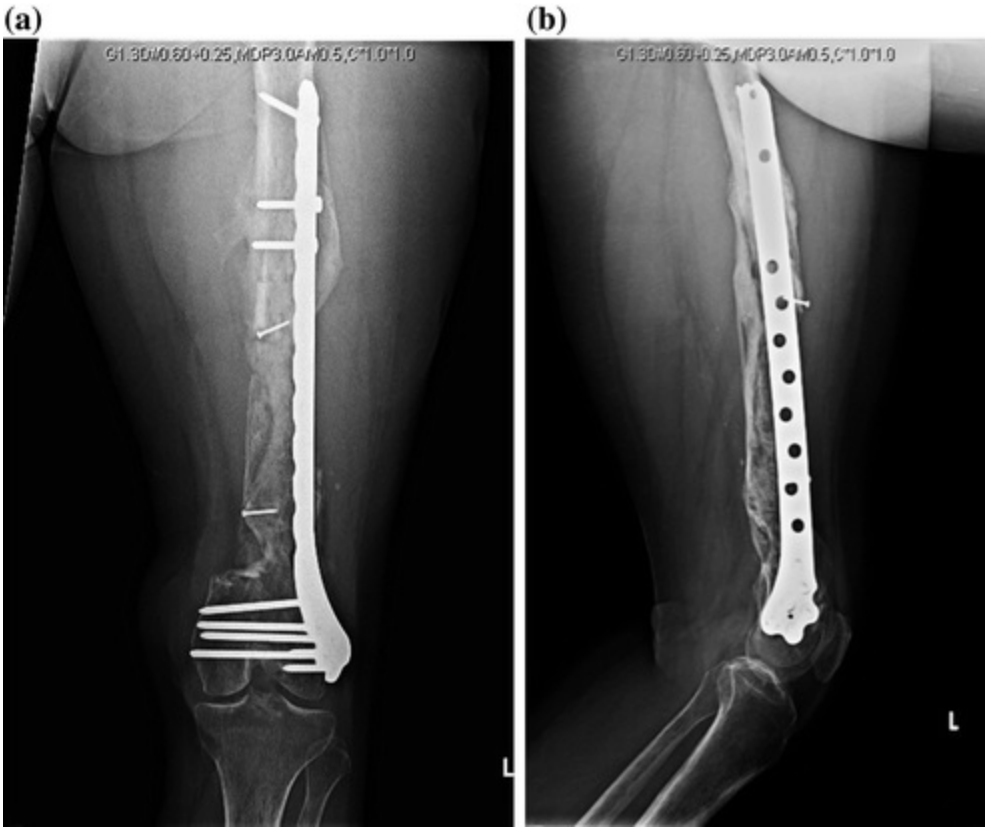


***Fig. 10.5*** Femur shaft nonunion at 8 months postbone grafting **(**a, **b)**

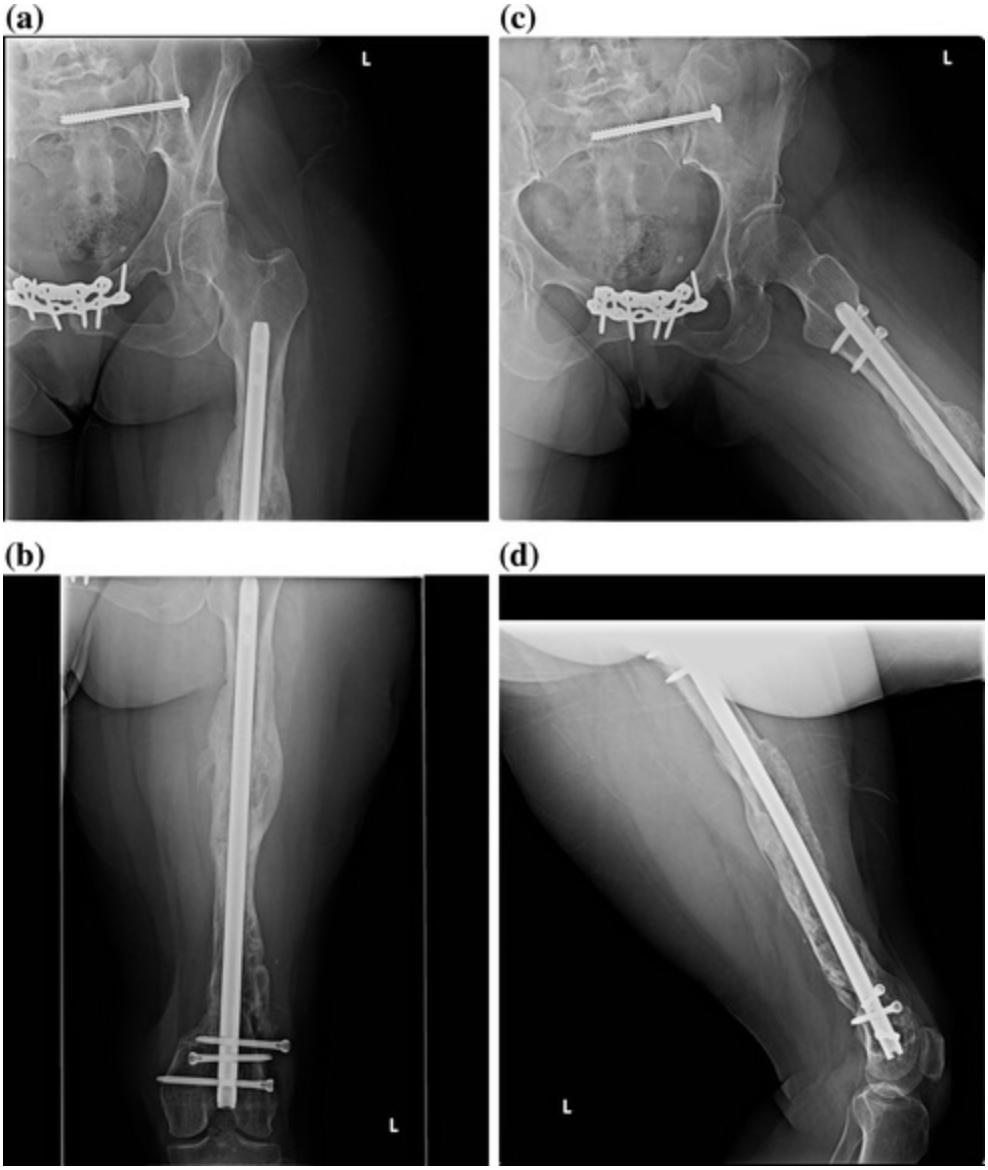



***Fig. 10.6*** Femur shaft union after removal of plate, intramedullary nailing, and combination of autograft and allograft **(a**–**d)**

## Case 2

18-year-old male with femoral shaft nonunion treated with a variety of techniques including initial intramedullary nail fixation, exchange intramedullary nailing and bone grafting, compression plating, and bone grafting. Femur healed after 10 months of treatments that required compression plating, intramedullary nail exchange, and bone grafting as final treatment (Figs. 10.7, 10.8, 10.9, 10.10 and 10.11).

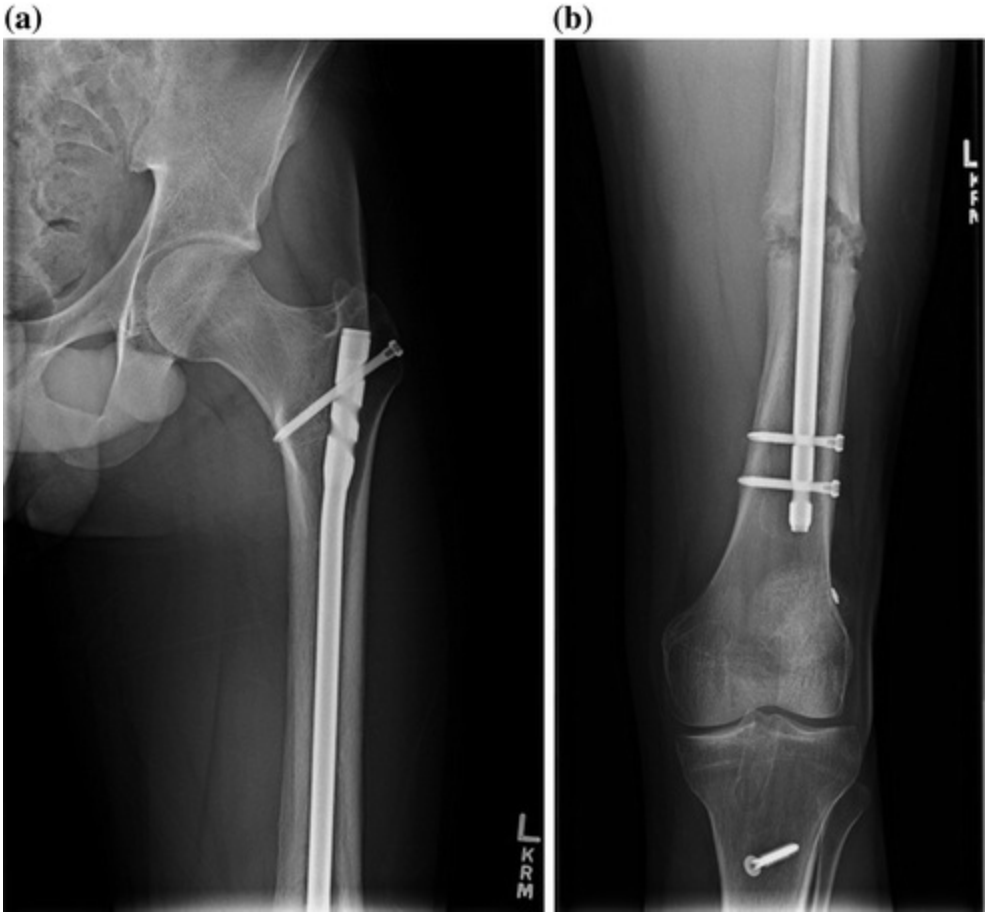



***Fig. 10.7*** Initial femur postoperative X-ray s at 3 months with nonunion (**a**, **b**)

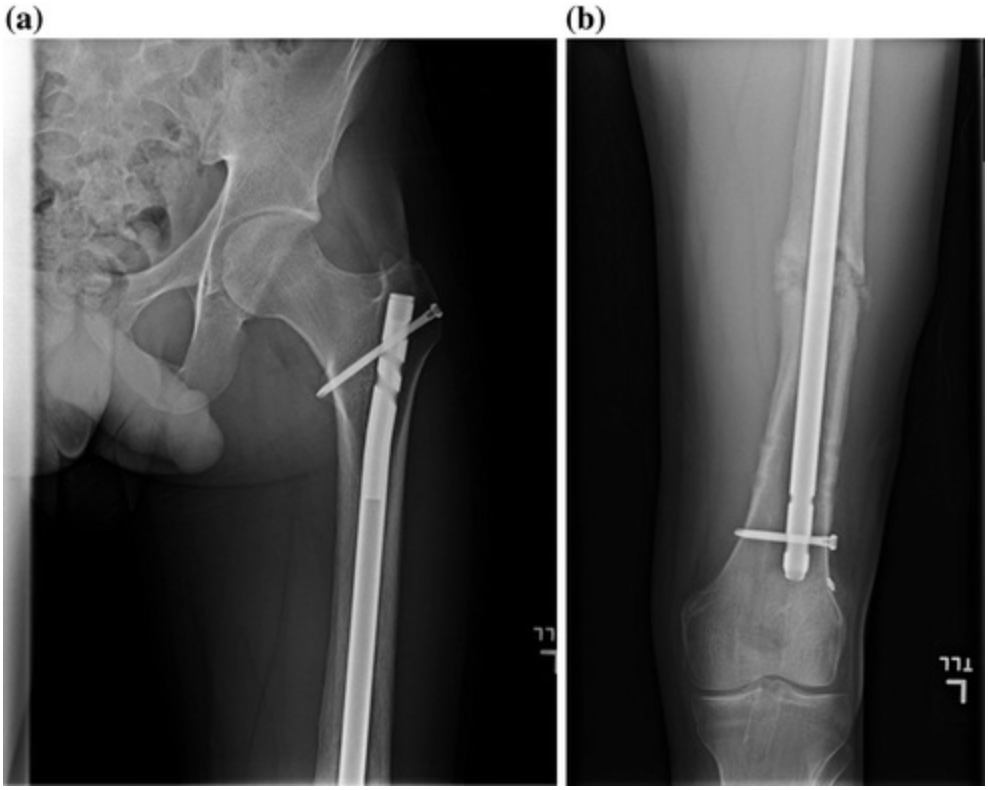



***Fig. 10.8*** Femur X-rays after exchange nailing and bone grafting (**a**, **b**)


***Fig. 10.9*** Femur X-ray after nail dynamization

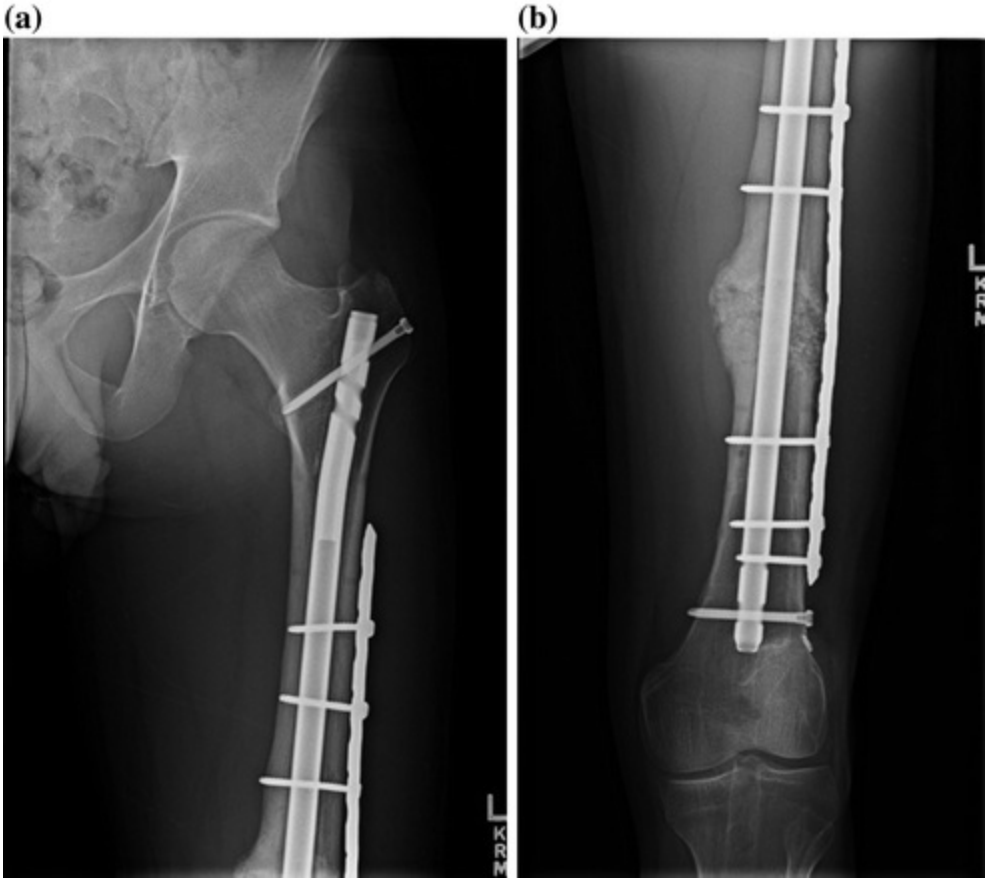



***Fig. 10.10*** Immediate postoperative femur X-rays after compression plating in addition to previous exchange nailing and bone grafting with nail dynamization (**a**, **b**)

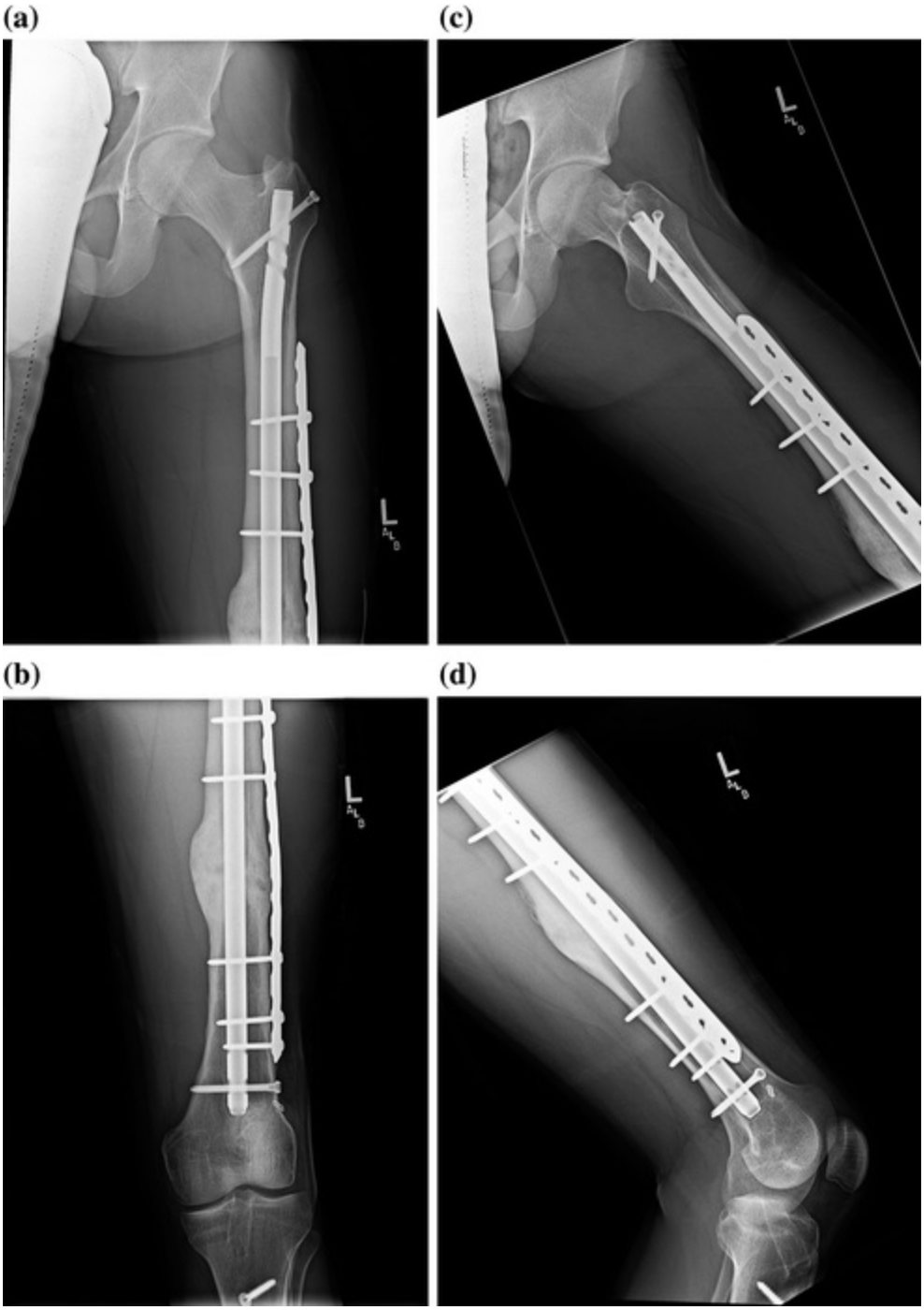



***Fig. 10.11*** Femur shaft union after compression plating in addition to previous exchange nailing and bone grafting with nail dynamization (**a**–**d**)

# References

1. Küntscher G, Maatz R. Technik der marknagelung. Leipzig: Georg Thieme Verlag; 1945. [Book in German].

2. Canadian Orthopaedic Trauma Society. Nonunion following intramedullary nailing of the femur with and without reaming: results of a multicenter randomized clinical trial. J Bone Joint Surg Am. 2003;85-A(11):2093–6.

3. Winquist RA, Hansen ST Jr, Clawson DK. Closed intramedullary nailing of femoral fractures: a report of five hundred and twenty cases. J Bone Joint Surg Am. 1984;66(4):529–39. [Crossref][PubMed]

4. Wolinsky PR, McCarty E, Shyr Y, Johnson K. Reamed intramedullary nailing of the femur: 551 cases. J Trauma. 1999;46(3):392–9. [Crossref][PubMed]

5. Babhutkar S, Pande K. Nonunion of the diaphysis of long bones. Clin Orthop Relat Res. 2005;431:50–6. [Crossref]

6. Finkemeier CG, Chapman MW. Treatment of femoral diaphyseal nonunions. Clin Orthop Relat Res. 2002;398:223–34. [Crossref]

7. Furlong AJ, Giannoudis PV, DeBoer P, Matthews SJ, MacDonald DA, Smith RM. Exchange nailing for femoral shaft aseptic non-union. Injury. 1999;30(4):245–9. [Crossref][PubMed]

8. Giannoudis PV, Matthews SJ, Smith RN. Exchange nailing for femoral nonunion. J Orthop Trauma. 2000;14(7):522–3. [Crossref][PubMed]

9. Kempf I, Grosse A, Rigaut P. The treatment of noninfected pseudarthrosis of the femur and tibia with locked intramedullary nailing. Clin Orthop Relat Res. 1986;212:142–54.

10. Webb LX, Winquist RA, Hansen ST. Intramedullary nailing and reaming for delayed union or nonunion of the femoral shaft: a report of 105 consecutive cases. Clin Orthop Relat Res. 1986;212:133–41.

11. Weiss RJ, Montgomery SM, Al Dabbagh Z, Jansson KA. National data of 6409 Swedish inpatients with femoral shaft fractures: stable incidence between 1998 and 2004. Injury. 2009;40(3):304. [Crossref][PubMed]

12. Arneson TJ, Melton LJ 3rd, Lewallen DG, O'Fallon WM. Epidemiology of diaphyseal and distal femoral fractures in Rochester, Minnesota, 1965–1984. Clin Orthop Relat Res. 1988;234:188–94.

13. Kanakaris N, Giannoudis PV. The health economics of the treatment of long-bone non-unions. Injury, Int J Care Injured. 2007;38(Suppl 2):S77–84.

14. Norris BL, Nowotarski PJ. Femoral shaft fractures. In: Stannard JP, Schmidt AH, editors. Kregor PJ, editors. Surgical treatment of orthopedic trauma. New York: Thieme; 2007. p. 611–32.

15. Bozic KJ, Rosenberg AG, Huckman RS, Herndon JH. Economic evaluation in orthopaedics. J

Bone Joint Surg Am. 2003;85A(1):129–42.

16. Calori GM, Albisetti W, Agus A, Iori S, Tagliabue L. Risk factors contributing to fracture non-unions. Injury. 2007;38(Suppl 2):S11–8.
[Crossref][PubMed]

17. Brinker MR, O'Connor DP, Monla YT, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trauma. 2007;21(8):557–70.
[Crossref][PubMed]

18. McKee MD, DiPasquale DJ, Wild LM, Stephen DJ, Kreder HJ, Schemitsch EH. The effect of smoking on clinical outcome and complication rates following Ilizarov reconstruction. J Orthop Trauma. 2003;17(10):663–7.
[Crossref][PubMed]

19. Schmitz MA, Finnegan M, Natarajan R, Champine J. Effect of smoking on tibial shaft fracture healing. Clin Orthop Relat Res. 1999;365:184–200.
[Crossref]

20. Giannoudis PV, MacDonald DA, Matthews SJ, Smith RM, Furlong AJ, De Boer P. Nonunion of the femoral diaphysis: the influence of reaming and non-steroidal anti-inflammatory drugs. J Bone Joint Surg Br. 2000;82(5):655–8.
[Crossref][PubMed]

21. Malik MH, Harwood P, Diggle P, Khan SA. Factors affecting rates of infection and nonunion in intramedullary nailing. J Bone Joint Surg Br. 2004;86(4):556–60.
[PubMed]

22. Arslan H, Subaşý M, Kesemenli C, Ersuz H. Occurrence and treatment of nonunion in long bone fractures in children. Arch Orthop Trauma Surg. 2002;122(9–10):494–8.
[Crossref][PubMed]

23. Noumi T, Yokoyama K, Ohtsuka H, Nakamura K, Itoman M. Intramedullary nailing for open fractures of the femoral shaft: evaluation of contributing factors on deep infection and nonunion using multivariate analysis. Injury. 2005;36(9):1085–93.
[Crossref][PubMed]

24. Triffin P, Greg PJ. Depression of bone blood flow after brunt trauma. Acta Orthop Scand. 1994;65(2):195–8.
[Crossref]

25. Pihlajamäki HK, Salminen ST, Böstman OM. The treatment of nonunions following intramedullary nailing of femoral shaft fractures. J Orthop Trauma. 2002;16(6):394–402.
[Crossref][PubMed]

26. Ostrum RF, Agarwal A, Lakatos R, Poka A. Prospective comparison of retrograde and antegrade femoral intramedullary nailing. J Orthop Trauma. 2000;14(7):496–501.
[Crossref][PubMed]

27. Duan X, Li T, Mohammed AQ, Xiang Z. Reamed intramedullary nailing versus unreamed intramedullary nailing for shaft fracture of femur: a systematic literature review. Arch Orthop

Trauma Surg. 2011;131(10):1445–52.
[Crossref][PubMed]

28. Zirkle LG Jr. Diversity drives design. American Academy of Orthopaedic Surgeons. AAOS Now. 2012;6(8):47. http://www.aaos.org/news/aaosnow/aug12/youraaos7.asp. Accessed 12 Feb 2016.

29. Bhattacharyya T, Bouchard KA, Phadke A, Meigs JB, Kassarjian A, Salamipour H. The accuracy of computed tomography for the diagnosis of tibial nonunion. J Bone Joint Surg Am. 2006;88(4):692–7.
[PubMed]

30. Nepola JV, Seabold JE, Marsh JL, Kirchner PT, el-Khoury GY. Diagnosis of infection in ununited fractures: combined imaging with indium-111-labeled leukocytes and technetium-99 m methylene diphosphonate. J Bone Joint Surg Am. 1993;75(12):1816–22.

31. Gristina AG, Naylor PT, Webb LX. Molecular mechanisms in musculoskeletal sepsis: the race for the surface. Instr Course Lect. 1990;39:471–82.
[PubMed]

32. Müller ME. Treatment of nonunions by compression. Clin Orthop Relat Res. 1965;43:83–92.
[PubMed]

33. Weber BG, Čech O. Pseudoarthrosis: pathology, biomechanics, therapy, results. Berne, Switzerland: Hans Huber Medical Publisher; 1976.

34. Yokota H, Tanaka SM. Osteogenic potentials with joint-loading modality. J Bone Miner Metab. 2005;23:302–8.
[Crossref][PubMed]

35. Richardson JB, Gardner TN, Hardy JR. Dynamization of tibial fractures. J Bone Joint Surg Br. 1995;77(3):412–6.
[Crossref][PubMed]

36. Wu CC, Lee ZL. Low success rate of non-intervention after breakage of interlocking nails. Int Orthop. 2005;29(2):105–8.
[Crossref][PubMed][PubMedCentral]

37. Pihlajamaki HK, Salminen ST, Bostman OM. The treatment of non-unions following intramedullary nailing of femoral shaft fractures. J Orthop Trauma. 2002;16(6):394–402.
[Crossref][PubMed]

38. Wu CC, Chen WJ. Healing of 56 segmental femoral shaft fractures after locked nailing: poor results of dynamization. Acta Orthop Scand. 1997;68(6):537–40.
[Crossref][PubMed]

39. Wu CC, Shih CH, Chen WJ. Effect of reaming bone grafting on treating femoral shaft aseptic non-union after plating. Arch Orthop Trauma Surg. 1999;119(5–6):303–7.
[Crossref][PubMed]

40. Wu CC. Treatment of femoral shaft aseptic non-union associated with plating failure: emphasis on the situation of screw breakage. J Trauma. 2001;51(4):710–3.

[Crossref][PubMed]

41. Megas P, Syggelos SA, Kontakis G. Intramedullary nailing for the treatment of aseptic femoral shaft non-unions after plating failure: effectiveness and timing. Injury. 2009;40(7):732–7.
[Crossref][PubMed]

42. Emara KM, Allam MF. Intramedullary fixation of failed plated femoral diaphyseal fractures: are bone grafts necessary? J Trauma. 2008;65(3):692–7.
[Crossref][PubMed]

43. Furlong AJ, Giannoudis PV, DeBoer P. Exchange nailing for femoral shaft aseptic non-union. Injury. 1999;30(4):245–9.
[Crossref][PubMed]

44. Weresh MJ, Hakanson R, Stover MD, Sims SH, Kellam JF, Bosse MJ. Failure of exchange reamed intramedullary nails for ununited femoral shaft fractures. J Orthop Trauma. 2000;14(5):335–8.
[Crossref][PubMed]

45. Hak DJ, Lee SS, Goulet JA. Success of exchange reamed intramedullary nailing for femoral shaft nonunion or delayed union. J Orthop Trauma. 2000;14(5):178–82.
[Crossref][PubMed]

46. Yu CW, Wu CC, Chen WJ. Aseptic nonunion of a femoral shaft treated using exchange nailing. Chang Gung Med J. 2002;25(9):591–8.
[PubMed]

47. Somford MP, van den Bekerom MP, Kloen P. Operative treatment for femoral shaft nonunions, a systematic review of the literature. Stat Traum Limb Recon. 2013;8:77–88.
[Crossref]

48. Banaszkiewicz PA, Sabboubeh A, McLeod I. Femoral exchange nailing for aseptic non-union: not the end to all problems. Injury. 2003;34(5):349–56.
[Crossref][PubMed]

49. Nadkarni B, Srivastav S, Mittal V. Use of locking compression plates for long bone non-unions without removing existing intramedullary nail: review of literature and our experience. J Trauma. 2008;65(2):482–6.
[Crossref][PubMed]

50. Hakeos WM, Richards JE, Obremskey WT. Plate fixation of femoral nonunions over an intramedullary nail with autogenous bone grafting. J Orthop Trauma. 2011;25(2):84–9.
[Crossref][PubMed]

51. Chen CM, Su YP, Hung SH, Lin CL, Chiu FY. Dynamic compression plate and cancellous bone graft for aseptic non-union after intramedullary nailing of femoral fracture. Orthopedics. 2010;33(6):393.
[PubMed]

52. Weber BG, Čech O. Pseudarthrosen. Bern: Hans Huber; 1973. [Book in German].

53. Rosen H. Compression treatment of long bone pseudarthroses. Clin Orthop Relat Res.

1979;138:154–66.

54. Meyer RW, Plaxton NA, Postak PD, Gilmore A, Froimson MI, Greenwald AS. Mechanical comparison of a distal femoral side plate and a retrograde intramedullary nail. J Orthop Trauma. 2000;14(6):398–404.
[Crossref][PubMed]

55. Prasarn ML1, Ahn J, Achor T, Matuszewski P, Lorich DG, Helfet DL. Management of infected femoral nonunions with a single-staged protocol utilizing internal fixation. Injury. 2009;40(11):1220–5.

56. Ring D, Jupiter JB, Sanders RA. Complex non-union of fractures of the femoral shaft treated by wave-plate osteosynthesis. J Bone Joint Surg Br. 1997;79(2):289–94.
[Crossref][PubMed]

57. Bellabarba C, Ricci WM, Bolhofner BR. Results of indirect reduction and plating of femoral shaft nonunions after intramedullary nailing. J Orthop Trauma. 2001;15(4):254–63.
[Crossref][PubMed]

58. Cove JA, Lhowe DW, Jupiter JB, Siliski JM. The management of femoral diaphyseal nonunions. J Orthop Trauma. 1997;11(7):513–20.
[Crossref][PubMed]

59. Rozbruch SR, Müller U, Gautier E, Ganz R. The evolution of femoral shaft plating technique. Clin Orthop Relat Res. 1998;354:195–208.
[Crossref]

60. Abdel-Aa AM, Farouk OA, Elsayed A, Said HG. The use of a locked plate in the treatment of ununited femoral shaft fractures. J Trauma. 2004;57(4):832–6.
[Crossref][PubMed]

61. Brinker MR, O'Connor DP. Ilizarov compression over a nail for aseptic femoral nonunions that have failed exchange nailing: a report of five cases. J Orthop Trauma. 2003;17(10):668–76.
[Crossref][PubMed]

62. Krishnan A, Pamecha C, Patwa JJ. Modified Ilizarov technique for infected nonunion of the femur: the principle of distraction–compression osteogenesis. J Orthop Surg. 2006;14(3):265–72.
[Crossref]

63. Saridis A, Panagiotopoulos E, Tyllianakis M, Matzaroglou C, Vandoros N, Lambiris E. The use of the Ilizarov method as a salvage procedure in infected nonunion of the distal femur with bone loss. J Bone Joint Surg Br. 2006;88(2):232–7.
[Crossref][PubMed]

64. Inan M, Karaogtu S, Cirri F, Turk CY, Harma A. Treatment of femoral nonunions by using cyclic compression and distraction. Clin Orthop Relat Res. 2005;436:222–8.
[Crossref]

65. Patil S, Montgomery R. Management of complex tibial and femoral nonunion using the Ilizarov technique, and its cost implications. J Bone Joint Surg Br. 2006;88(7):928–32.
[Crossref][PubMed]

66. de Boer HH, Wood MB. Bone changes in the vascularized fibular graft. J Bone Joint Surg Br. 1989;71(3):374–8.
[PubMed]

67. Han CS, Wood MB, Bishop AT, Cooney WP 3rd. Vascularized bone transfer. J Bone Joint Surg Am. 1992;74(10):1441–9.
[Crossref][PubMed]

68. Pelissier P, Casoli V, Demiri E, Martin D, Baudet J. Soleus-fibula free transfer in lower limb reconstruction. Plast Reconstr Surg. 2000;105(2):567–73.
[Crossref][PubMed]

69. Green S, Jackson J, Wall D, Marinow H, Ishkanian J. Management of segmental defects by the Ilizarov intercalary bone transport method. Clin Orthop. 1992;280:136–42.

70. Masquelet AC. Muscle reconstruction in reconstructive surgery: soft tissue repair and long bone reconstruction. Langenbecks Arch Surg. 2003;388(5):344–6.
[Crossref][PubMed]

71. Masquelet AC, Fitoussi F, Begue T, Muller GP. Reconstruction of the long bones by the induced membrane and spongy autograft. Ann Chir Plast Esthet. 2000; 45(3):346–53. [Article in French].

72. Pelissier P, Martin D, Baudet J, Lepreux S, Masquelet AC. Behavior of cancellous bone graft placed in induced membranes. Br J Plast Surg. 2002;55(7):598–600.
[Crossref]

73. Giannoudis PV, Faour O, Goff T, Kanakaris N, Dimitriou R. Masquelet technique for the treatment of bone defects: tips-tricks and future directions. Injury. 2011;42(6):591–8.
[Crossref][PubMed]

74. Stafford PR, Norris BL. Reamer-irrigator-aspirator bone graft and Masquelet technique for segmental bone defect nonunions: a review of 25 cases. Injury. 2010;41(Suppl 2):S72–7.
[Crossref][PubMed]

75. Barker AT, Dixon RA, Sharrard WJ, Sutcliffe ML. Pulsed magnetic field therapy for tibial non-union: interim results of a double-blind trial. Lancet. 1984;1(8384):994–6.
[Crossref][PubMed]

76. Sharrard WJ. A double-blind trial of pulsed electromagnetic fields for delayed union of tibial fractures. J Bone Joint Surg Br. 1990;72(3):347–55.
[PubMed]

77. Wu CC, Chen WJ. Treatment of femoral shaft aseptic nonunions: comparison between closed and open bone-grafting techniques. J Trauma. 1997;43(1):112–6.
[Crossref][PubMed]

78. Emara KM, Allam MF. Intramedullary fixation of failed plated femoral diaphyseal fractures: are bone grafts necessary? J Trauma. 2008;65(3):692–7.
[Crossref][PubMed]

79. Kanakaris NK, Lasanianos N, Calori GM, Verdonk R, Blokhuis TJ, Cherubino P. Application of bone morphogenetic proteins to femoral non-unions: a 4-year multicenter experience. Injury.

2009;40(Suppl. 3):S54–61.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_11

# 11. Distal Femoral Nonunions

Animesh Agarwal[1]

(1) Division of Orthopedic Traumatology, Department of Orthopedics, University of Texas Health Science Center, San Antonio, TX, USA

**Animesh Agarwal**
**Email:** Agarwal@uthscsa.edu

**Keywords** Distal femur fractures – Nonunions – Exchange nailing – Bone grafting – Locked plating

## 11.1 Introduction

### 11.1.1 Distal Femur Fractures

Distal femur fractures are defined as those fractures involving the distal 9–15 cm of the femur. They may be entirely extra-articular (AO/OTA Type A), partial articular (AO/OTA Type B) or intra-articular (AO/OTA Type C) [1].

The management of these fractures depends on the type. The type A fractures are generally best managed with either intramedullary (IM) nailing or open reduction and internal fixation (ORIF) [2]. The decision usually is based on the amount of intact distal femur. If there is less than 4 cm, generally ORIF is preferred to obtain adequate fixation in the distal segment. If above 4 cm, then there usually is enough of the distal femur to allow for two interlocking screws with a retrograde nail [3]. Antegrade nailing can be used but meticulous detail must be adhered to, to insure an anatomical restoration of the limb [2]. Type B fractures require ORIF with screws ± small plates as needed since they are partial articular fractures. The type C fractures generally are best managed with ORIF with locking large fragment

plates to allow fixed angle fixation or dedicated fixed angle devices; if the intra-articular component is a simple split, direct reduction of the joint with screw fixation and subsequent retrograde nailing can be performed. In all cases, it is important to realign the mechanical axis of the limb.

Open reduction and internal fixation of the distal femur historically required fixed angle devices [4]. The 95° dynamic condylar screw (DCS) and 95° blade plate provided excellent fixation options for all extra-articular distal femur fractures and select type C fractures with simple intra-articular splits. More comminuted articular fractures required other options such as non-locking condylar plates which often times required a second plate medially to provide sufficient support. These gave way to locking plates, with one of the earliest being the *less invasive stabilization system* (LISS) [4]. Early reports of its use in the management of distal femur fractures were encouraging [5, 6]. Weight and Collinge reported a 100% healing rate at 13 weeks [5]. In a larger series of 103 fractures, Kregor et al. [6] reported a healing rate of 93% without adjunctive bone grafting. They had two nonunions.

### 11.1.2 Incidence of Nonunions

The incidence of nonunions in the management of distal femoral fractures has been reported historically anywhere from 0 to 19% in the literature with variation depending upon the implant used [4]. This was prior to the widespread use of current locked condylar plating systems. As locking plate constructs became more widely used, the rate of nonunion seems to have increased with an incidence as high as 32% [7–9]. Additionally, as the population increases, the number of geriatric distal femur fractures has increased with a nonunion incidence of 24% in this subgroup [10].

### 11.1.3 Ramifications of Nonunions

These distal femoral nonunions can be severely disabling and lead to poor function [11]. Many of these patients have been unable to bear weight through the affected limb for months if not years. These patients also have malalignment that affects the mechanical axis of the lower extremity. They often have a leg length discrepancy due to bone loss from the injury, which is then exacerbated by the multiple procedures that have been performed. However, with proper attention to the principles of nonunion management,

repair of the distal femoral nonunion can lead to healing and improved function [12].

## 11.2 Causes of Nonunions

Chapter 1 reviewed the various risk factors for the development of nonunions. There are, however, factors that have been implicated in nonunion development that are specific to the distal femur. The majority of nonunions are related to the mechanical environment that is created by the fixation construct. Fortunately, the femur has a circumferential soft tissue envelope. However, damage to the soft tissues can occur when the fracture is open. Additionally, periosteal stripping and bone loss can occur in such high-energy injuries. This can provide a significant biological insult and put the patient at risk for the development of a nonunion as well. The surgeon can inadvertently cause additional stripping during fixation. Thus, it is important to minimize soft tissue dissection, especially on the medial side.

### 11.2.1 Mechanical Considerations

Locked plating has been found to be a potential risk factor for the development of nonunion. Healing problems with the use of locked plates have recently been reported as high as 32% [7–9]. Hoffman et al. [7] reported an 18% nonunion rate with locked plating and a 20% recalcitrant nonunion rate after secondary procedures in this subgroup. Henderson et al. [8] retrospectively evaluated a group of 86 distal femur fractures (82 patients) treated with locked plating and found a nonunion incidence of 20% which was much higher than the reported literature at the time. They felt that callus inhibition was occurring from too stiff of a construct. A similar decrease in callus formation when using locking plates for distal femur fractures was seen in comparison to intramedullary nails [13]. Lujan et al. [14] retrospectively evaluated 64 consecutive patients that underwent osteosynthesis of a distal femur fracture with either titanium plates or stainless steel plates. They found that locking constructs did result in asymmetric callus formation, which was inconsistent. Most notably they found that titanium constructs exhibited significantly more callus formation early on up to 12 weeks. Although no increased risk of nonunion with the use of stainless steel implants was seen in this study, others have indicated a

potential relationship. Rodriguez et al. [15], in a multicenter retrospective study, showed that the use of stainless steel plate was an independent risk factor for nonunion. They showed that the probability of intervention for a nonunion was 21% if a stainless steel plate was used initially versus 4% if a titanium plate had been used at the index operation. In a follow-up study, Rodriguez et al. [16] showed a 41% nonunion rate in stainless steel constructs, but only 10% nonunion rate in titanium constructs which was statistically significant. However, the overall nonunion rate was 13.3% indicating that most of the cases in their series were treated with titanium plates (239 T vs. 32 SS). They indicated that the plate material was an independent predictor of nonunion development.

Historically, fixed angle devices , such as the dynamic condylar screw, were the implants of choice for distal femur fractures. The device had excellent results with 0–10% nonunion rates [4]. In a multicenter study by the Canadian Orthopedic Trauma Society, the use of the DCS was revisited [17]. In a prospective randomized controlled trial, the DCS group had a union rate of 91% compared to 52% of the LISS group at 12 months. There was a higher complication and revision rate in the LISS group. A similar implant to the DCS, the 95° angled blade plate was compared to the *locking condylar plate* (LCP) [18]. They showed that the LCP group had more malunions and nonunions, and a statistically higher incidence of complications resulting in significantly more secondary procedures. In a study comparing the use of the LISS plate to that of locking compression plates (LCP) , similar nonunion rates were seen—22.1% (LISS) and 20.7% (LCP) [19].

In an effort to determine the cause of the healing issues, Bottlang et al. [20] evaluated the biomechanics of locked plating in the distal femur. They showed that biomechanically there clearly was asymmetric interfragmentary motion, with the least amount of motion at the cortex adjacent to the plate. Clinically, this was seen as inhibition of callus formation and nonunion development of 19% despite intact hardware. In an attempt to promote callus formation and enhance the mechanical environment with locked plating, they described a technique called far cortical locking. In this technique, the screws locked into the plate but did not engage the near cortex and only obtained fixation in the far cortex. Biomechanically, this reduced the overall stiffness of the construct and promoted interfragmentary motion. In an animal model, they compared far cortical locking to standard locking in a tibia gap model. The far cortical locking group had significantly greater callus formation,

which was symmetric, had stronger calluses and complete healing. In a biomechanical cadaveric distal femur model, the far cortical locking technique showed an 81% decrease in construct stiffness and enables parallel interfragmentary motion. Specially designed far cortical locking screws were used in a prospective observational study by Bottlang et al. [21]. Thirty-two patients with 33 distal femur fractures were treated with this technique, with thirty-one available for follow-up. They had a statistically significant increase in periosteal callus at weeks 12 and 24 when compared to their previous published historic controls [14]. There was only one nonunion in this series. Despite the promising results, this technique has not gained wide acceptance.

It is clear that too stiff of a construct can inhibit fracture healing, but conversely inadequate stability can lead to failure of the construct and a subsequent nonunion. It has been suggested that by using a longer plate (>9 holes in shaft length but with at least 8 holes proximal to the fracture) can minimize plate failure [20]. In a different study, plate length was not found to be predictor of nonunion [16]. Most likely it is a combination of plate length, the number of screws, plate material and the type of screws placed that modulate the healing response.

### 11.2.2 Biological Considerations

An open fracture with resultant bone loss or defect can be a predisposing factor to the development of a nonunion [15, 19, 22]. An open fracture was found to increase the probability of an intervention for nonunion from 21 to 52% for a stainless steel plate and from 4 to 14% for a titanium plate [15]. In a separate study, 37% of open fractures required reoperation for a nonunion, compared to only 10% of closed fractures [22].

An open distal femur fracture is the result of a high-energy injury with resultant comminution, which itself has been suggestive of nonunion [11]. It has been suggested that bone grafting of these highly comminuted injuries should be considered early to help prevent failure of the hardware and subsequent nonunion [23]. Barei and Beingessner [24] bone grafted 55% of distal femurs with bone loss in their series at an average of 70 days, all of which achieved union. Those with bone loss, which did not undergo bone grafting, all healed. All of these were found to have posterior cortical continuity. The presence of posterior cortical continuity despite bone loss indicated that bone grafting was unnecessary. These open fractures also have significant soft tissue disruption, and thus, further insult with extensive

exposures can further disrupt the already compromised soft tissue envelope.

Infection has been reported in 0–10% after ORIF. Many of the same things leading to a nonunion can predispose one to infection. Infection itself has been shown to be a risk factor for the development of a nonunion [11, 15, 19]. Infection was found to increase the probability of an intervention for nonunion from 21 to 66% for a stainless steel plate and from 4 to 24% for a titanium plate [15]. Thus, it is imperative, when evaluating a nonunion, that infection is ruled out with the appropriate laboratory studies and radiographic imaging (see Chap. 1).

### 11.2.3 Patient Considerations

Patients with osteoporosis may have tenuous fixation and are at risk for hardware failure. Locked plating can certainly be helpful in such cases. Despite this, the geriatric population has high incidence of nonunion despite the use of locked plates. Moloney et al. [10] performed a multicenter retrospective cohort study of 176 patients. The mortality at one year was 25% with a 24% incidence of nonunion in the survivors. The long-term functional outcome of such injuries in the geriatric population has been very poor as well [25].

Non-compliance with weight bearing may put undue stress on plate fixation and can lead to early failure. Smoking can certainly delay fracture healing and may lead to a nonunion [22]. Diabetes and other endocrinopathies can also lead to a delay in healing or a nonunion [22].

Morbid obesity has also been shown to be a risk factor for the development of a nonunion specifically in distal femur fractures [15, 22]. Obesity was found to more than double the probability of an intervention for a nonunion despite the material of the implant [15]. Ricci et al. [22] found that proximal implant failure was associated with a higher BMI. The implant failure then leads to the development of a nonunion.

---

## 11.3 Evaluation and Diagnosis

The general evaluation and diagnosis of nonunions has been covered in Chap. 1. The same principles apply. However, specific points to address in relation to distal femoral nonunions will be discussed below.

### 11.3.1 History

A clear understanding of the original mechanism of injury can provide information to assist in evaluating the nonunion. It is important to understand the mechanism of injury of the original fracture. Was it a high- or low-energy injury? Was it an open fracture? If it was open, how many surgeries prior to definitive fixation? What was done at the time of the original surgery? This can be hard to ascertain if the patient has undergone several surgeries prior to their presentation. Requesting the medical records from the original surgeon can be enlightening. Did they have any problems after fixation? Obtaining an accurate history regarding any previous infection is paramount. It is important to determine when weight bearing began especially when there is hardware failure. Early failure may indicate non-compliance with the postoperative regimen. A social history should be obtained to include the use of nicotine, narcotics and illicit drugs. A careful medical history, to determine whether any comorbidities contributed to the development of the nonunion especially diabetes, is critical.

### 11.3.2 Physical Examination

The patient should be evaluated for gross motion at the nonunion site in cases of hardware failure. The limb should be inspected for signs of infection such as erythema or draining sinus tracts. Knee motion should be assessed as best possible. In cases where the hardware has failed, the patient may have too much discomfort or pain to assess accurate range of motion. A thorough neurovascular exam should be performed. Many of these patients may have concomitant ligamentous injuries of the knee, which may have gone unrecognized. Therefore, a careful knee exam to assess for stability should be performed if possible. Gross motion at the nonunion site may preclude an accurate assessment of knee stability. The patient should be evaluated for leg length discrepancy, as many of these patients will have developed shortening from the numerous previous surgeries [26].

### 11.3.3 Laboratories

This has been covered previously, but a full evaluation for infection (complete blood count [CBC], erythrocyte sedimentation rate [ESR] and C-reactive protein [CRP] ) and metabolic issues should be performed. Vitamin

D deficiency should be addressed. Endocrinopathies and other metabolic abnormalities may require evaluation by an endocrinologist. Diabetics should have better glucose control. Osteoporosis should be managed with appropriate medications.

### 11.3.4 Radiographs

Standard anteroposterior (AP) and lateral radiographs of the entire femur should be obtained. Standing bilateral AP and laterals from the hip to the ankle can help to assess for any associated deformity with the nonunion. This also allows evaluation of the mechanical axis of the limb and to rule out any other associated deformities in the tibia. Stress examination of the nonunion site can be obtained to determine whether any motion is present in the cases of stiff nonunions where clinical evaluation may be equivocal.

### 11.3.5 Computed Tomography/Magnetic Resonance Imaging

A computed tomography (CT) scan should be obtained to define the nonunion. If there is concern for malrotation, a CT scan of both hips and knees can be obtained to compare the injured side to the unaffected side for a more accurate determination. A magnetic resonance image (MRI) is warranted in select cases where the hardware has already been removed (no metal artifact) and in infected cases to better assess the presence and extent of osteomyelitis. In general, an MRI is not needed for the aseptic nonunion.

### 11.3.6 Nuclear Imaging

These studies can be useful in evaluating nonunions when there is a concern for infection. If laboratory studies (CBC, ESR and CRP) are elevated, then nuclear medicine studies may add additional information. In the case of aseptic nonunions, these studies are usually not indicated.

---

## 11.4 Treatment

In a systematic review of the literature regarding distal femoral nonunions, the most common treatment involved fixed angle plating with cancellous autografting resulting in a 97.4% union rate [11]. It is important to determine

whether there are any causative factors which may have contributed to the nonunion. Correctable factors should be addressed such as smoking cessation and vitamin D replacement (which should correct secondary hyperparathyroidism). Treatment is based on a number of factors. The type of nonunion, whether it is hypertrophic or atrophic, will determine whether bone grafting is needed. The presence of intact or failed hardware can influence the treatment of choice. For fractures that were intra-articular, a determination of whether or not the intra-articular portion has healed can determine whether revision ORIF is needed or whether the nonunion is isolated to the meta-diaphyseal region. It is clear from the literature that no clear consensus exists as to the best treatment option for these nonunions [11, 12].

Chapman et al. [27] used either single or double plate fixation with autologous bone grafting in the management of distal femoral nonunions in 18 patients. In their retrospective review, they had 100% union rate. In another study by Bellabarba et al. [28], twenty patients with nonunions were managed with indirect reduction techniques and application of either a 95° condylar blade plate, condylar buttress plate or a locking condylar plate. Only 45% (atrophic and oligotrophic nonunions) underwent adjunctive autologous bone grafting. They reported a 100% union rate. All of these patients had been initially treated with similar plate screw constructs, but none had bone grafting as part of the original fracture treatment. The same authors had used similar techniques in a series of twenty-three patients with femoral nonunions that had been initially treated with intramedullary nailing for their femur fracture [29]. There were only eight distal femoral nonunions. These were all treated with a 95° condylar blade plate and all healed. The overall success rate for all fractures was 91%. Bone grafting was performed on all biologically deficient nonunions. Gardner et al. [12] reviewed a single surgeon case series of 31 distal femoral nonunions treated with a fixed angled implant. Lag screws across the nonunion site were used in all patients as well as bone graft augmentation (71% autologous bone). They had a 97% union rate at 15.9 weeks with return to a pre-injury functional status in 84% of patients. Deformity correction was an important part of the treatment. Wang and Weng [30] treated thirteen patients with distal femoral nonunions with open reduction and internal fixation combined with both cortical allograft struts and autogenous iliac crest bone grafts. They used predominately blade plates or condylar buttress plates and a few antegrade nails . They achieved 100% union at an average of 5 months. Amorosa et al. [31] used 95° angled

blade plates to treat 32 cases of distal femoral nonunions. They had a 92.5% healing rate with the one surgery in the 27 patients with follow-up.

An alternative approach to complete revision ORIF has recently been described by Holzman et al. [32], where a medial locking plate is added to a preexisting intact lateral locking plate construct. They treated 22 patients with 23 distal femoral nonunions with either the addition of a medial plate and autogenous bone grafting when the lateral plate was stable (16 cases) or a two-stage procedure where the broken lateral plate was removed and replaced, followed by a medial locking plate and bone graft two months after the first stage (7 cases). They had a 95.2% success rate in the 21 cases with follow-up. They concluded that adding a medial plate in cases with stable lateral fixation was a successful alternative to complete revision surgery.

The use of intramedullary nailing in the management of distal femoral nonunion has also been studied. However, the early supracondylar nails initially developed were fraught with complications due to the multiple hole configurations of these implants. Koval looked at a series of 16 distal femoral nonunions treated with the supracondylar nail and had only a 25% success rate with a high rate of hardware failure and complications [33]. Wu treated 21 distal femoral nonunions with antegrade nails placed in a retrograde fashion and dynamically locked [26]. In the 18 patients followed for an average of 3.3 years, 88.9% healed at an average of 4.2 months. All were bone grafted with autogenous bone graft obtained from the ipsilateral medial tibial condyle at the time of the nailing. In a similar series, Wu also treated 13 distal femoral nonunions where the initial fracture was treated with an antegrade nail [3]. They again utilized an antegrade nail placed in retrograde fashion, locked dynamically with bone grafting from the medial tibial condyle. Plate fixation was added in some cases. They had a 100% union rate at an average of 4.5 months.

Since many of these nonunions have associated leg length discrepancy and deformity, external fixation has been described as an option for the management of distal femoral nonunions. Ali and Saleh [34] treated 15 cases of distal femoral nonunion in which all had either a leg length discrepancy or malalignment requiring correction. Five of the cases were infected. They had success in 14 of 15 (93.3%) cases with the one case uniting after intramedullary nailing. They were able to correct angular deformities as well as regain length in these patients. The biggest issue was poor motion with an average range of motion of 80$^{o}$.

As a salvage procedure in patients with a persistent nonunion of the distal femur, especially in the elderly, prosthetic replacement has been described [35–37]. Haidukewych et al. [35] performed a total knee arthroplasty (TKA) in 17 patients (ages 38–86; mean of 66) that had either failed treatment of a distal femur fracture or nonunion. They had a five-year survivorship of 91%. They did have a 29% rate of both intra-operative and postoperative complications. They felt that it provided reliable pain relief as well as functional improvement, but the overall results were inferior to that of primary TKA. In cases of the elderly patient with a persistent nonunion, the use of a megaprosthesis has also been reported [36, 37]. These patients are cited as having poor bone quality, arthritis, joint contractures and previous implant failure. Revision is felt to be a poor option in these elderly patients. Vaishya et al. [37] treated ten patients with a persistent nonunion and arthritis with a megaprosthesis. All knees had satisfactory alignment and range of motion, but two patients had minor wound problems. They felt that this was a viable one-stage salvage procedure for the patient with a difficult nonunion. The advantage for prosthetic replacement is that is allows for early ambulation [36].

Many options exist for the treatment of distal femoral nonunions, and there is no clear algorithm for the best treatment in terms of implant. Revision plating, intramedullary nailing and even circular external fixation are all viable options but need to be based on the stability of the pre- existing fixation as well as the local biology. Prosthetic replacement should be considered in the elderly with poor bone quality and arthritis.

## 11.4.1 Treatment Based on Nonunion Type

### *11.4.1.1 Hypertrophic*

Hypertrophic nonunions need stability and thus improvement of the mechanical environment is paramount. These do not usually require bone grafting. In the majority of cases, the hardware has failed and revision of the fixation is required. Either plate fixation or retrograde intramedullary nailing has been successful. If a retrograde intramedullary nail is utilized, the intra-articular component must be healed. The nail can address the meta-diaphyseal component only. If the intra-articular nonunion is simple, lag screw compression with bone graft may be needed. The mechanical axis needs to be realigned regardless of the implant used. The hypertrophic

nonunion is usually mobile enough to allow for deformity correction. If a nail is used, blocking screws can aid in deformity correction. If plates are used, fixed angle devices can help correct the deformity. Small leg length discrepancies can be tolerated and managed with a shoe lift. Healing of the hypertrophic nonunion is the goal.

#### *11.4.1.2 Atrophic or Oligotrophic*

The decision for bone grafting is clear and should be performed in cases of atrophic or oligotrophic nonunions. If the hardware is stable, autogenous bone grafting can be performed without a need for hardware revision. If, however, the hardware has failed, then both revision fixation and bone grafting are required for a successful outcome. As in all cases, the mechanical axis should be re-established. If the joint component is healed, then retrograde nailing with use of the 'reamer–irrigator–aspirator' (Synthes, Paoli PA, USA) for harvesting of autogenous bone graft from the femoral canal can be performed. Fixation with a retrograde nail that has fixed angle capabilities in the distal segment should be utilized. The reamings obtained can be packed into the nonunion site.

#### *11.4.1.3 Infected*

In cases of infected nonunions, a two-stage if not three-stage procedure may be warranted. In the first stage, removal of the hardware, debridement of the infected nonunion site, obtaining cultures, application of antibiotic cement into the defect with or without temporary external fixation are performed. Once the infection is cleared, stabilization along with placement of a cement spacer is performed. Fixation can be with either a retrograde intramedullary nail (preferred) or a locking plate or a fixed angle device. In the final stage, bone grafting into the defect is done after the cement spacer is removed (Masquelet technique). If the amount of bone requiring debridement is extensive, the use of circular external fixation and distraction osteogenesis to fill the defect can be considered. This technique is highly specialized and should be undertaken by someone experienced.

### 11.4.2 Author's Preferred Methods of Treatment

1. *Stable Hardware (Rare) and Hypertrophic*: Adjunctive plate fixation can often provide sufficient stability to promote union. This situation is rare.

2. *Stable Hardware and Atrophic/Oligotrophic*: If the hardware is stable, simply bone grafting the nonunion site should be sufficient to promote union. The harvest site for the bone graft should be based upon the amount of bone graft needed.

3. *Failed Hardware (Common)*: In cases where the initial lateral locked plating has failed, the joint component has healed and the meta-diaphyseal area has gone on to a mobile hypertrophic nonunion, removal of hardware and retrograde intramedullary nailing with a nail allowing for a fixed angle distally works well. The largest diameter nail should be used to obtain stability. With the advent of the reamer–irrigator–aspirator (RIA) system (DePuy Synthes, Warsaw IN, USA), it is easy to obtain autogenous bone graft from the intramedullary canal at the time of reaming. The bone graft can be packed into the nonunion. This technique is our method of choice for most distal femoral nonunions regardless of the nonunion type. Nail stabilization allows for earlier weight bearing. Bone grafting provides a biological stimulant as many of these patients have already had several operations at the time of presentation. Correction of any deformity can usually be accomplished with the nail as most are mobile. If the nonunion site is stiff, a fixed angle device (95° angled blade plate or DCS) to correct the deformity may be a better option as long as the joint injury is healed.

4. *Failed Hardware with Nonunion of Intra-Articular Component*: If the hardware has failed and the joint component is not healed, then complete removal of the previous plate and revision ORIF with restoration of the joint congruity, realignment of the mechanical axis and bone grafting is needed. We prefer to use a locking plate, either the locking compression LISS plate or locking condylar plates after compression and fixation of the joint component.

5. *Use of External Fixation* : Circular external fixation for nonunion management is reserved for those cases where, despite bone grafting to large defects, the nonunion persists. It can also be useful in cases of septic nonunion where internal fixation may be problematic despite debridement. It can also be used in cases of multiplanar deformities in combination with a nonunion, especially when it is a stiff hypertrophic nonunion. The patient must understand the procedure and the length of time such a device will be on as it can be life altering during the time the fixator is on the thigh.

6. *Prosthetic Replacement*: In cases of the elderly patient with a distal femoral nonunion, consideration to a total knee arthroplasty should be given. The ideal candidate should be one with poor bone quality where fixation may be problematic with ORIF. If they also have preexisting arthritis or as a result of the original injury, then a total knee arthroplasty may be preferred. A megaprosthesis (distal femoral replacing) can be considered when the bone stock is deficient and unable to support a standard or stemmed total knee arthroplasty.

## 11.4.3 Case Discussions

**Case 1**

Patient is a 50-year-old white male originally involved in motor vehicle accident (MVA) in 2008. Patient sustained a right Grade III A open distal femur fracture AO Type C3. He underwent irrigation and debridement of the open fracture (I&D) and temporary bridging external fixation. He then required several washouts due to the contamination. He subsequently underwent definitive ORIF approximately 2 weeks after the initial injury with a 7-hole LCP-LISS plate. Patient was followed by the original surgeon and then referred for a nonunion, with hardware failure at 5 months with AP and lateral radiographs shown in Fig. 11.1.

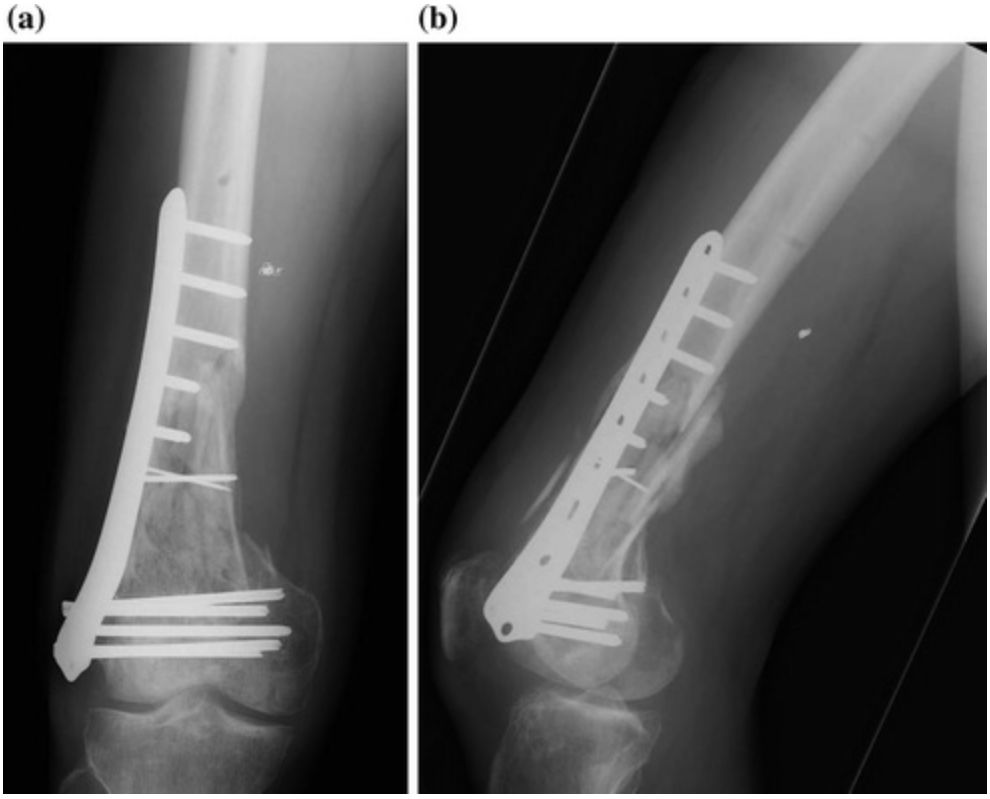



***Fig. 11.1*** **a** Anteroposterior and **b** lateral radiographs of the right knee showing hardware failure, shortening and varus

The patient was evaluated and found only to have hepatitis C. The patient denied any history of wound problems or infections after the definitive procedure. The patient had not smoked for 30 years and quit drinking 10 years prior to presentation. Laboratory markers were all within normal limits for his white blood cell (WBC) count , C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) . Dual-energy X-ray absorptiometry (DEXA) scan had been obtained by his primary care provider and was normal. He had normal 25-OH vitamin D levels. His physical examination showed well-healed surgical scars as well as traumatic lacerations from the original injury, varus malalignment of the limb at the nonunion site and flexion only to 30°. A CT scan with coronal and sagittal reconstructions (Fig. 11.2) was obtained, which showed healing of the intra-articular component but a clear nonunion of the metaphyseal portion with varus collapse with pullout of screws as well as broken screws.

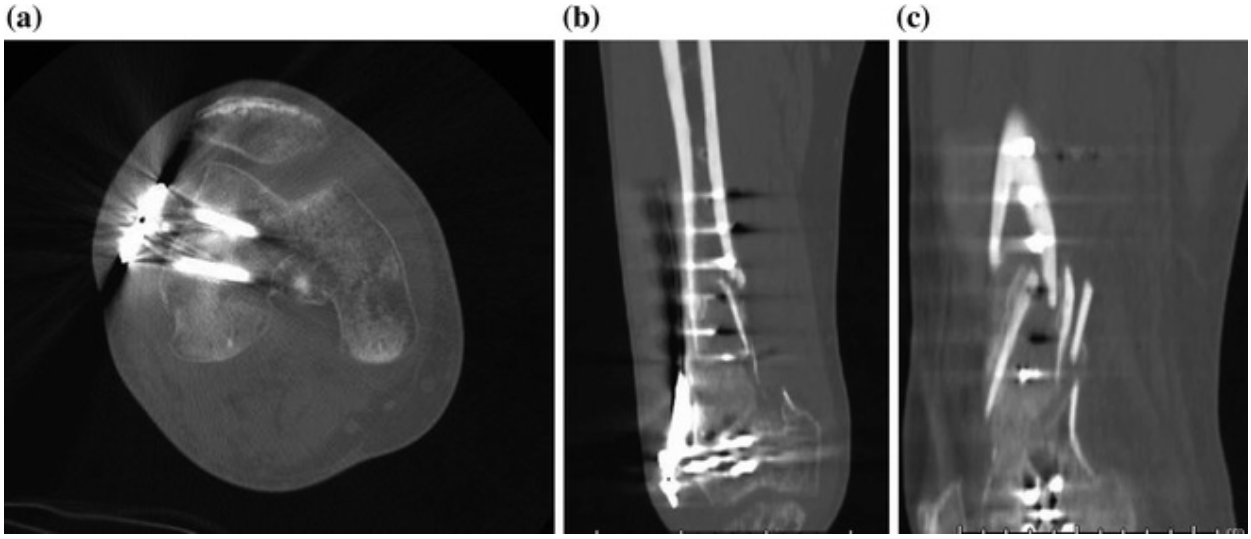



***Fig. 11.2*** **a** Axial computed tomography (CT) image showing healing of the intra-articular component; the gap between the plate and the bone is well visualized; **b** coronal CT image showing the varus alignment, failure of hardware and the metaphyseal nonunion; **c** sagittal image also showing the nonunion

The patient underwent hardware removal with debridement of all fibrous tissue from the nonunion site. The RIA system was utilized in a retrograde fashion to obtain bone graft from the femoral canal of the affected leg. A retrograde nail with a fixed angle blade component distally was inserted and statically locked proximally with two screws. The RIA bone graft was packed into the nonunion. The postoperative images are shown in Fig. 11.3.

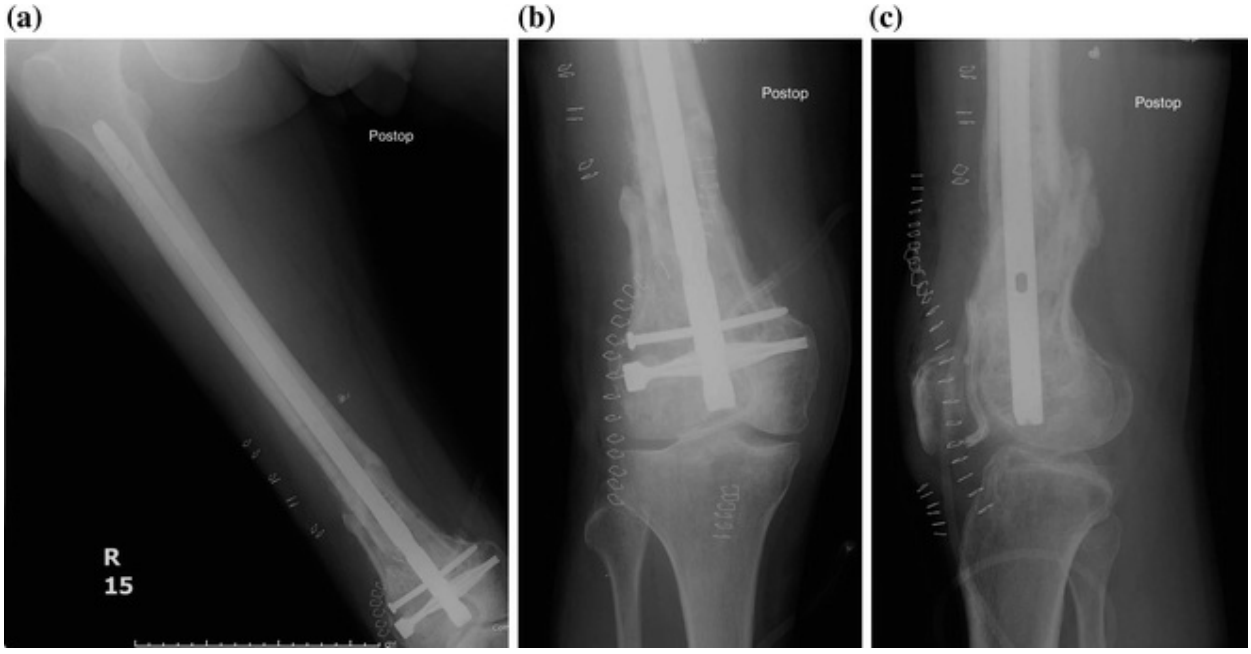


***Fig. 11.3*** Immediate postoperative images after retrograde intramedullar nailing and bone grafting. **a** Full length right femur showing re-establishment of femoral anatomical axis; **b**, **c** anteroposterior and lateral of the right knee showing the nonunion site with bone graft

The patient went onto heal the nonunion by 7 months (Fig. 11.4). At this point, he underwent manipulation under anesthesia of his right knee, quadricepsplasty and an arthrotomy with lysis of adhesions for persistent poor knee motion (0° to 65°). The patient eventually achieved 110° of motion.

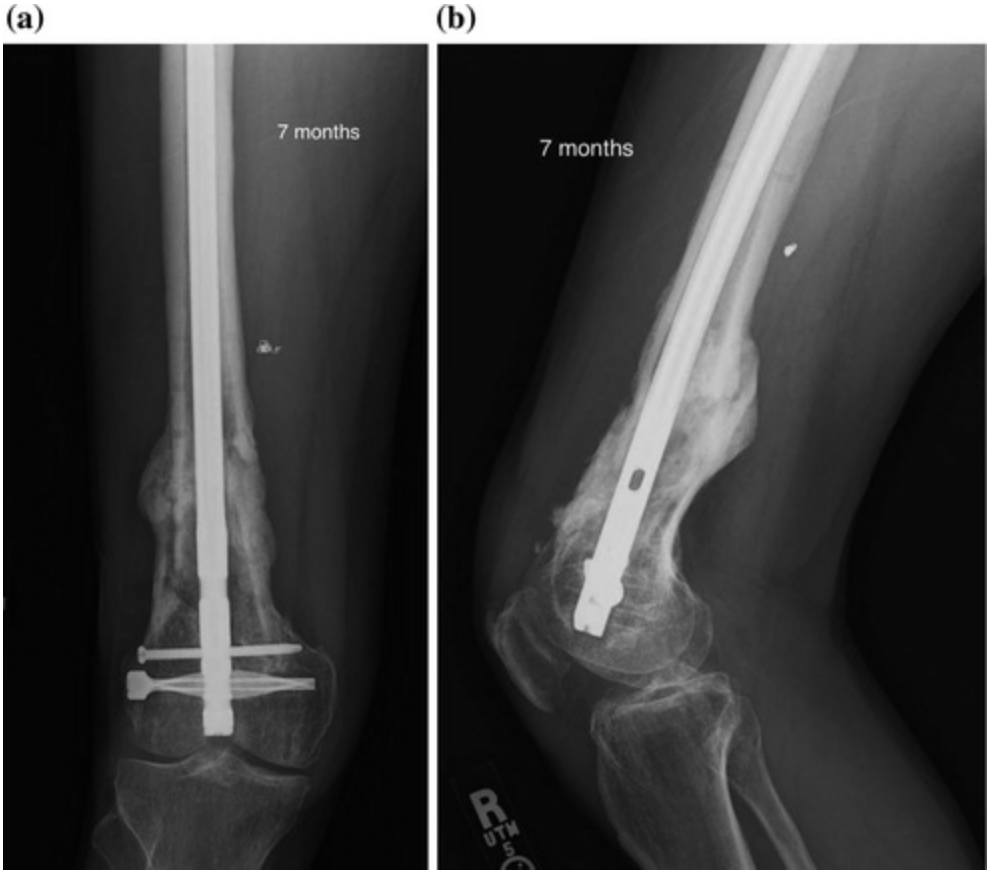



***Fig. 11.4*** **a** Anteroposterior and **b** lateral radiographs of the right knee at 7 months showing consolidation of the nonunion site

The patient did well and returned to his activities, which included downhill skiing. Patient returned 7 years later with complaints of knee pain, which was felt to be consistent with arthritic-like symptoms and probably a degenerative medial meniscal tear (Fig. 11.5). He was also having hardware symptoms distally at the lateral aspect of the knee. Arthroscopic debridement along with hardware removal was discussed with the patient since the patient

was going under anesthesia. The patient had arthroscopic debridement of the knee. He was found to have Grade III medial tibial compartment disease but only Grade I lateral compartment disease. The nail was removed without difficulty (Fig. 11.6). Patient returned to his snow skiing and has improved motion to 120° of flexion and has always maintained his extension.

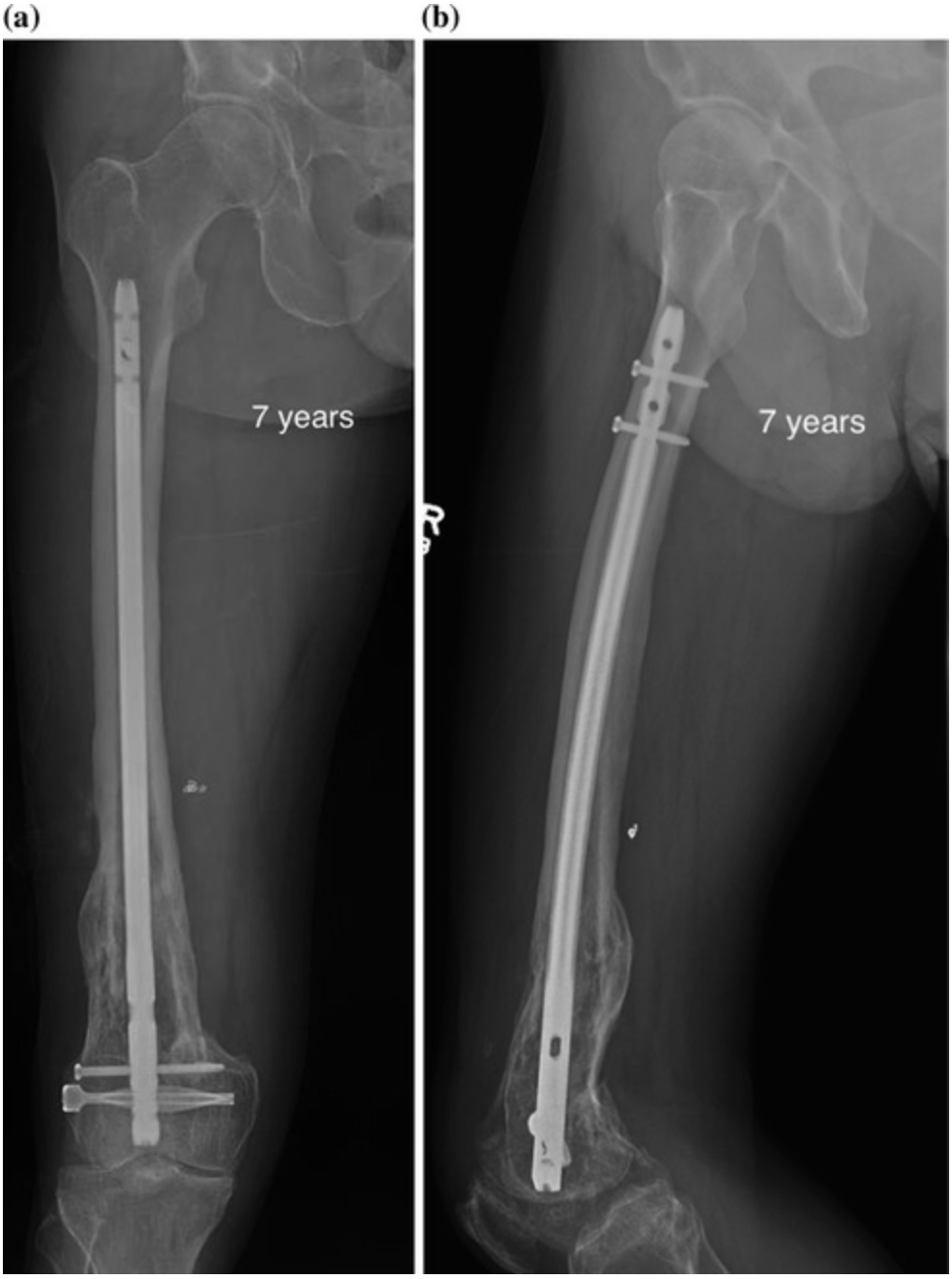



***Fig. 11.5*** **a** Anteroposterior and **b** lateral radiographs of the right femur at 7 years showing well-healed femur with stable hardware

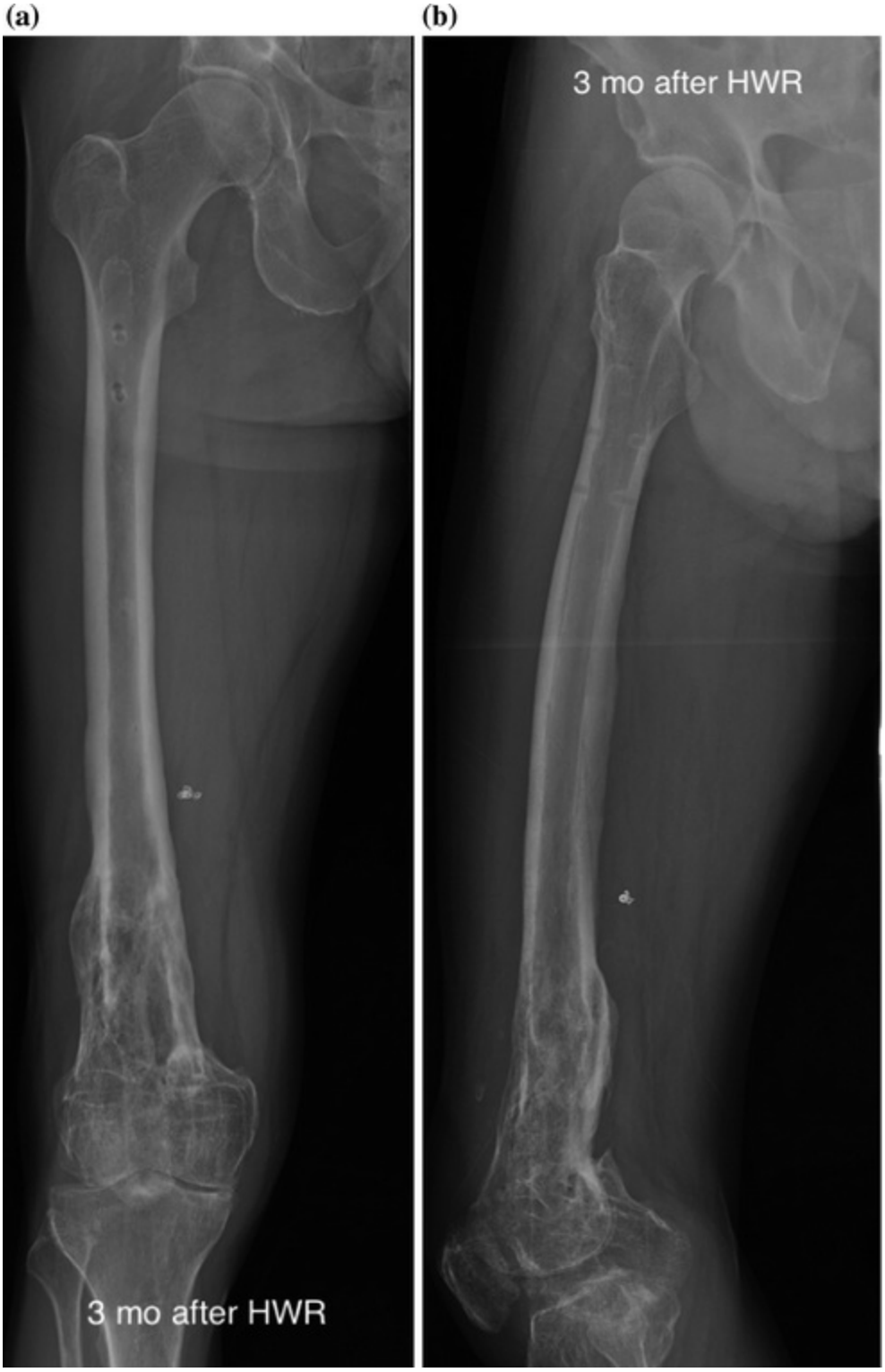



***Fig. 11.6*** **a** Anteroposterior and **b** lateral radiographs of the right femur at 3 months after hardware removal

## Case 2

The patient is a 54-year-old Latin American female who sustained multiple injuries in an MVA in 2006. The patient was treated for a left distal femur fracture with ORIF at an outside institution. The patient was followed for approximately 17 months, after which she was told she was healed and discharged. She apparently was fully weight bearing.

She then presented 2 years out from the initial injury with hardware failure and a nonunion of the left distal femur (Fig. 11.7). The patient was unable to give details of the injury as to whether or not it was an open fracture. The patient is morbidly obese. She has diabetes, hypertension and a history of deep vein thrombosis. Her laboratory evaluation showed a normal WBC but an elevated ESR of 74 and CRP of 21.3. Her other laboratory studies were within normal limits. The nuclear medicine studies obtained were negative. Clinically, she did not have any evidence of infection nor did she report ever having any wound problems or any other issues after the index procedure until 22 months later when she noticed the sudden pain. A CT scan was obtained and confirmed the nonunion and hardware failure. The joint was healed (Fig. 11.8).

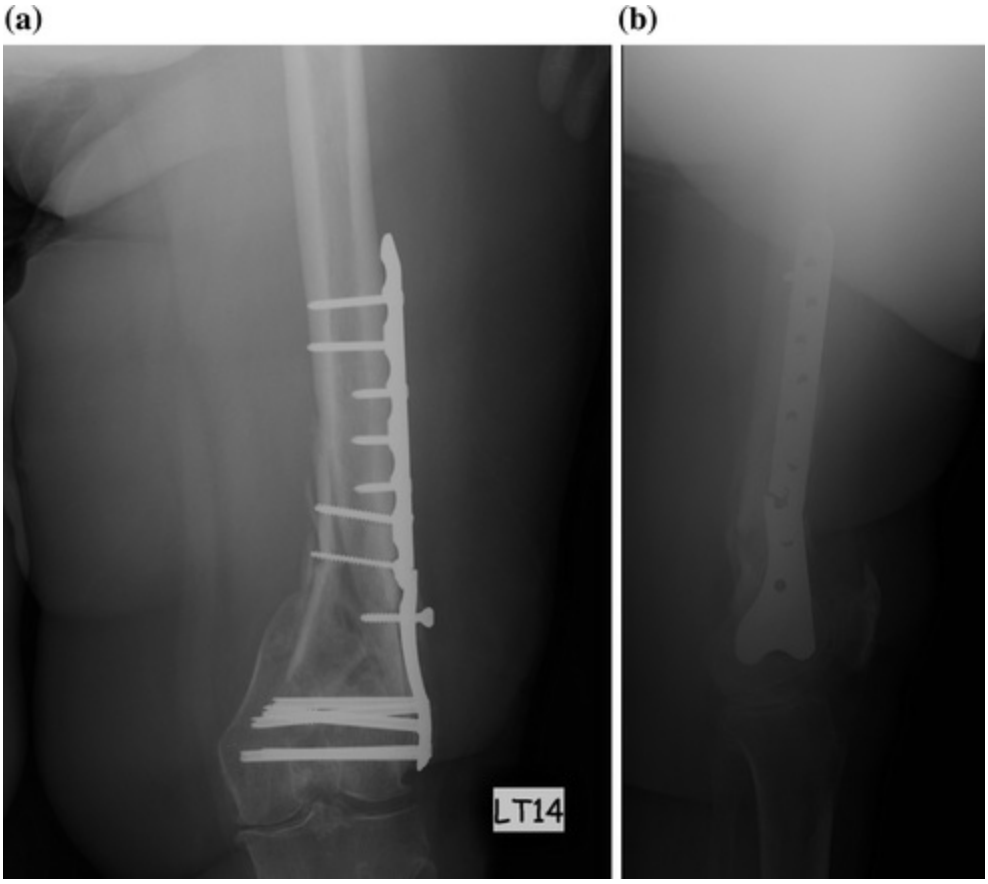


***Fig. 11.7*** **a** Anteroposterior and **b** lateral radiographs of the left knee 2 years after the initial fixation showing loosening of hardware and nonunion**. a** The loose screw is easily visualized**; b** the break in the plate is well visualized as well as the recurvatum deformity at the nonunion site

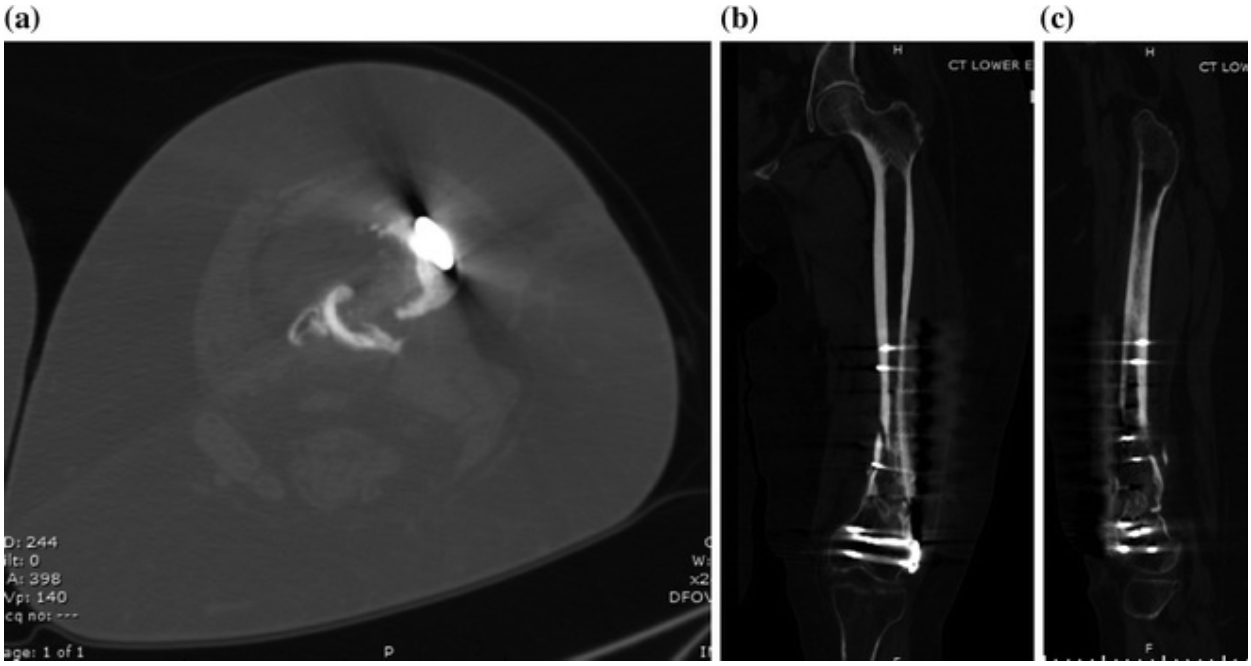



***Fig. 11.8*** Computed tomography scan images showing the nonunion: **a** axial image showing lack of bone, **b** coronal image showing the varus and nonunion, **c** sagittal image showing the recurvatum deformity and nonunion

The patient underwent removal of the hardware, RIA of the femur for bone graft and placement of a retrograde nail with a fixed angle blade component distally. It was statically locked proximally with two screws. Her postoperative images are shown in Fig. 11.9.

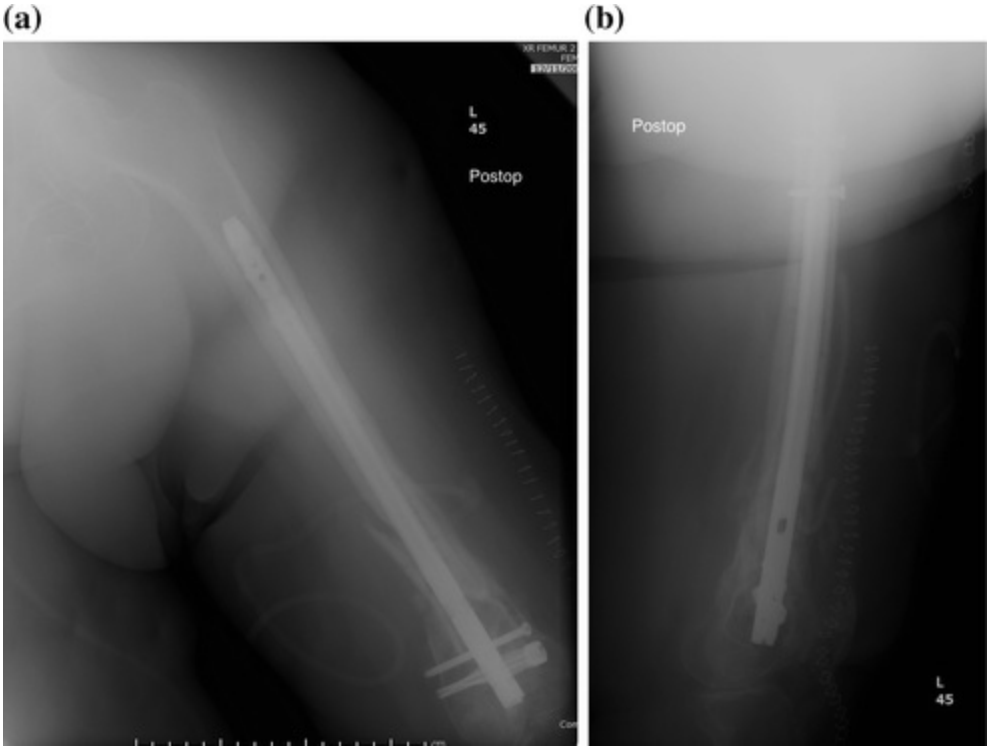



***Fig. 11.9*** Immediate **a** anteroposterior and **b** lateral postoperative left femur radiographs showing stabilization of the nonunion with a retrograde nail and bone grafting

The patient was allowed to be immediately weight bearing and went on to heal by 6 months. (Figure 11.10). At her last follow-up of 13 months, she was ambulating fully with the use of a cane for long distances. She was pain-free with 0° to 95° of motion (Fig. 11.11).

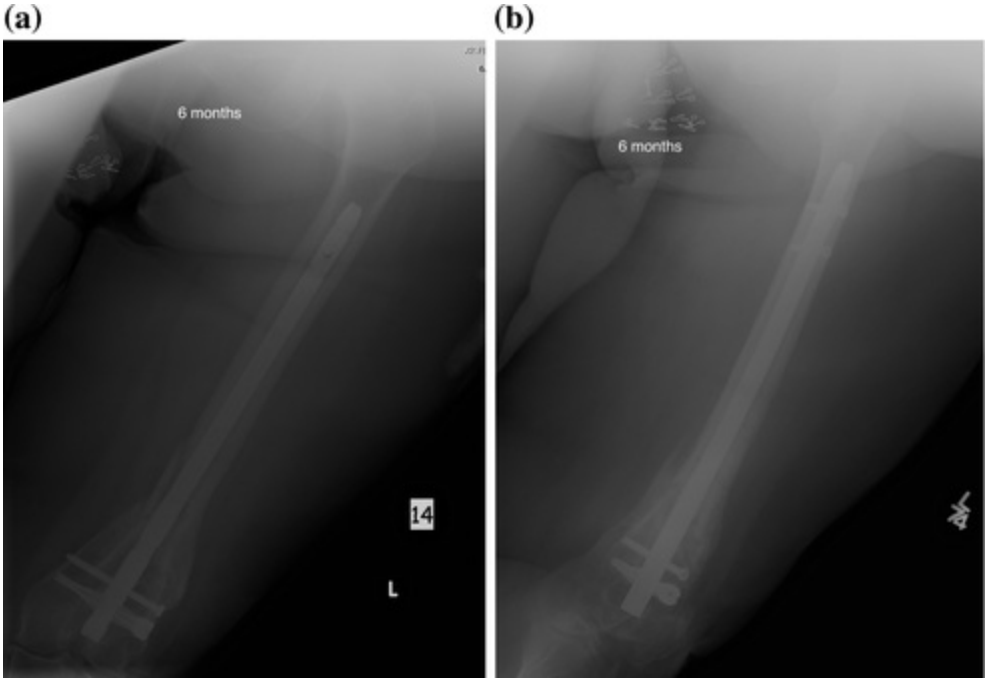



***Fig. 11.10*** **a** Anteroposterior and **b** lateral left femur radiographs at 6 months showing consolidation across the nonunion site

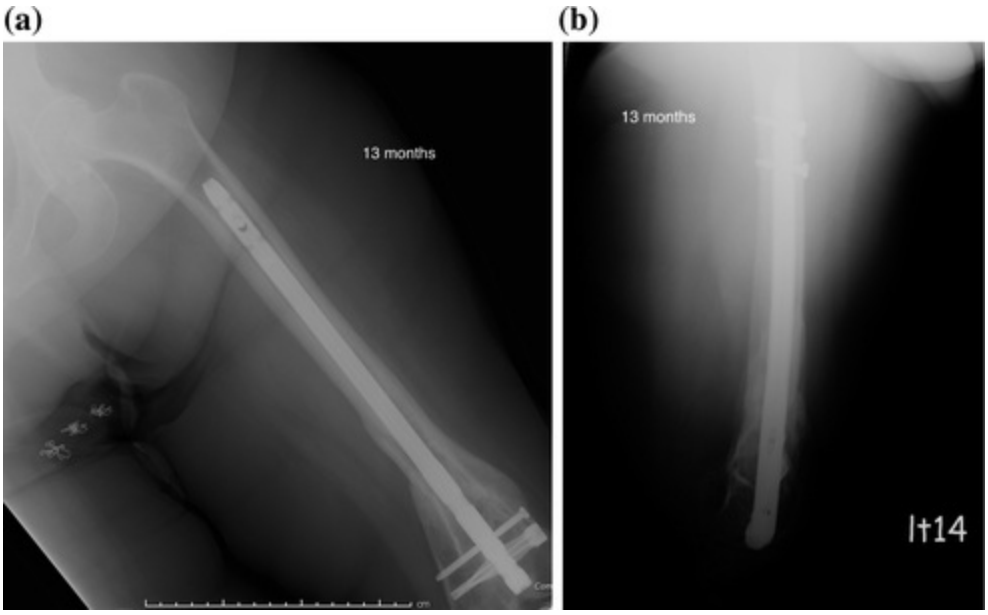



***Fig. 11.11*** Final follow-up **a** anteroposterior and **b** lateral left femur radiographs at 13 months showing a well-healed femur without hardware complications

## Case 3

The patient is a 38-year-old white male who was initially injured in an MVC while working out of town. He had sustained a left Grade IIIA open distal

femur fracture/dislocation. His operative report indicated that both the anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) were out, but no indication regarding the status of his collaterals. He had an initial irrigation and debridement with application of a temporary bridging external fixator. He subsequently underwent ORIF at the outside institution. Patient returned to the area and presented to our institution approximately 6 weeks out (Fig. 11.12).

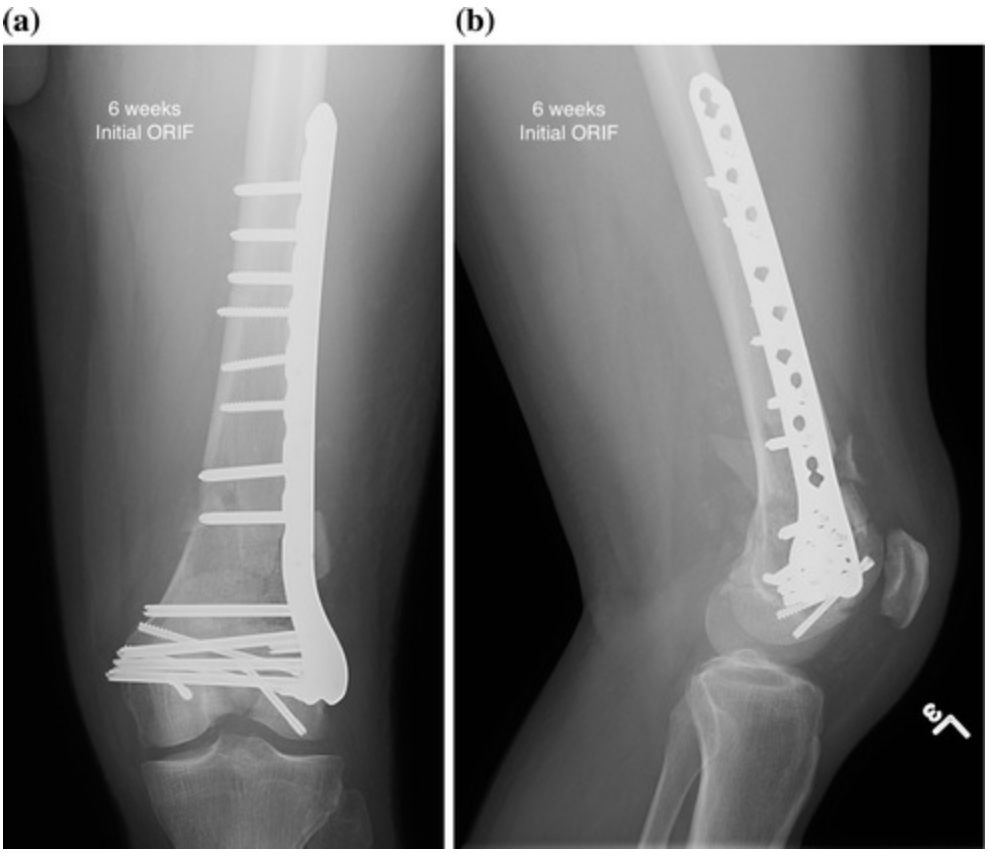



***Fig. 11.12*** **a** Anteroposterior and **b** lateral left knee radiographs at 6 weeks after open reduction internal fixation. The fracture appears well reduced and restoration of the anatomical axis

The patient is otherwise healthy. His physical examination at that time showed well-healed incisions and traumatic lacerations. He was followed and felt to be progressively healing (Fig. 11.13; 6 months). He was fully weight bearing, but at 9 months he developed increased pain. The radiographs showed subsidence of the hardware and some collapse (Fig. 11.14). The patient underwent a CT scan (Fig. 11.15), which showed a persistent nonunion of the metaphyseal area as well as part of the intra-articular region. The allograft bone had not been incorporated.

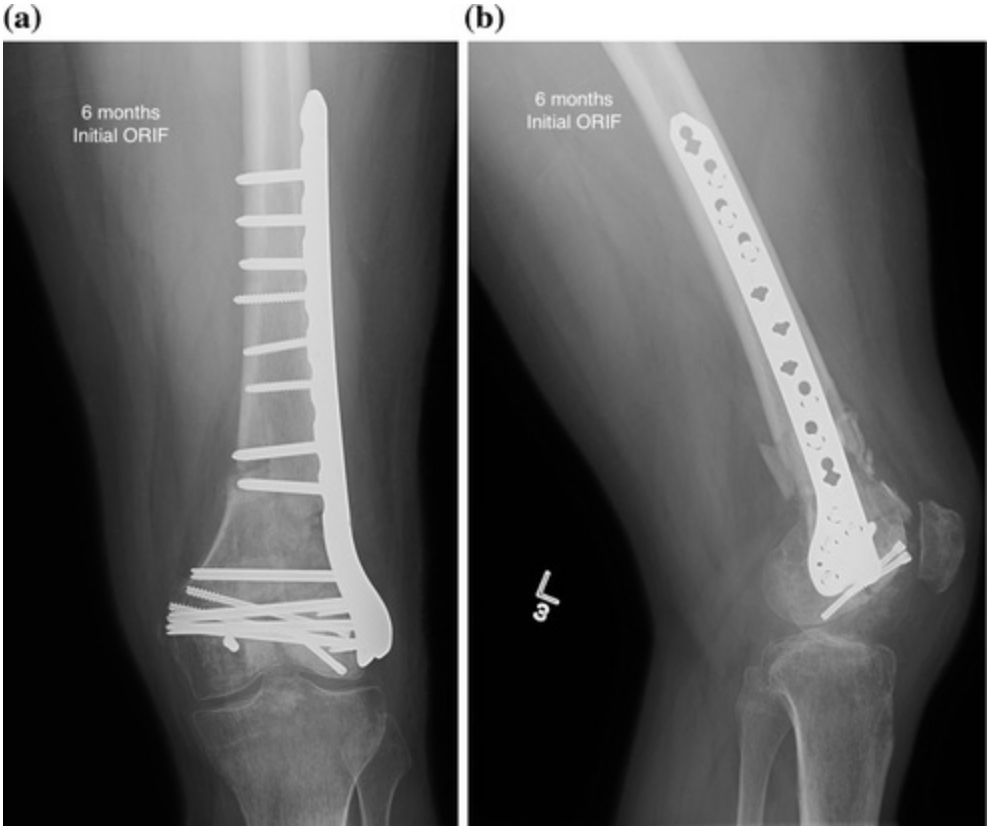



***Fig. 11.13*** **a** Anteroposterior and **b** lateral left knee radiographs at 6 months. **a** There appears to be some consolidation at the medial cortex as well as in the metaphyseal region, but some subsidence of the plate is seen with collapse at the fracture site but stable hardware; **b** lateral shows increasing consolidation anteriorly

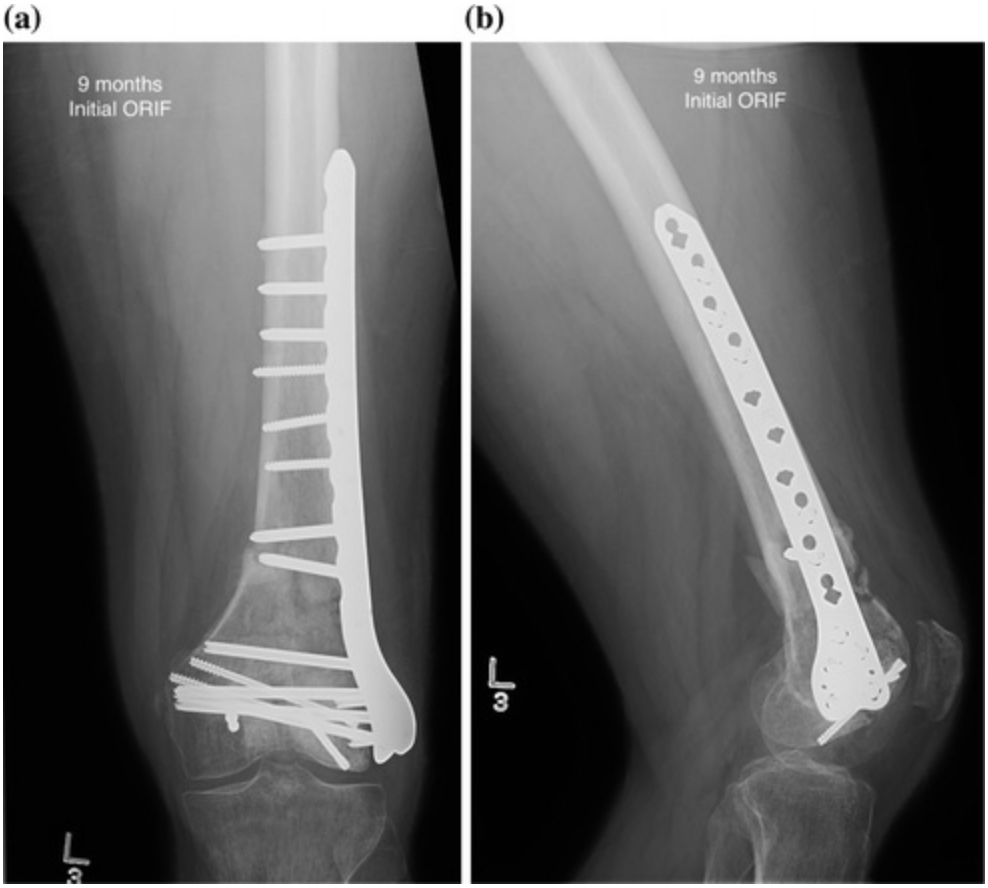



***Fig. 11.14*** **a** Anteroposterior and **b** lateral left knee radiographs at 9 months. **a** There appears to be further subsidence of the plate and thus collapse at the fracture site; **b** lateral shows increasing consolidation posteriorly and intact plate

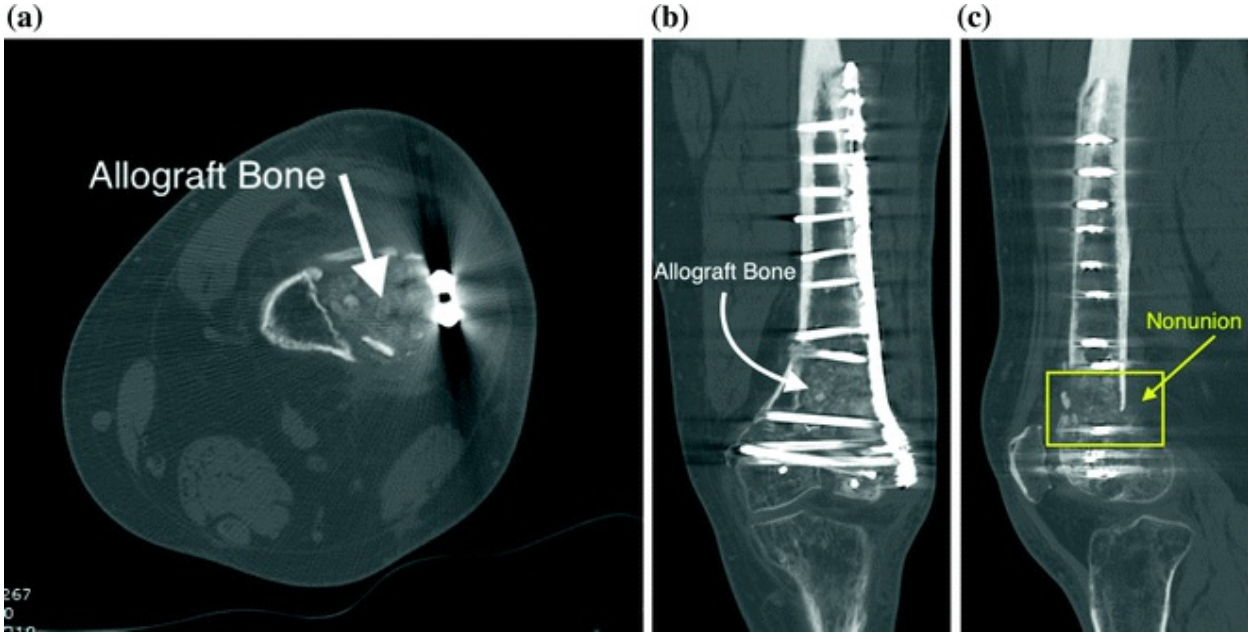


***Fig. 11.15*** A computed tomography scan was obtained to evaluate the fracture site. **a** Axial image showing the allograft bone still unincorporated and a lack of bridging; **b** coronal image showing again the allograft bone and its lack of incorporation as well as subtle varus collapse; **c** the obvious nonunion is clearly visualized on the sagittal image

The patient underwent revision ORIF as opposed to nailing because of concern for a persistent intra-articular nonunion. The hardware was removed, and the allograft bone was nonviable and had not incorporated; it was debrided, resulting in the large void shown in Fig. 11.16. The intra-articular nonunion was stabilized with a screw (Fig. 11.17). Bone graft was obtained via the RIA system from the left femur after the hardware was removed. It was done retrograde through the nonunion site (Fig. 11.18). Revision ORIF with a variable angle locked plate was performed and the bone graft placed into the nonunion site with additional bone graft extender (demineralized bone matrix [DBM]) (Fig. 11.19). The final postoperative radiographs are shown in Fig. 11.20.

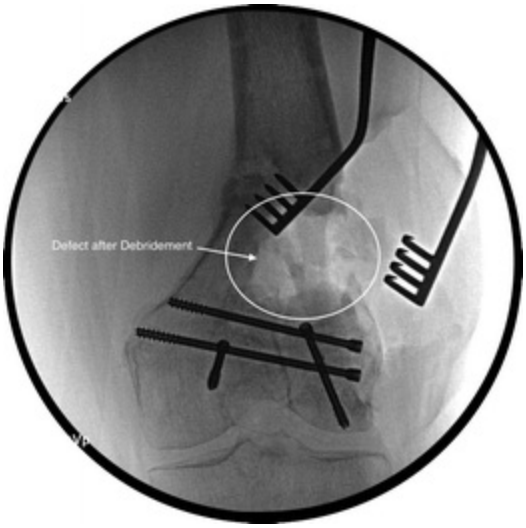



***Fig. 11.16*** Intra-operative fluoroscopic image after plate removal and debridement of the allograft showing the large void


***Fig. 11.17*** Intra-operative fluoroscopic image showing the additional partially threaded cancellous screw for lag screw fixation of the articular nonunion


***Fig. 11.18*** Intra-operative fluoroscopic image showing the reamer for the reamer–irrigator–aspirator (RIA) going retrograde through the mobile nonunion site


***Fig. 11.19*** Intra-operative fluoroscopic image after stabilization and bone grafting of the nonunion site

***Fig. 11.20*** Immediate postoperative **a** anteroposterior and **b** lateral left knee images showing the final construct

Patient went onto heal the nonunion with abundant bone around the site and was functioning well at 18 months. His range of motion was 0° to 115°. His last follow-up radiographs are shown in Fig. 11.21.


***Fig. 11.21*** Follow-up **a** anteroposterior and **b** lateral radiographs at 18 months after the revision open reduction internal fixation and bone grafting of the left knee, showing consolidation of the nonunion site

## Case 4

The patient is a 51-year-old morbidly obese woman who is referred for a nonunion of her right distal femur. She is approximately one year out from her initial injury, which was a right grade III A open distal femur fracture. She was managed with ORIF at an outside institution. The radiographs

showed bending of the plate and loosening of the screws distally. There was an obvious nonunion of the meta-diaphyseal region (Fig. 11.22).

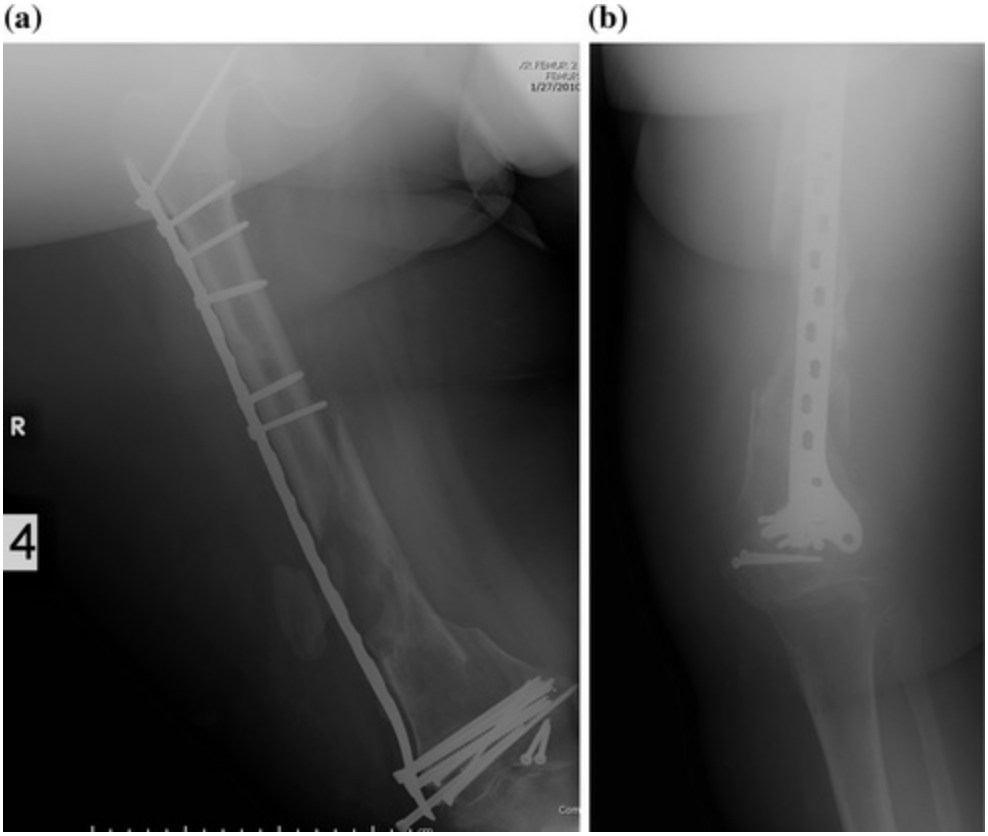



***Fig. 11.22*** **a** Anteroposterior and **b** lateral radiographs of the right femur showing the bending of the original fixation and loss of fixation distally. The large soft tissue density can also be appreciated

She reports no immediate complications after her initial surgery and denies any history of infection. Her only medical problem is morbid obesity (BMI 64). She is a smoker (half pack per day). Smoking cessation was recommended. She had been previously prescribed an ultrasound unit in an attempt to aid consolidation. Physical examination showed well-healed surgical scars and lacerations without signs of infection. Her range of motion was 0° to 100° compared to 0° to 120° on her unaffected side. Her laboratory evaluation showed her CRP to be 19.5, WBC 11.9 and ESR of 22. Her 25-OH vitamin D was less than 15. She was immediately started on vitamin D2 at 50,000 units weekly. She responded to the dosing with her 25-OH vitamin D increasing to 39. Her nuclear medicine studies showed uptake consistent with degenerative changes in the knee joint but no evidence of infection.

The patient underwent repair of her nonunion with removal of all

hardware with obvious motion seen at the nonunion site. The nonunion was stabilized with a retrograde nail after obtaining bone graft using the RIA system. We obtained 40 cc of bone graft, which was all placed into the nonunion site and supplemented by demineralized bone matrix. The immediate postoperative images are shown in Fig. 11.23.

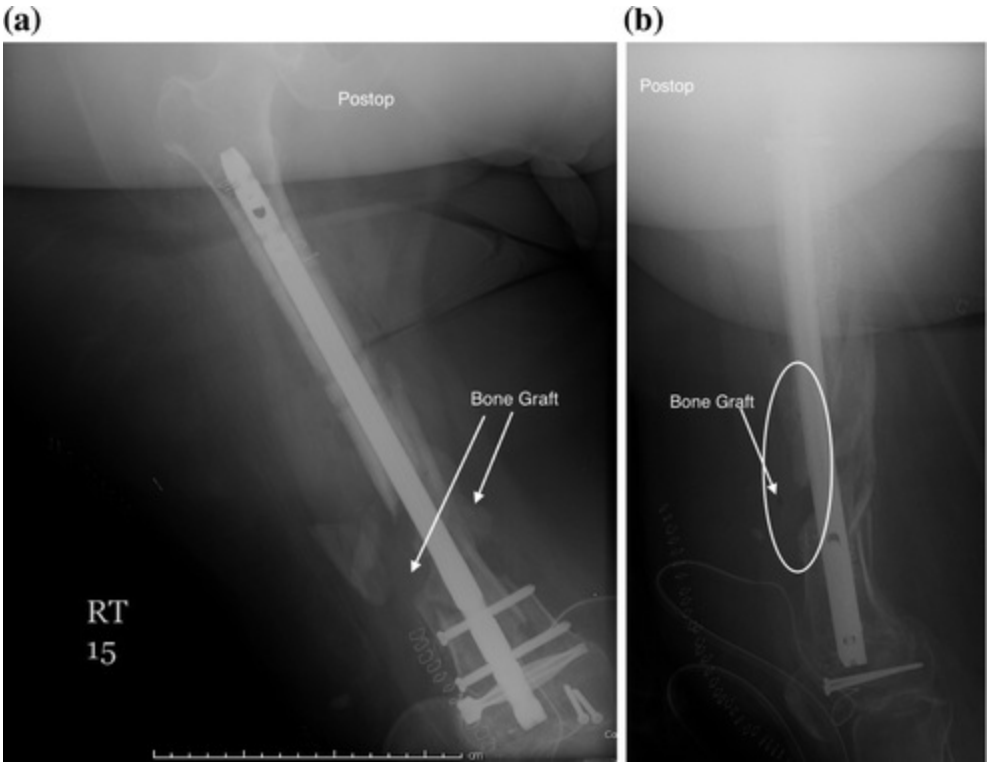



***Fig. 11.23*** Immediate postoperative **a** anteroposterior and **b** lateral radiographs of the right femur showing stabilization of the nonunion site with a retrograde nail and placement of the bone graft

The patient was followed and felt to have healed by 6 months with bridging bone (Fig. 11.24). At her last follow-up at four years, she was ambulating without assistive devices, had only a 5 mm leg length discrepancy managed with a shoe insert, and had regained her full knee range of motion (ROM) (Fig. 11.25).

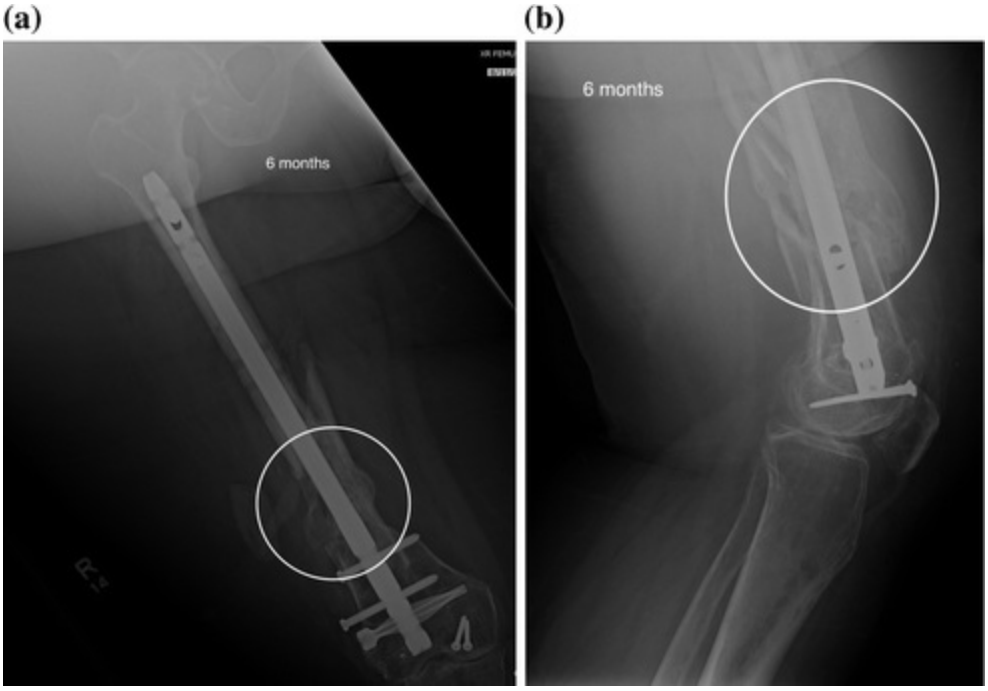



***Fig. 11.24*** **a** Anteroposterior and **b** lateral radiographs of the right femur at 6 months showing increased consolidation and bridging of the nonunion. Abundant bone formation is visualized within the *marked areas*

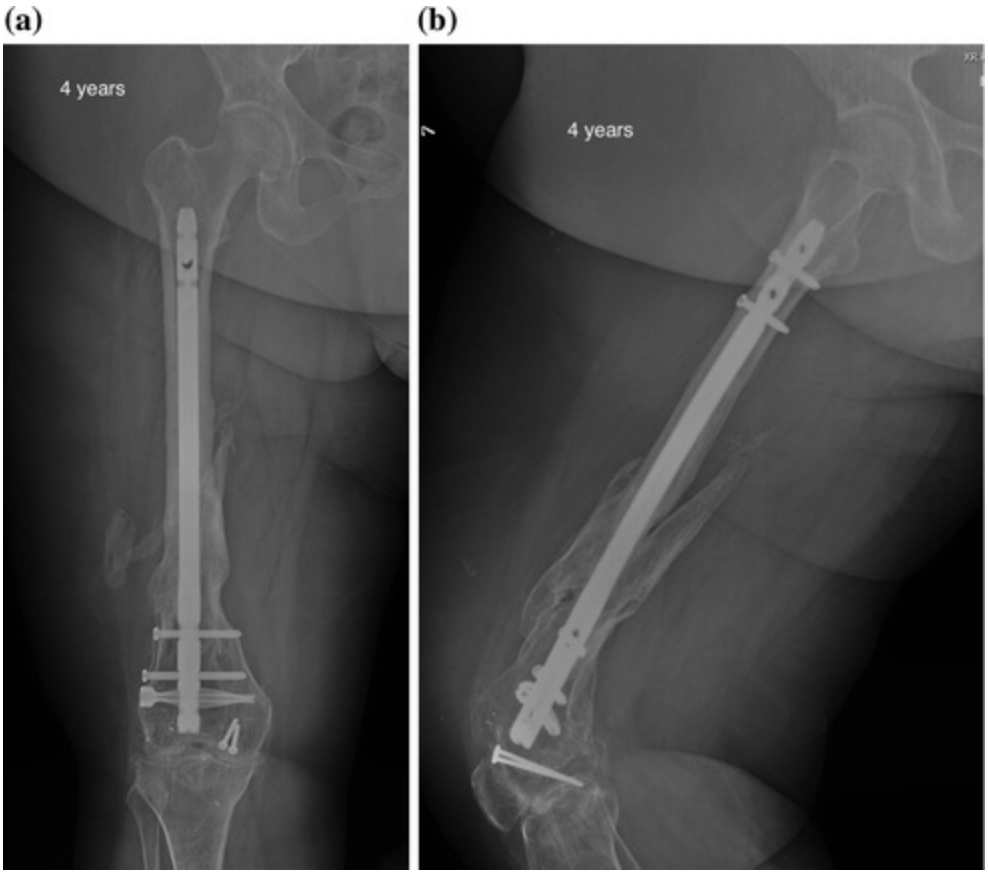



***Fig. 11.25*** Last follow-up **a** anteroposterior and **b** lateral radiographs of the right femur at 4 years showing resolution of the nonunion and stable hardware. There has been further consolidation across the nonunion site

## Case 5

The patient is a 48-year-old Latin American female who presents with pain, discomfort and inability to bear weight on her right lower extremity. Radiographs obtained at the time show a right distal femoral nonunion with hardware failure (Fig. 11.26).

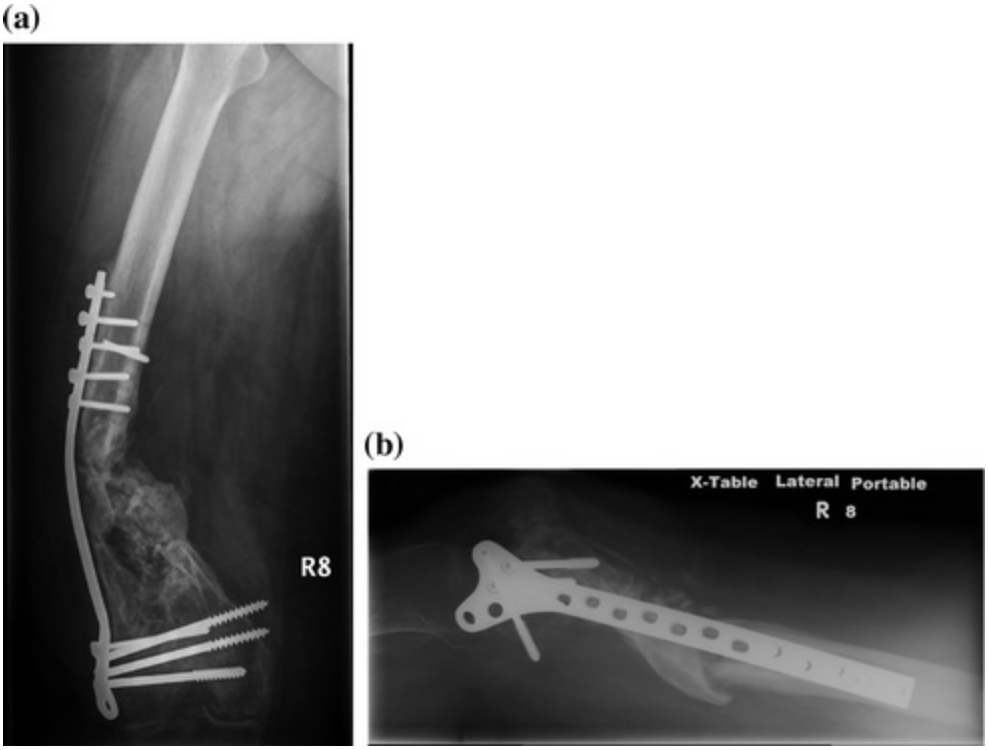



***Fig. 11.26*** **a** Anteroposterior and **b** lateral radiographs of the right femur showing failed hardware with significant varus deformity, hardware failure and nonunion

Her original injury was 4 years prior at which time she was treated at an outside facility for a right grade IIIA open distal femur fracture. She reports having multiple surgeries (10) afterward for various reasons, including infection. She is morbidly obese and has hypothyroidism (on thyroid replacement). She does not smoke. Her physical examination showed well-healed surgical incisions and lacerations. Her range of motion was 5° to 35°, and it appeared that she had about 30° to 40° of malrotation. She had no signs of infection.

Nonunion evaluation was performed. Her WBC was 9.6, ESR 29 and a CRP 19.9. She also had hypovitaminosis D. Nuclear medicine studies were performed which showed positive bone scan, indium scan but discordant uptake on sulfur colloid scan, indicating a concern for infection (Fig. 11.27). The CT scan showed an obvious nonunion (Fig. 11.28).

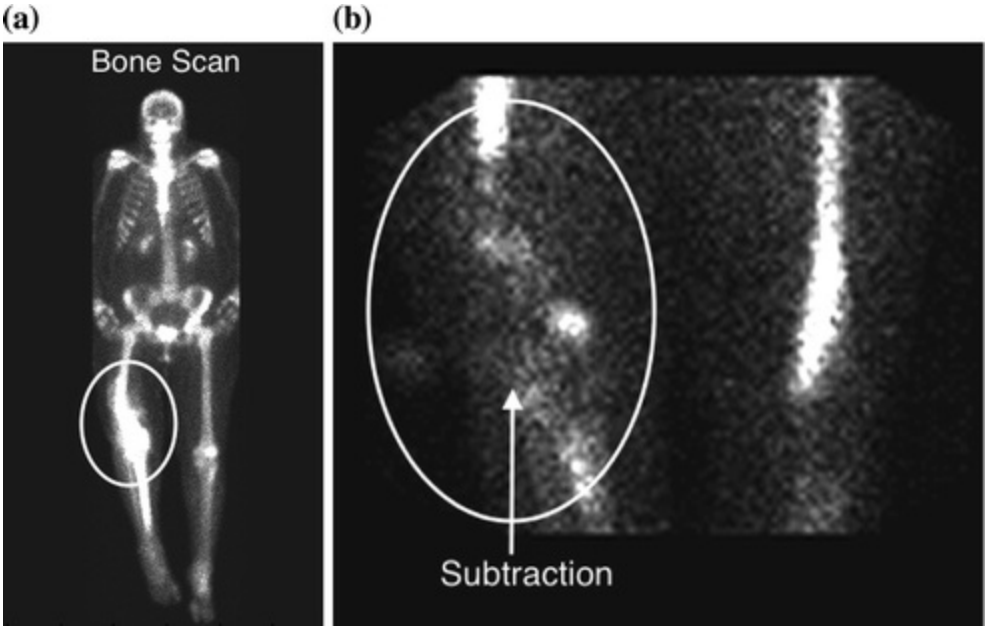



***Fig. 11.27*** Nuclear medicine studies: **a** bone scan showing increased uptake in the entire distal half of the right femur (*circled*); **b** subtraction image of sulfur colloid from indium showing areas with increased activity indicating discordant uptake and suggestive of infection

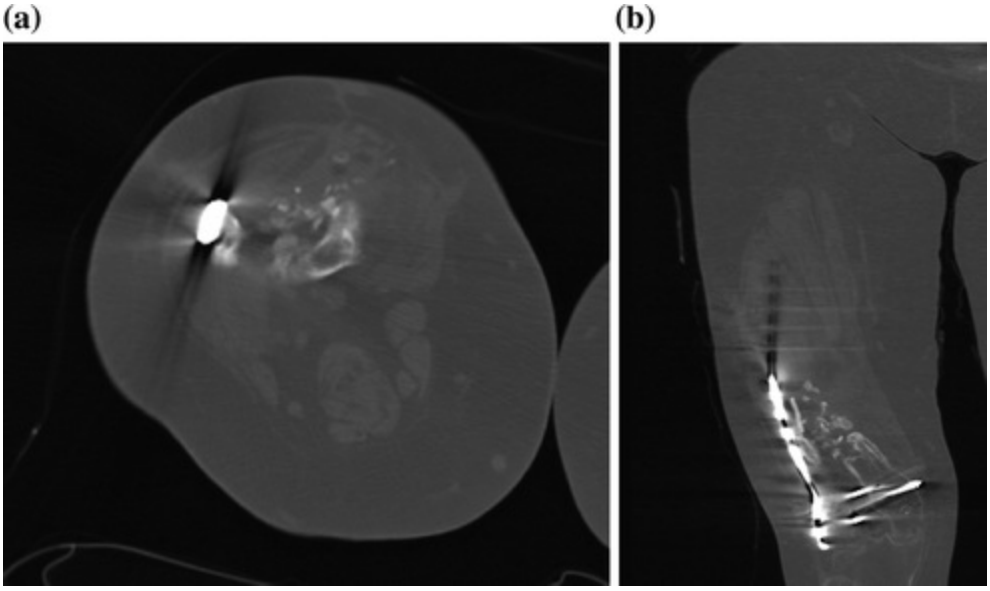



***Fig. 11.28*** Computed tomography scan images showing the lack of bridging bone and obvious nonunion; **a** axial image; **b** coronal image

Surgical options were discussed with the patient, including staging the definitive management, if there was presence of an infection. At the time of surgery, the nonunion site was evaluated after all the hardware was removed.

The native bone appeared normal. There was a significant amount of allograft 'croutons' that were loose and thus were debrided from the nonunion site. Intra-operative cultures were sent as well as a stat Gram's stain, which was negative for bacteria and only 2–3 polymorphonuclear cells (PMN) per high-power field (HPF) on frozen section of the tissue. There was no purulence, just the unincorporated bone graft. The decision was made to proceed with definitive management with the benign appearance of the nonunion site and the negative intra-operative studies. She underwent correction of her deformity through the mobile nonunion site, both angulation and rotation. There was a 50% circumferential defect for a length of about 10-cm. Retrograde nailing was performed after obtaining autogenous bone graft using the RIA system. Reaming to 16 mm was performed, and a 15 mm diameter nail was placed. The defect was packed first around the nail with calcium sulfate beads impregnated with vancomycin (off-label use) after which the autograft was packed on top. The entire defect was filled. Her knee was then manipulated after closure. We were able to passively fully extend her and flex her to 95° (Fig. 11.29).

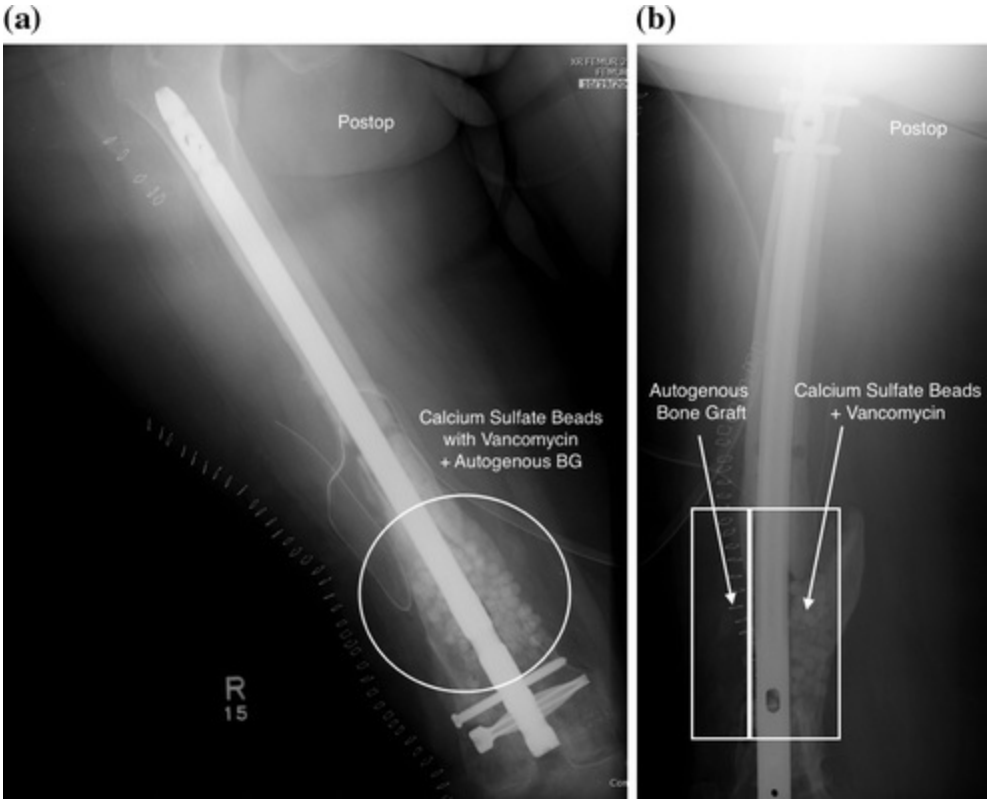



***Fig. 11.29*** Immediate postoperative **a** anteroposterior and **b** lateral right femur images showing

correction of the deformity as well as stabilization of the nonunion with a retrograde nail. **a** The nonunion site is packed with the calcium sulfate beads with vancomycin (off-label use) and the bone graft; **b** the layering of the bone graft on top of the calcium sulfate is better delineated

The patient went on to heal by 8 months as seen in Fig. 11.30. At her three-year follow-up, she maintained her 95° of flexion, had a slight leg length discrepancy of 1.5 cm managed with a shoe lift, and was ambulating with the use of a cane on occasion. She reported only occasional discomfort with weather changes (Fig. 11.31).

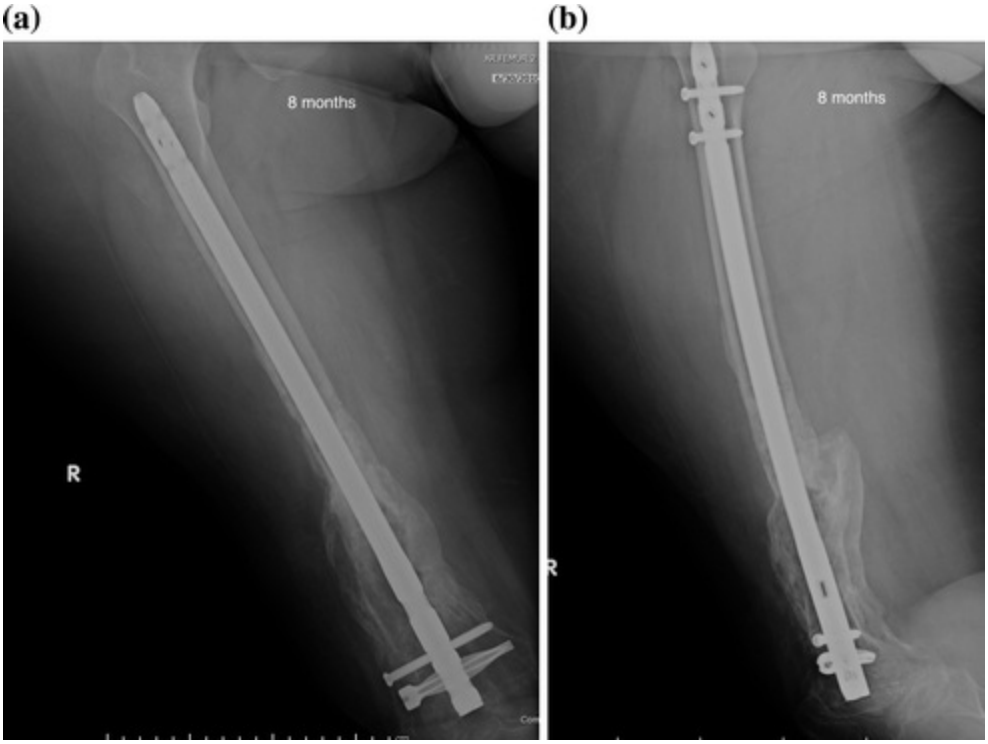



***Fig. 11.30*** **a** Anteroposterior and **b** lateral radiographs of the right femur at 8 months showing complete bridging of all 4 cortices and stable hardware


***Fig. 11.31*** Three-year follow-up **a** anteroposterior and **b** and lateral radiographs of the right femur showing continued stable hardware and further consolidation of the nonunion site

## Case 6

The patient is a 33-year-old Latin American female who was involved in a head-on MVA and sustained multiple bilateral lower extremity injuries, including a left grade IIIA open distal femur fracture with intra-articular involvement. In addition to damage control management of her other injuries, she underwent I&D and temporary bridging external fixation across the left knee (Fig. 11.32). The patient returned to the ICU and her condition improved.

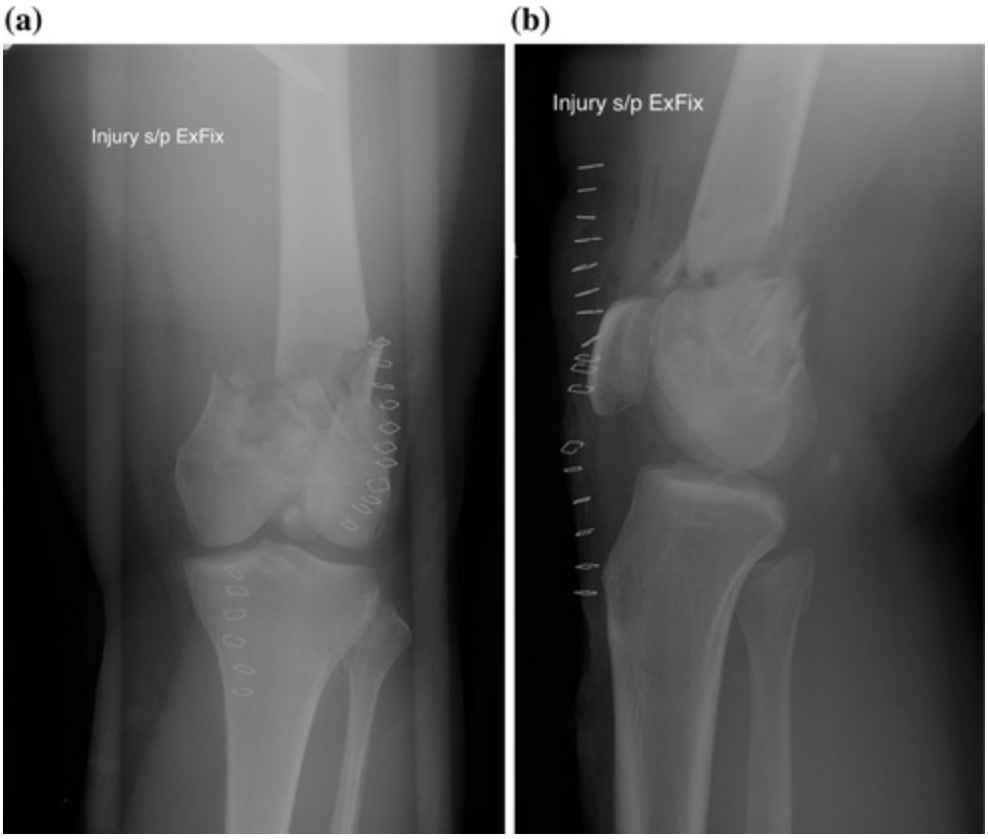



***Fig. 11.32*** Initial **a** anteroposterior and **b** lateral radiographs of the left knee after irrigation and debridement and bridging external fixation

The patient underwent definitive ORIF of her left distal femur fracture once she was stable. There was extensive comminution and bone loss in the meta-diaphyseal region extending into the trochlear region. The patient also had calcium sulfate impregnated with vancomycin and supplemented by DBM, placed into this large defect (Fig. 11.33)

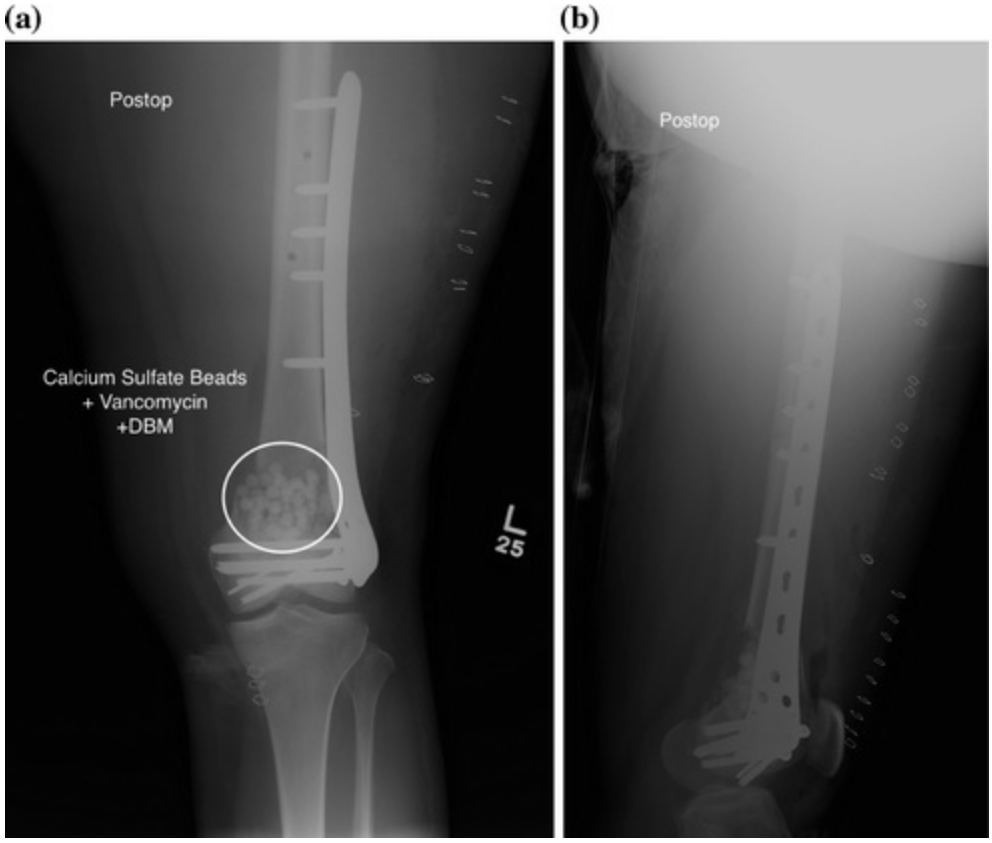



***Fig. 11.33*** Immediate postoperative **a** anteroposterior and **b** lateral images of the left femur after open reduction internal fixation and placement of calcium sulfate bead with vancomycin (off-label use) and demineralized bone matrix (DBM). A stainless steel locking condylar plate (LCP)—less invasive stabilization system (LISS) plate was used

The patient was followed and went on to heal all her other fractures, which included a left tibial plafond fracture and right patella fracture. Her femur continued to progress, and the calcium sulfate slowly resorbed with some consolidation. At seven months, she was ambulating with a cane but with some discomfort (Fig. 11.34). Due to concern over incomplete healing, a CT scan was obtained (Fig. 11.35). It revealed a large anteromedial defect with healing of the lateral cortex only. The posterior cortex appeared to have some bridging. The intra-articular component had healed completely. It was felt to be a meta-diaphyseal nonunion.

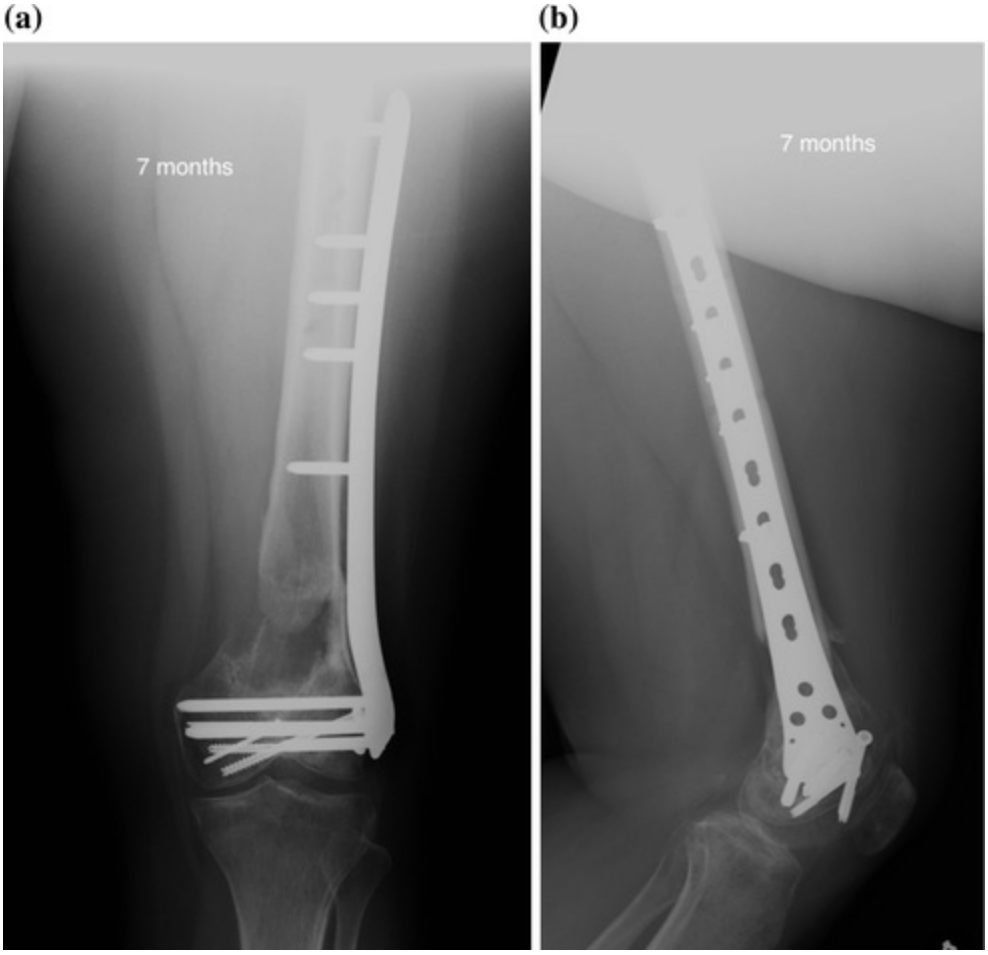



***Fig. 11.34*** At 7 months, **a** anteroposterior and **b** lateral radiographs of the left femur show complete absorption of the calcium sulfate and bridging laterally. The hardware is stable

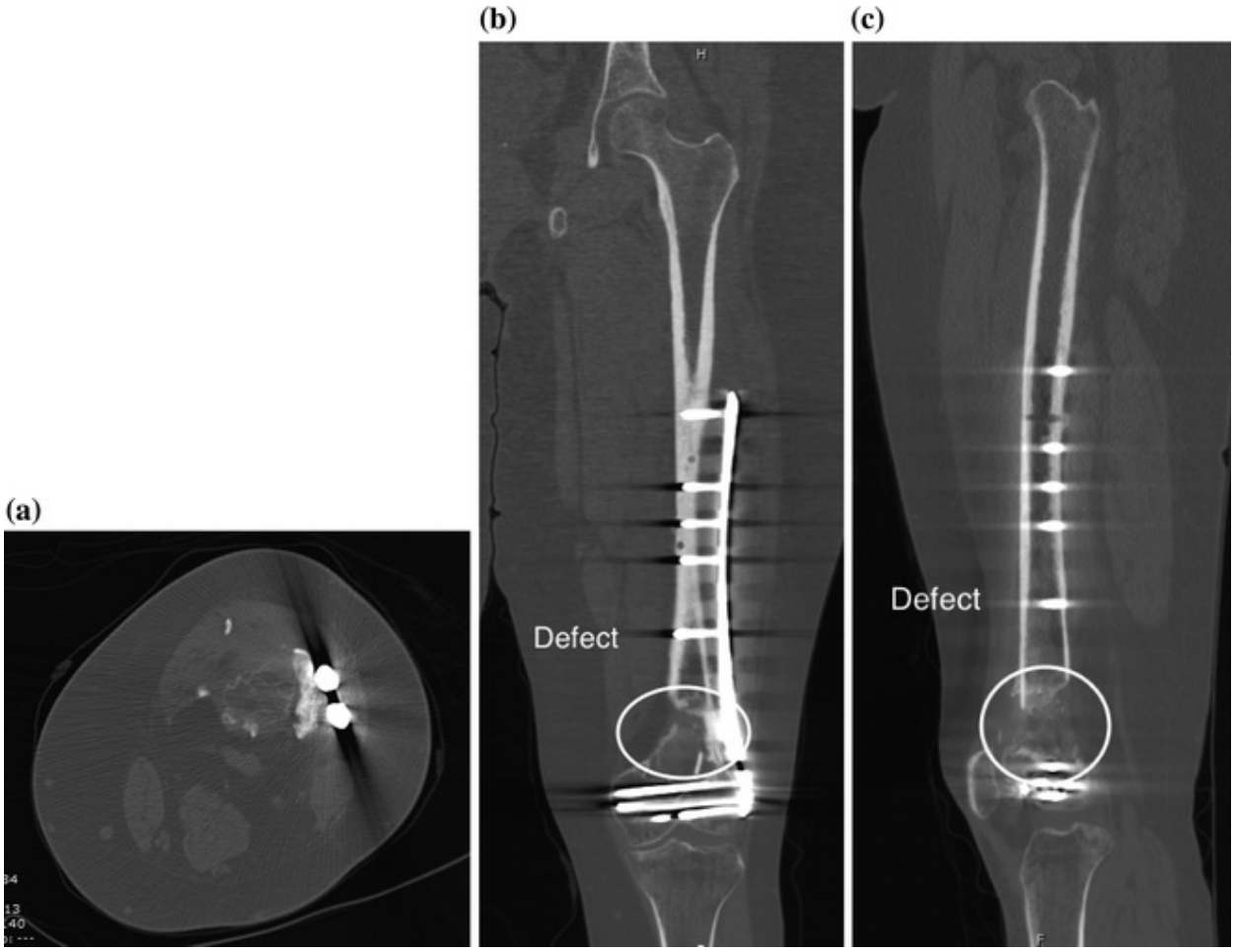



***Fig. 11.35*** Computed tomography scan images: **a** axial image shows the lateral bridging but the central nonunion; **b** coronal image shows the defect centrally but the healed lateral cortex; **c** sagittal image shows the lack of bridging bone anterior or posterior with central defect

The patient never had any problems postoperatively in terms of infection and never showed any signs of infection. All of her laboratory studies were within normal limits. She did have limited ROM to only 90° of flexion. Repair of the nonunion was discussed and she underwent surgery. Multiple options were discussed with the patient to include just autogenous bone grafting. She did not want harvesting of bone from any other site. It was decided to remove the plate and screws and place a retrograde nail during which the RIA system would be used to obtain bone graft, which could then be placed into the defect (Fig. 11.36). An open lysis of adhesions was performed while the hardware was removed. After the nail and bone graft was placed, the knee was manipulated and full flexion was obtained. The patient went on to heal by 4.5 months (Fig. 11.37). At her last follow-up

(13 months out from her nonunion repair), she had full ROM and was ambulating without assistive devices (Fig. 11.38).

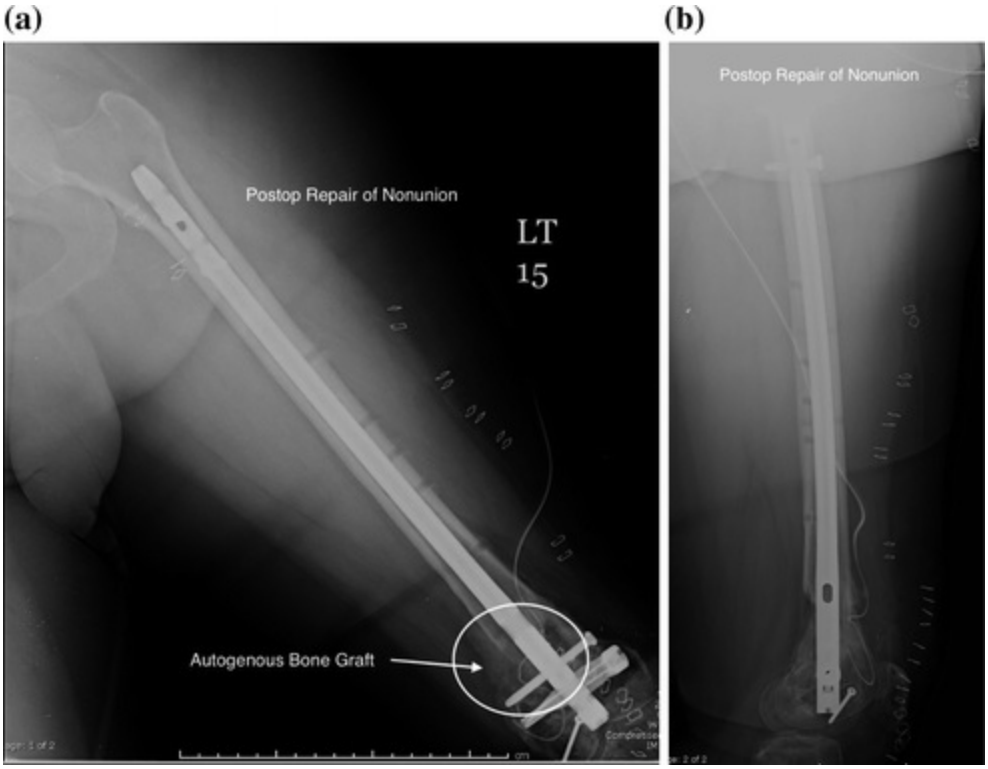



***Fig. 11.36*** Immediate postoperative **a** anteroposterior and **b** lateral images of the left femur after removal of hardware and placement of a retrograde intramedullary nail with the bone graft

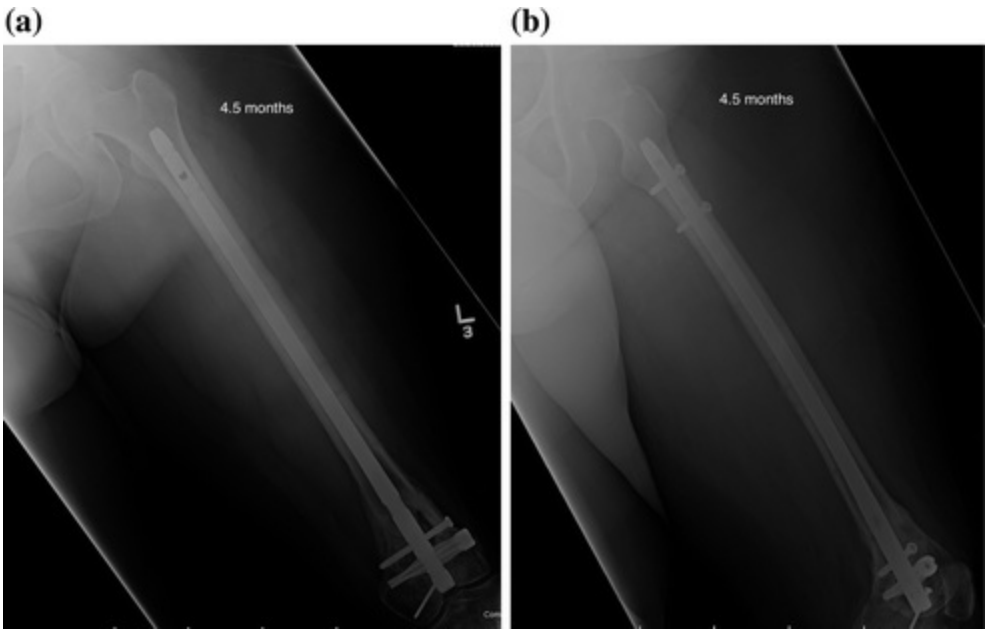


***Fig. 11.37*** At 4.5 months, the **a** anteroposterior and **b** lateral images of the left femur showed complete bridging of the nonunion site. It was felt that the patient was healed

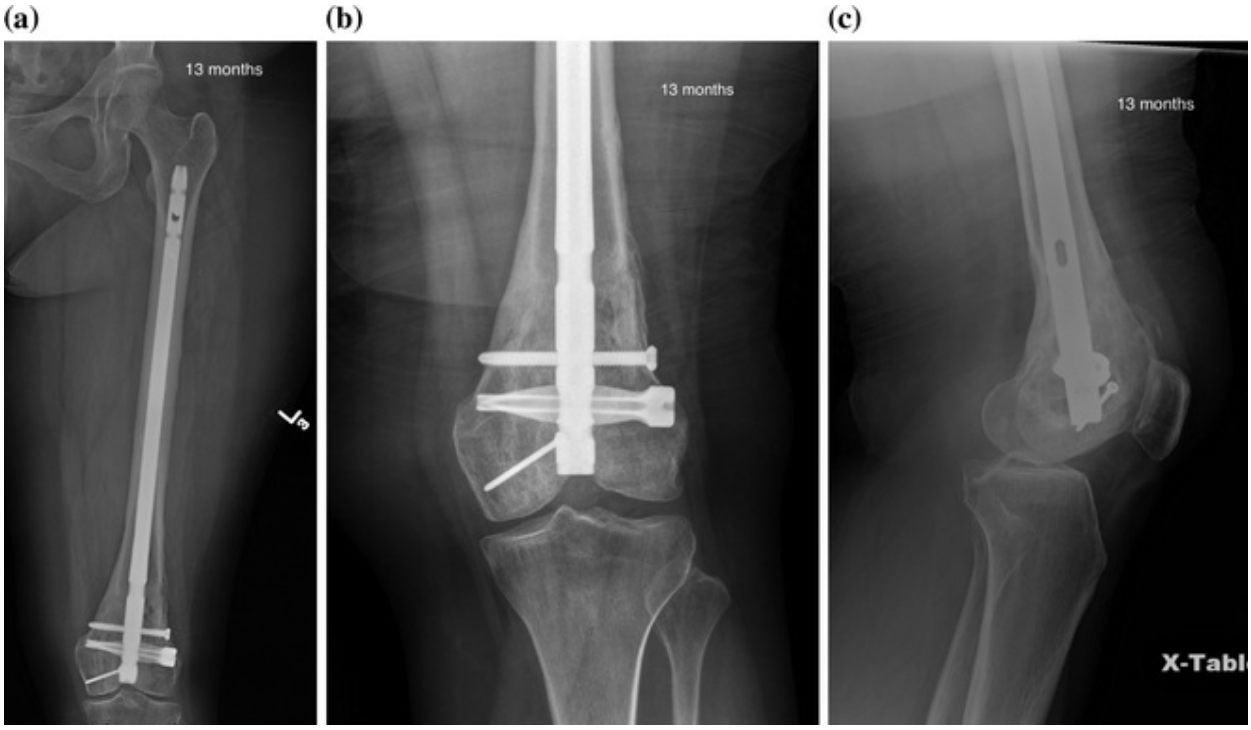



***Fig. 11.38*** At 13 months, **a** anteroposterior of the left femur and close-up; **b**, **c** anteroposterior and lateral images of the left knee showed solid consolidation of the nonunion site

# References


1. Gangavali AK, Nwachuku CO. Management of distal femur fractures in adults: an overview of options. Orthop Clin N Am. 2016;47(1):85–96.
[Crossref]

2. Ehlinger M, Ducrot G, Adam P, Bonnomet F. Distal femur fractures. Surgical techniques and a review of the literature. Orthop Traumatol. 2013;99(3):353–60.

3. Wu CC. Retrograde dynamic locked nailing for aseptic nonunion of femoral supracondyle after antegrade locked nailing. Arch Orthop Trauma Surg. 2011;131(4):513–7.
[Crossref][PubMed]

4. Forster MC, Komarsamy B, Davison JN. Distal femoral fractures: A review of fixation methods. Injury. 2006;37(2):97–108.
[Crossref][PubMed]

5. Weight M, Collinge C. Early results of the less invasive stabilization system for mechanically unstable fractures of the distal femur (AO/OTA types A2, A3, C2, and C3). J Orthop Trauma. 2004;18(8):503–8.

[Crossref][PubMed]

6. Kregor PJ, Stannard JA, Zlowodzki M, Cole PA. Treatment of distal femur fractures using the less invasive stabilization system: surgical experience and early clinical results in 103 fractures. J Orthop Trauma. 2004;18(8):509–20.
[Crossref][PubMed]

7. Hoffmann MF, Jones CB, Sietsema DL, Tornetta P 3rd, Koenig SJ. Clinical outcomes of locked plating of distal femoral fractures in a retrospective cohort. J Orthop Surg Res. 2013;8:43.
[Crossref][PubMed][PubMedCentral]

8. Henderson CE, Lujan TJ, Kuhl LL, Bottlang M, Fitzpatrick DC, Marsh JL. 2010 Mid-America Orthopaedic Association physician in training award: healing complications are common after locked plating for distal femur fractures. Clin Orthop Relat Res. 2011;469(6):1757–65.
[Crossref][PubMed][PubMedCentral]

9. Henderson CE, Kuhl LL, Fitzpatrick DC, Marsh JL. Locking plates for distal femur fractures: is there a problem with fracture healing? J Orthop Trauma. 2011;25(Suppl 1):S8–14.
[Crossref][PubMed]

10. Moloney GB, Pan T, Van Eck CF, Patel D, Tarkin I. Geriatric distal femur fracture: are we underestimating the rate of local and systemic complications. Injury. 2016;47(8):17326.
[Crossref]

11. Ebraheim NA, Martin A, Sochacki KR, Liu J. Nonunion of distal femoral fractures: a systematic review. Orthop Surg. 2013;5(1):46–50.
[Crossref][PubMed]

12. Gardner MJ, Toro-Arbelaez JB, Harrison M, Hierholzer Christian, Lorich DG, Helfet DL. Open reduction and internal fixation of distal femoral nonunions: long-term functional outcomes following a treatment protocol. J Trauma. 2008;64(2):434–8.

13. Henderson CE, Lujan T, Bottlang M, Fitzpatrick DC, Madey SM, Marsh JL. Stabilization of distal femur fractures with intramedullary nails and locking plates: differences in callus formation. Iowa Orthop J. 2010;30:61–8.
[PubMed][PubMedCentral]

14. Lujan TJ, Henderson CE, Madey SM, Fitzpatrick DC, Marsh JL, Bottlang M. Locked plating of distal femur fractures leads to inconsistent and asymmetric callus formation. J Orthop Trauma. 2010;24(3):156–62.
[Crossref][PubMed]

15. Rodriguez EK, Boulton C, Weaver MJ, Herder LM, Morgan JH, Chacko AT, et al. Predictive factors of distal femoral fracture nonunion after lateral locked plating: a retrospective multicenter case-control study of 283 fractures. Injury. 2014;45(3):554–9.
[Crossref][PubMed]

16. Rodriguez EK, Zurakowski D, Herder L, Hall A, Walley KC, Weaver MJ, Appleton PT, Vrahas M. Mechanical construct characteristics predisposing to non-union after locked lateral plating of distal femur fractures. J Orthop Trauma. 2016;30(8):403–8.
[Crossref][PubMed]

17. Canadian Orthopaedic Trauma Society. Are locking constructs in distal femoral fractures always best? A prospective multicenter randomized controlled trial comparing the less invasive stabilization system with the minimally invasive dynamic condylar screw system. J Orthop Trauma. 2016;30(1):e1–6.
[Crossref]

18. Vallier HA, Immler W. Comparison of the 95-degree angled blade plate and the locking condylar plate for the treatment of distal femoral fractures. J Orthop Trauma. 2012;26(6):327–32.
[Crossref][PubMed]

19. Southeast Fracture Consortium. LCP versus LISS in the treatment of open and closed distal femur fractures: does it make a difference? J Orthop Trauma. 2016;30(6):e212–6.
[Crossref]

20. Bottlang M, Doornink J, Lujan TJ, Fitzpatrick DC, Marsh JL, Augat P, et al. Effects of construct stiffness on healing of fractures stabilized with locking plates. J Bone Joint Surg Am. 2010;92(Suppl 2):12–22.
[Crossref][PubMed][PubMedCentral]

21. Bottlang M, Fitzpatrick DC, Sheerin D, Kubiak E, Gellman R, Vande Zandschulp C, et al. Dynamic fixation of distal femur fractures using far cortical locking screws: a prospective observational study. J Orthop Trauma. 2014;28(4):181–8.
[Crossref][PubMed]

22. Ricci WM, Streubel PN, Morshed S, Collinge CA, Nork SE, Gardner MJ. Risk factors for failure of locked plate fixation of distal femur fractures: an analysis of 335 cases. J Orthop Trauma. 2014;28(2):83–9.
[Crossref][PubMed]

23. Vallier HA, Hennessey TA, Sontich JK, Patterson BM. Failure of LCP condylar plate fixation in the distal part of the femur: A report of six cases. J Bone J Surg Am. 2006;88(4):846–53.

24. Barei DP, Beingessner DP. Open distal femur fractures treated with lateral locked implants: union, secondary bone grafting, and predictive parameters. Orthopedics. 2012;35(6):e843–6.
[Crossref][PubMed]

25. Kammerlander C, Riedmuller P, Gosch M, Zegg M, Kammerlander-Knauer U, Schmid R, Roth T. Functional outcome and mortality in geriatric distal femoral fractures. Int J Care Injured. 2012;43(7):1096–101.
[Crossref]

26. Wu CC. Retrograde dynamic locked nailing for femoral supracondylar nonunions after plating. J Trauma. 2009;66(1):195–9.
[Crossref][PubMed]

27. Chapman M, Finkemeier CG. Treatment of supracondylar nonunions of the femur with plate fixation and bone graft. J. Bone Joint Surg. 1999;81(9):1217–28.
[Crossref][PubMed]

28. Bellabarba C, Ricci WM, Bolhofner BR. Indirect reduction and plating of distal femoral nonunions. J Orthop Trauma. 2002;16(5):287–96.
[Crossref][PubMed]

29. Bellabarba C, Ricci WM, Bolhofner BR. Results of indirect reduction and plating of femoral shaft nonunions after intramedullary nailing. J Orthop Trauma. 2001;15(4):254–63.
[Crossref][PubMed]

30. Wang JW, Weng LH. Treatment of distal femoral nonunion with internal fixation, cortical allograft struts, and autogenous bone-grafting. J Bone Joint Surg Am. 2003;85A(3):436–40.
[Crossref]

31. Amorosa LF, Jayaram PR, Wellman DS, Lorich DG, Helfet DL. The use of the 95-degree-angled blade plate in femoral nonunion surgery. Eur J Orthop Surg Traumatol. 2014;24(6):953–60.
[Crossref][PubMed]

32. Holzman MA, Hanus BD, Munz JW, O'Connor DP, Brinker MR. Addition of a medial locking plate to an in situ lateral locking plate results in healing of distal femoral nonunions. Clin Orthop Relat Res. 2016;474(6):1498–505.
[Crossref][PubMed][PubMedCentral]

33. Koval KJ, Seligson D, Rosen H, Fee K. Distal femoral non-union: treatment with retrograde inserted locked intramedullary nail. J Orthop Trauma. 1995;9(4):285–91.
[Crossref][PubMed]

34. Ali F, Saleh M. Treatment of distal femoral nonunions by external fixation with simultaneous length and alignment correction. Injury. 2002;33(2):127–34.
[Crossref][PubMed]

35. Haidukewych GJ, Springer BD, Jacofsky DJ, Berry DJ. Total knee arthroplasty for salvage of failed internal fixation or nonunion of the distal femur. J Arthroplasty. 2005;20(3):344–9.
[Crossref][PubMed]

36. Davila J, Malkani A, Paiso JM. Supracondylar distal femoral nonunions treated with a megaprosthesis in elderly patients: a report of two cases. J Orthop Trauma. 2001;15(8):574–8.
[Crossref][PubMed]

37. Vaishya R, Singh AP, Hasija R, Singh AP. Treatment of resistant nonunion of supracondylar fractures femur by megaprosthesis. Knee Surg Sports Traumatol Arthrosc. 2011;19(7):1137–40.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_12

# 12. Nonunions of the Tibial Plateau and Proximal Tibial Metaphysis

Thomas F. Higgins[1]

(1) Chief of Orthopaedic Trauma, Richard L. Stimson Presidential Endowed Chair, Department of Orthopedics, University Orthopedic Center, University of Utah Health Sciences, 590 Wakara Way, Salt Lake City, UT 84108, USA

**Thomas F. Higgins**
**Email:** Thomas.Higgins@hsc.utah.edu

**Keywords** Proximal tibial nonunion – Plateau nonunion

## 12.1 Epidemiology

The risk of nonunion after operative treatment of intra-articular tibial plateau fracture is considered to be very low [1, 2]. Perhaps the reason for the very low rate of nonunion is the rich extraosseous blood supply of the metadiaphyseal region, as thoroughly elucidated by Borrelli et al. [3]. Branches of the popliteal artery perfuse this metaphyseal zone from the anterior tibial artery laterally and from the posterior tibial artery medially. Given the relative paucity of cases in the literature on this topic, the best way to examine the incidence is by separating articular from extra-articular injuries, and further dividing by method of treatment.

Historically, nonunion of intra-articular proximal tibial fractures is considered quite rare. Shatzker et al. [4] published a 1979 series of 94 plateau fractures with various treatments and all united. In 1987, Moore and

colleagues from the University of Southern California described between one- and ten-year follow-up on 399 plateau fractures treated with or without operation, and reported no nonunions [5]. Nonunion of articular injuries after nonoperative treatment was felt to be so rare that in 1991 King and Schatzker deemed the report of this occurrence to be worthy of a case report [6]. In a large series of open reduction and internal fixation (ORIF) with plate and screw fixation, Barei et al. [7] reported one case out of 83 bicondylar fractures that went onto nonunion. Egol's series of 36 plateau fractures treated with a LISS (Less Invasive Stabilization System) plate featured two cases that were "prophylactically bone grafted" at the two month mark for metaphyseal bone loss [8]. Phisitkul's [9] series of minimally invasive plating of plateau fractures demonstrated very high infection (22%) and malunion (22%) rates in their series of mostly 41-C fractures, but they did not report on nonunions. Similarly, Rademaakers et al. [10] reported long-term follow-up on a series of 109 fractures treated with ORIF in which 4% were reported as malunion, but no nonunions were described. However, in stark contrast to most papers on ORIF, a 2014 paper by Ruffolo et al. [11] showed a 10% nonunion rate in their series of 138 bicondylar plateau fractures treated with plates and screws .

Skinny wire (or circular) external fixation also seems to demonstrate a low rate of nonunion. A series of 24 plateau fractures from University of California at Los Angeles treated with Ilizarov technique had no nonunions [12]. Similarly, the Canadian Orthopedic Trauma Society randomized 83 patients to circular frames or ORIF for their plateau fractures, and reported no nonunions [13]. Watson and Coufal [14] reported a series of 14 unicondylar plateau fractures treated with circular frames, and all went on to union. Weiner's series of 50 proximal tibial fractures treated with "hybrid" external fixation also demonstrated a 100% union rate [15]. Ahern also described a series of 54 plateau fractures treated with either circular frames or locking plates, and all fractures went onto union [16] .

A number of papers address extra-articular proximal tibia fractures, and various methods used to address these injuries. Martinez and coauthors reported on their series of 108 proximal tibia extra-articular fractures treated closed with a functional brace [17]. The resultant three nonunions yielded a nonunion rate of 2.7%. Naik et al. [18] reported on percutaneous locked plating for extra-articular proximal tibia fractures in 2013. Similar to the Phisitkul study, which concerned mostly articular injuries, these 49 fractures

had a high malunion rate (20%), but the three nonunions in the series (6%) were all in infected cases. A large amount of the data on extra-articular tibia fractures is based on efforts to use intramedullary fixation to address proximal tibial metaphyseal injuries. Ricci's series of eleven intramedullary nails with blocking screws for extra-articular proximal tibia produced one nonunion [19]. Freedman and Johnson [20] reported malalignment in 7 of 12 nailed proximal tibias, but did not report on nonunion . Krettek's series of 21 tibia fractures treated with Poller screws mentions one case that required eventual autograft [21]. Tornetta's series of 25 proximal tibial fractures treated with a nail, using a semi-extended technique, reported zero subsequent nonunions [22]. In contrast, Lang and coauthors found in their series of proximal third tibia fractures treated with a nail that 9 of 32 (28%) required exchange nailing or bone grafting to unite, and two failed to unite entirely [23]. A 2012 series by Kulkarni et al. [24] showed 5 nonunions out of 75 proximal tibial fractures (7%) treated with Poller screws and intramedullary nailing.

## 12.2 Diagnosis

Diagnosis of proximal tibia nonunion is based on persistent discomfort with ambulation, radiographic evidence of nonunion, or progressive deformity of the proximal tibia over time. It is not clear how far after repair it is reasonable to declare a fracture nonunited. The development of sclerotic margins along the intra- or extra-articular portion of the fracture on consecutive radiographic images may be indicative of an impending or established nonunion. Definitive diagnosis is often aided by a computed tomography (CT) scan , which is also helpful in planning for repair if the diagnosis is confirmed. Although rare, intra-articular nonunion may be very difficult to definitively establish on plain radiographs due to overlying hardware, and so a CT scan is practically mandatory to establish this diagnosis with confidence.

After establishing the diagnosis, etiology must be evaluated before treatment can be pursued. Patient behavioral and metabolic factors must be evaluated as potential causative agents. (For the etiology of nonunions, see Chap. 1, "Nonunions: Diagnosis, Evaluation and Management.")

For all open fractures and all fractures that have been operatively repaired, infection must be entertained as a potential etiological factor for nonunion. Workup for all previously operated *or* open fractures should

include complete blood count, erythrocyte sedimentation rate, and C-reactive protein. Any persistent drainage should be considered evidence of infection until proven otherwise. A patient history of intermittent prescription of oral antibiotic to suppress drainage should also be considered *highly* suggestive of infection. Nuclear medicine examinations such as tagged white blood cell scan have been recommended if the presence or absence of infection cannot be definitively established, but the results of these tests are frequently ambiguous or confusing. Magnetic resonance imaging with gadolinium contrast has also been suggested to evaluate infection, but in the presence of hardware from prior surgery, this is often not feasible or helpful. If any of these investigations indicate infection, a staged approach to treatment, including open biopsy before definitive fixation, is recommended.

The final etiologic contributors to nonunion may be grouped under the heading of "surgeon factors." These include malalignment and excessive stripping of the surrounding soft tissues at the time of index surgery. Malalignment of the proximal tibial metaphysis may preclude union, due to alterations in the mechanical axis, and may be suggested on either physical examination or radiographic evaluation. Patient history may include subjective progressive loss of limb alignment, which may be confirmed with a full-length weight-bearing film of the bilateral lower extremities to evaluate side-to-side difference and limb length discrepancy. Rotational malalignment may be harder to discern definitively, but must be assessed either with gunsight CT examination or prone physical examination (thigh-foot axis) to uncover any tibial rotational asymmetry. Malalignment must be addressed at the time of definitive fixation in order to make union possible.

Surgical insult to the soft tissues and blood supply may be evidenced radiographically with a "wide surgical footprint." The placement of multiple plates on multiple aspects of the tibial metaphysis through a single incision may indicate that there was extensive stripping of the blood supply at the time of index surgery. In addition to stable fixation, nonunions that show radiographic signs of having been previously aggressively devascularized most often require autograft to address the biologic deficit.

## 12.3 Treatment

Assuming a staged approach for cases where infection is suspected, treatment will focus on repair of the aseptic nonunion.

In cases of suspected or confirmed infection, a staged approach is fairly standard. Briefly, this consists of standard laboratory values, to include complete blood count with differential, erythrocyte sedimentation rate, and C-reactive protein. These will serve as baseline levels. Prior to the index debridement and cultures, the patient is given a one-week holiday from antibiotics, presumably to maximize the sensitivity of tissue cultures. The goal of debridement is twofold: to eliminate infected tissue and to obtain accurate cultures. If debridement of nonviable bone results in a defect, antibiotic cement beads may be placed locally to deliver treatment directly to the affected site. If the defect is sizable or results in instability, a cement spacer may be placed to fill this defect, with the potential secondary benefit of enhancing stability. If the debridement results in substantial increase in instability, temporary external fixation may need to be considered. In the event of positive cultures, the patient is treated with a minimum of 6 weeks (or longer, if recommended by infectious disease consultation) of antibiotics. The requirements to resume definitive treatment of the nonunion after treatment of infection are as follows:

1. A dry healed incision with no drainage or warmth

2. Absence of fevers or other constitutional symptoms of infection

3. Two weeks off antibiotics without recurrence of drainage or symptoms

4. Improvement, if not complete normalization, of laboratory inflammatory parameters.

A more persistent or chronic infection in the setting of nonunion may influence the choice of definitive fixation, weighing in favor of circular frame methods.

Intra-articular nonunion, although rare, requires a very thorough understanding of the fragment morphology before repair can be undertaken. This is most often achieved with a CT scan. In some instances, part of an intra-articular fragment may be united, and part not. Because articular congruity and absolutely stability are the primary goals in order to preserve the joint surface and achieve healing, open reduction of the joint stabilized with plate and screw fixation are the treatments of choice. The nonunited

fragments are usually partial articular (essentially “B-type” patterns), so lag screws and nonlocking buttress fixation are the standard methods of fixation.

However, prior to rigidly repairing the nonunion site, the metaphyseal extension of the fragment must be thoroughly debrided. This will often entail removing some healed bony tissue where the fragment is partially united at the metaphysis. But if the fibrous tissue and callus are not completely removed from the nonunion site, the fragment cannot be anatomically reduced to the remaining tibia. This may often be quite time consuming and tedious, but obtaining correct reduction without this step is not possible. Dissection of the cortical side of the nonunited fragment should be avoided to minimize the vascular insult to this site, but the fragment must be adequately mobilized to achieve correct alignment of the fragment **(**Fig. 12.1). An excellent description of the technique of René Marti, as applied with an opening wedge osteotomy for valgus malunion after plateau fracture, can be found in a paper by Kerkhoffs and coauthors [25].

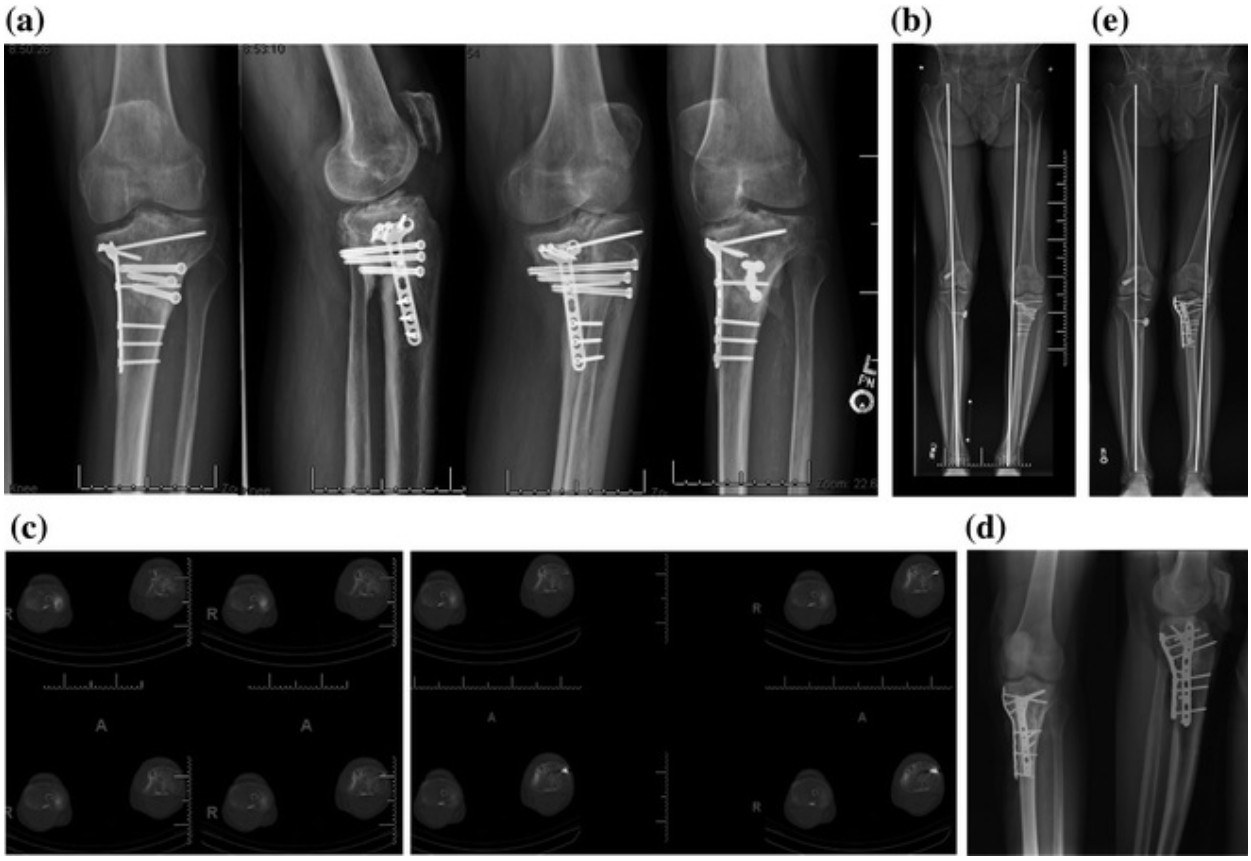



***Fig. 12.1*** **a** Knee radiographs demonstrate varus deformity and apparent intra-articular nonunion. **b** Full-length weight-bearing view of the bilateral lower extremities confirms varus malalignment as well

as the relative limb shortening that results from varus. **c** Computed tomography scan better defines the morphology of the nonunited fragment and the partial union of the metaphysis. **d** Correction with compression and buttress plating through a single posteromedial approach restores articular congruity and alignment. **e** Films at one-year follow-up demonstrate good healing with restoration of alignment and articular congruity. Long-standing films actually demonstrate some valgus malalignment following surgical correction. Patient reported markedly improved function clinically

Autologous bone grafting may or may not be necessary in this setting. If needed, iliac crest has been utilized as the conventional harvest site for cancellous bone. When operating on the lower extremity, the proximal tibial metaphysis is a convenient source of autograft, but this may be difficult if the proximal tibia is the focal point of the established nonunion. However, when working on a lateral articular nonunion, the medial metaphysis may be used as a source, and when working on lateral side, medial graft may be collected. Once placed within the metaphyseal nonunion site, the bone graft may be so bulky as to be an impediment to anatomic reduction of a partial articular fragment, and in this case, the graft must be packed into a vacancy in the metaphysis, or not utilized.

Once anatomic reduction is achieved, clamps may be utilized to provisionally stabilize the nonunion and provide compression. Buttress fixation then relies on a well-contoured, or even slightly “under-contoured” plate, with the initial point of fixation being just distal to the apex of the nonunited fragment. If the patient is especially osteopenic, due to age, disease, or prolonged nonweight-bearing, cortical purchase of the buttress screws may be compromised, requiring the use of locking fixation. Otherwise, nonlocking fixation should be ideal for this indication, and lag screws may be used, either through the plate or around the plate, for further rigid fixation of the nonunited articular fragment **(**see Fig. 12.1d).

Extra-articular nonunion of the proximal tibia presents a wider array of treatment options. Given the location, both hypertrophic and oligotrophic nonunions would likely benefit from compression in this area, but this can be achieved with nailing, plate and screw constructs, or circular frames.

As discussed earlier, correction of alignment is of the highest priority in addressing proximal tibial nonunions. Extra-articular nonunions will usually feature deformity in one or more of these planes (coronal, sagittal, rotational). In the author’s experience, autogenous bone graft is routinely utilized for almost all of these nonunions.

The two most important variables in determining the ultimate method of fixation are (1) the size of the proximal segment and (2) the degree of

deformity. For larger proximal segments, where adequate fixation may be achieved with an intramedullary device, this method may be preferred **(**Figs. 12.2 and 12.3 **)**. Blocking screws may be employed to maintain correct alignment and enhance stability, and a variety of methods may be used to achieve compression with an intramedullary rod. Blocking screws are generally left in place at the conclusion of the operation (as opposed to removing them after rodding) to help maintain alignment as the fracture heals. Compression may be achieved by locking the rod distally first, and then "backslapping" the intramedullary rod, or via compression devices applied through the application jig of many nail systems. Another method for compression over a rod is dynamic locking, but this may not be recommended in the setting of a potentially less stable metaphyseal nonunion.

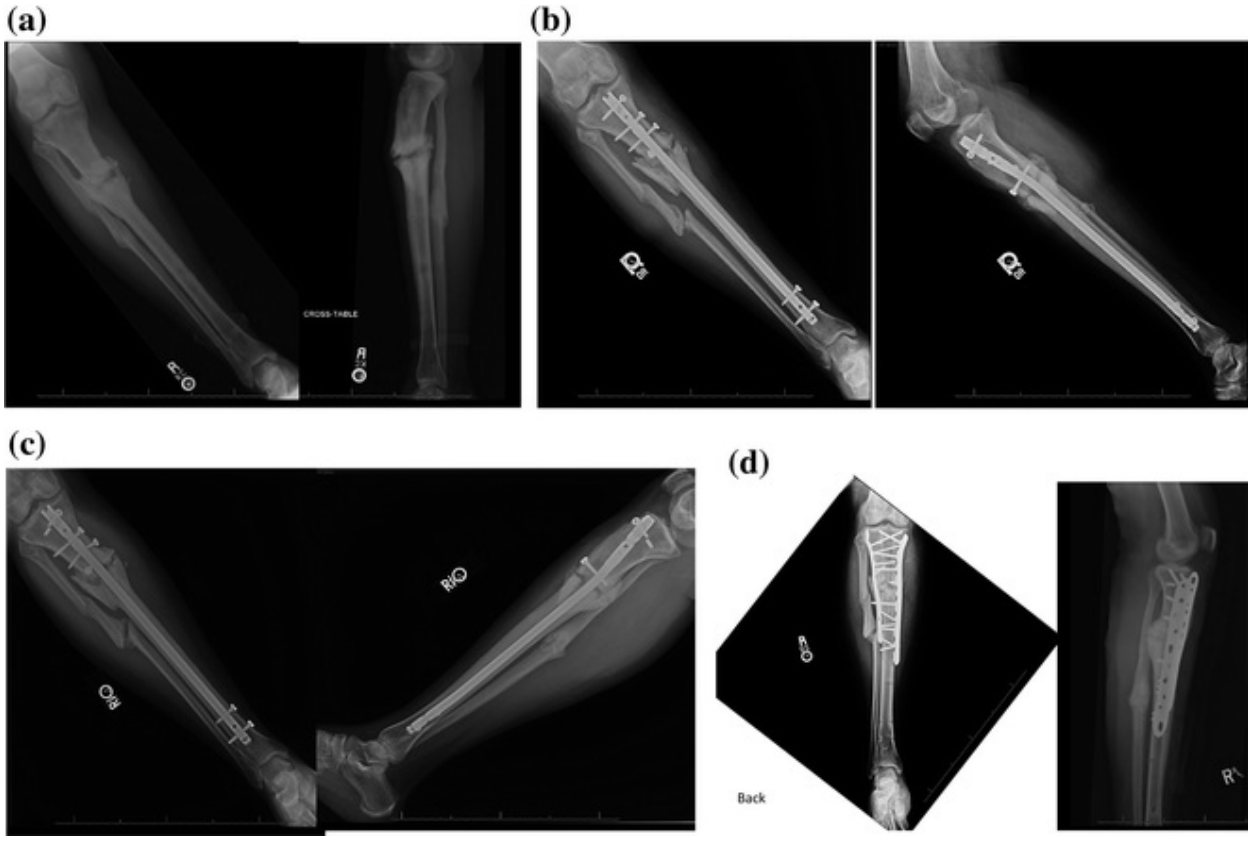



***Fig. 12.2*** **a** Presenting films show frank nonunion of the proximal tibial metaphysis and varus/procurvatum deformity. **b** Correction was performed in an open manner with autogenous cancellous bone graft and osteotomy of previously healed fibular fracture. The repair was fixed with an intramedullary rod utilizing blocking screws and compression through the rod. Immediate weight bearing was permitted. **c** The return of pain at the nonunion site and broken screws after six months demonstrated persistent nonunion. **d** Medial and lateral compression plating with bone grafting

ultimately achieved osseous union


***Fig. 12.3*** **a** Presenting radiographs 4 months after fracture demonstrate acceptable alignment and some effort to heal the fracture, despite the nonunion. **b** Assuming the fracture would benefit from further stability but minimal insult to the local biology, a closed nailing was performed with resulting union and good function

Smaller articular-epiphyseal segments may be better captured and

compressed with either locking plate constructs or with Ilizarov techniques. The degree of deformity may drive choice of fixation based on (1) suitability of implants to the corrected deformity or (2) the pliability of soft tissue coverage at the concavity of the nonunion. For difficult soft tissue coverage situations, circular frame correction over time may allow for gradual correction and slow expansion of the soft tissue envelope. This technique may also be helpful in the setting of questionable patient compliance, as a circular frame may permit immediate weight bearing **(**Fig. 12.4). For small peri-articular blocks, the author prefers dual locking plates applied separately through two incisions, but familiarity with Ilizarov techniques is required if soft tissues will not tolerate immediate on-table complete correction of alignment.

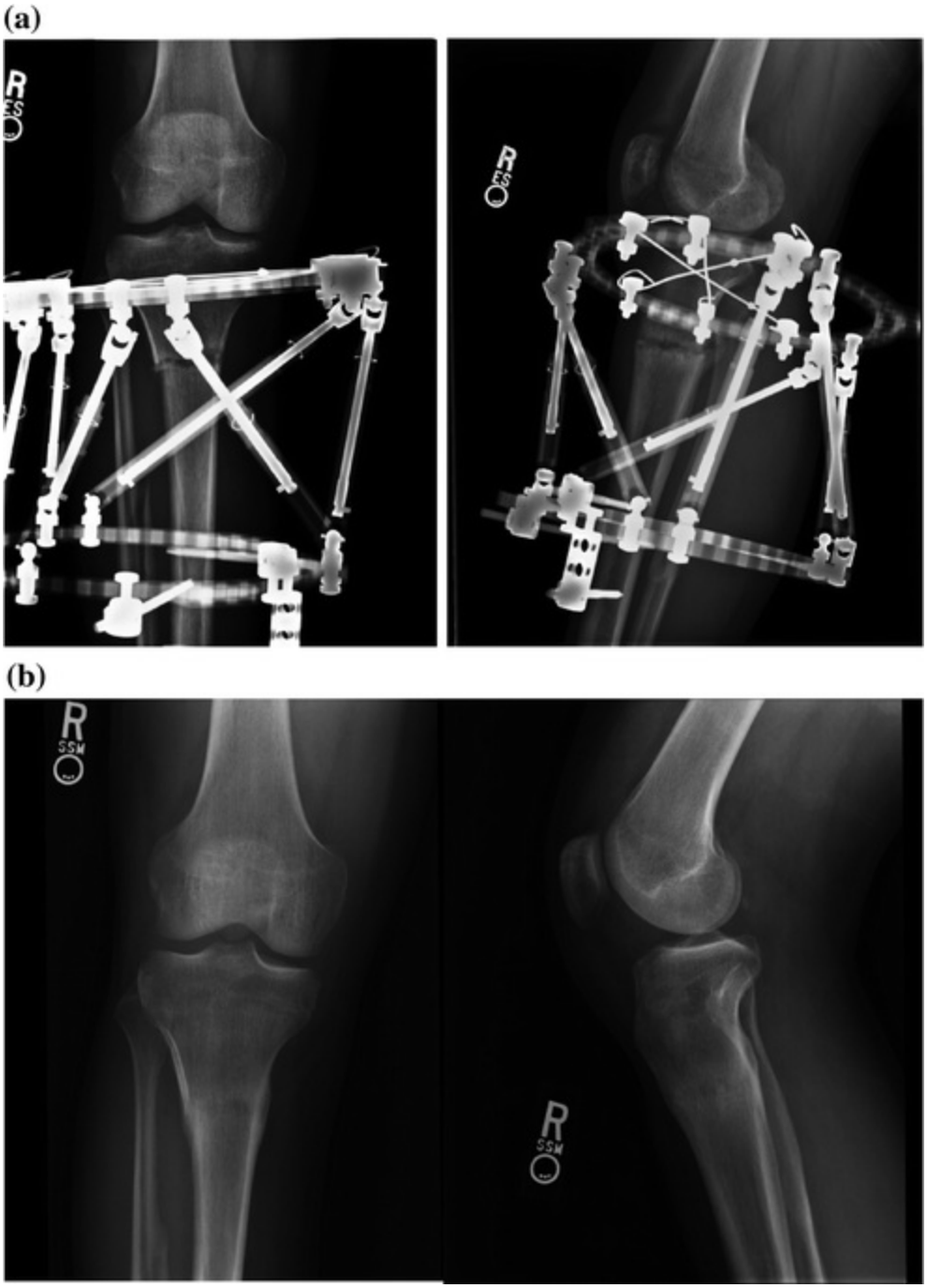



***Fig. 12.4*** **a** Nonunion with ring fixator applied. **b** Healed nonunion after removal of frame

To utilize locking plates for small peri-articular blocks, start with adequate debridement of the metaphyseal nonunion site and correction of alignment. Alignment correction may be aided with an AO (Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation) universal distractor, or by leveraging the plates that will

ultimately be used for fixation. The author prefers to establish proximal locking fixation both medially and laterally before imparting compression and final fine-tuning of alignment. Balanced compression of each plate may then be used to fine-tune coronal plane alignment (Figs. 12.5 and 12.6 **)**. Intraoperatively, fluoroscopy may not be able to adequately confirm coronal plane alignment, and this may require an intraoperative plain radiograph before finalizing the distal fixation (see Fig. 12.6c).

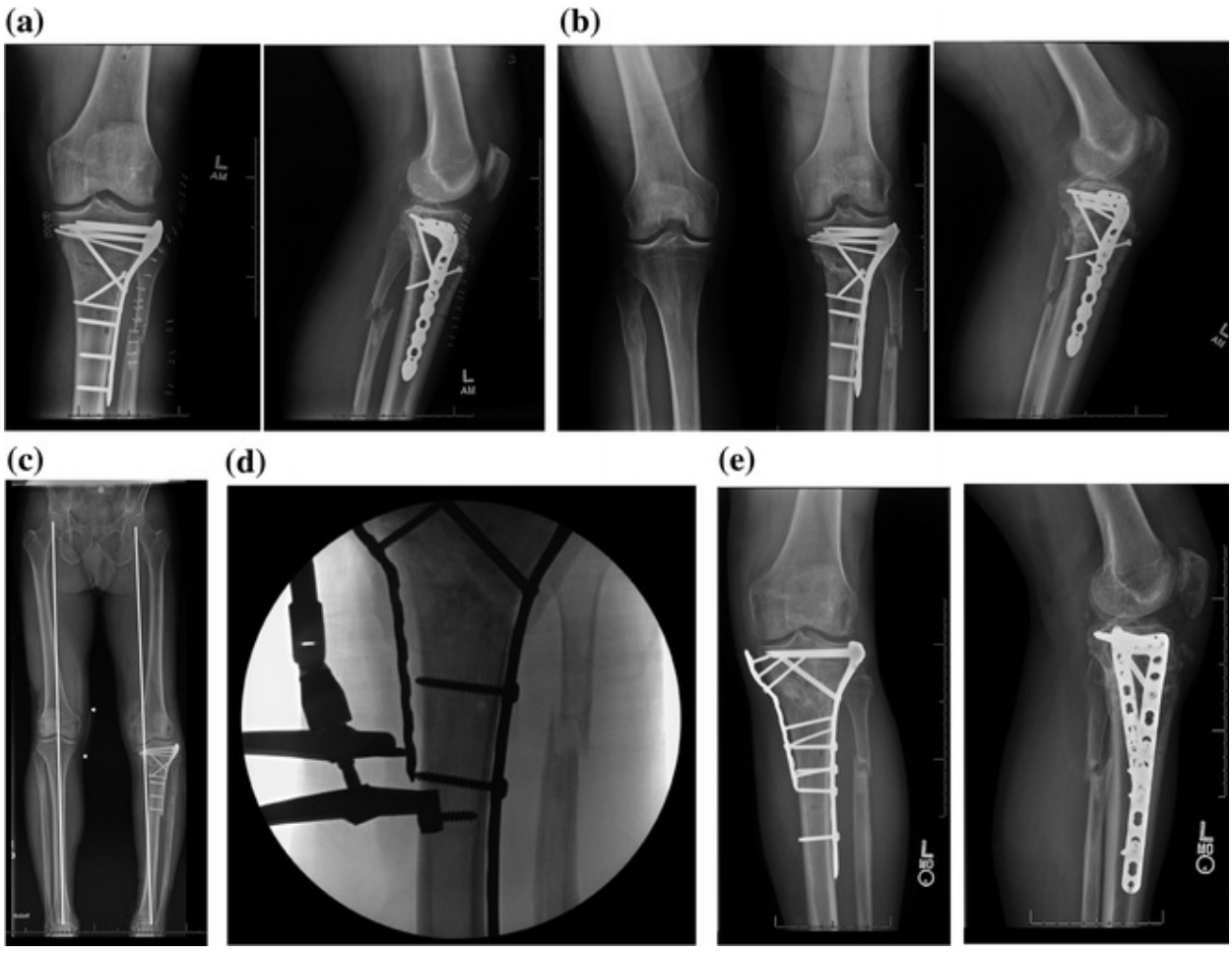



***Fig. 12.5*** **a** Original postoperative films show restoration of correct alignment. **b** Four months postoperatively, deformity is present and fracture is not healed. **c** Full-length weight-bearing view of the bilateral lower extremities confirms varus alignment. **d** After grafting, lateral locked plating is applied in some valgus. Subsequent application of a medially based articulated tensioning device provides compression and correction of alignment to anatomic. **e** Three-month follow-up films already demonstrate solid union

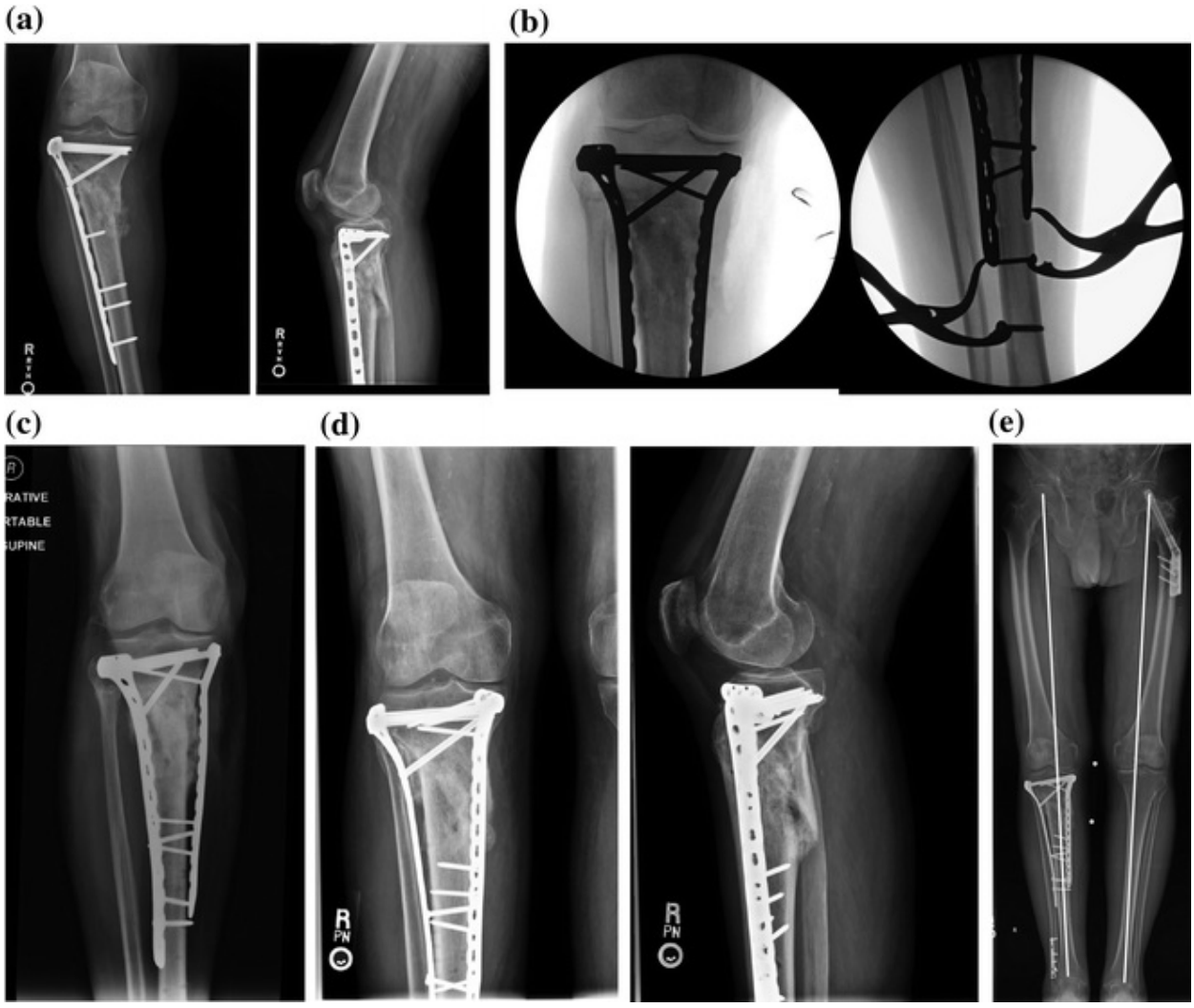



***Fig. 12.6*** **a** Presenting films demonstrate varus deformity that was progressive since the time of his initial stabilization in near-anatomic alignment. Sagittal plane alignment appears good. **b** After establishing locking fixation in the proximal segment medially and laterally, compression and fine-tuning of alignment were achieved with medial and lateral Verbrugge clamps and push-pull screws distally. **c** Intraoperative plain films are taken on 17″ × 14″ cassettes to confirm coronal plane alignment with a larger field of view than offered by the fluoroscopy. **d** After 4 months, osseous union is confirmed radiographically. **e** Full-length weight-bearing view of the bilateral lower extremities confirms restoration of alignment

On rare occasions of substantial articular bone loss, or multiple failed attempts to get a small epiphyseal segment to heal to the metaphysis without success, more extreme measures may be employed. Arthrodesis has been discussed as an option, but there are no series in the literature detailing this approach for proximal tibial nonunion. In addition to not addressing the underlying problem of metaphyseal nonunion, arthrodesis comes with very significant functional limitations. This should only be considered as an option

of last resort.

Arthroplasty has also been employed with substantial articular loss or especially small epiphyseal segments. This mandates a stemmed tibial component to bypass the nonunited site. This is demonstrated in Fig. 12.7, where a very poor ORIF of a bicondylar plateau fracture led to a nonunited metaphysis and degenerative joint. Like arthrodesis, this does not necessarily address the underlying problem (nonunion) but rather bypasses it in exchange for a return to function and diminished discomfort.

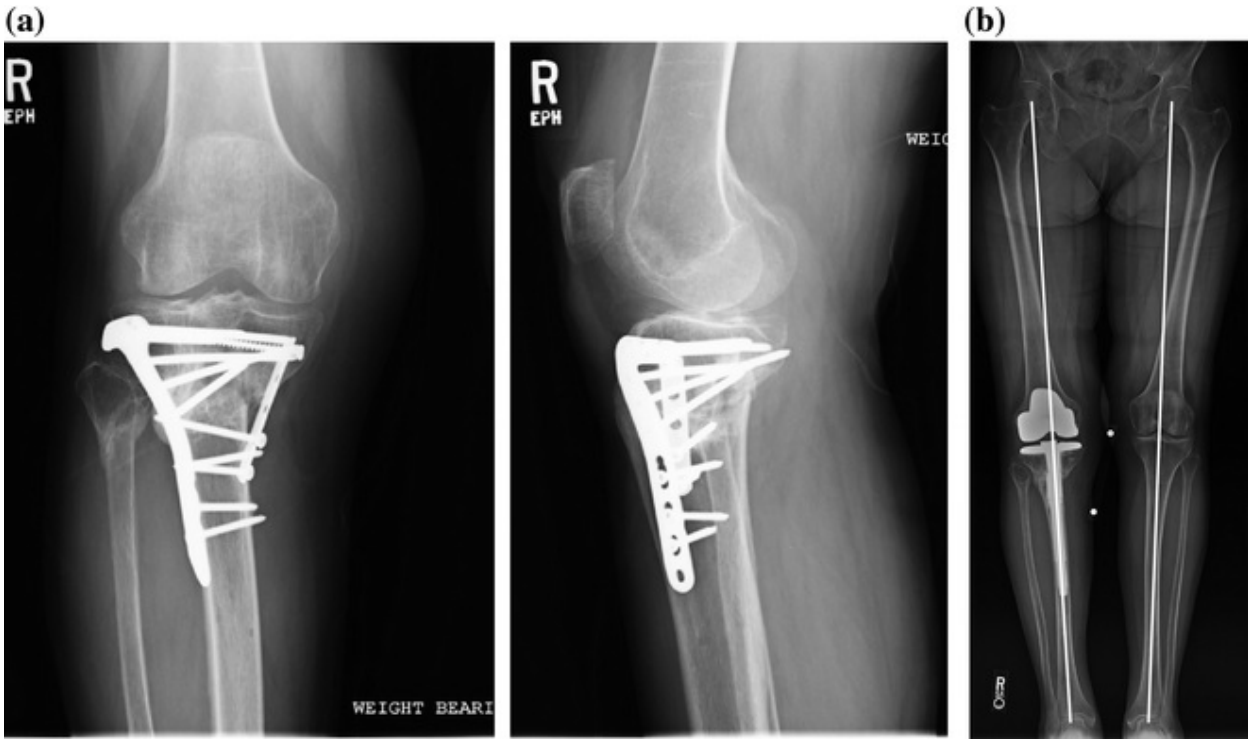



***Fig. 12.7*** **a** Presentation films one year after injury show varus malreduction with lateral joint arthrosis and metaphyseal nonunion. **b** Stemmed total knee arthroplasty is used to correct alignment and bypass nonunited metaphysis. (Case example and image courtesy of Christopher L. Peters, MD, University of Utah, Salt Lake City, Utah, USA)

## 12.4 Outcomes

There are scarce hard data on outcomes after repair of proximal tibial nonunions. Toro-Arbelaez et al. [26] reported a series of five intra-articular nonunions treated with correction of deformity, lag screws, and a buttress plate. Four of five returned to pre-injury function, but two of five required total knee arthroplasty after achievement of union. From the same institution,

Michael Gardner et al. reported sixteen extra-articular nonunions treated with deformity correction, bone grafting, lateral plating, and compression. All healed within four months, within five degrees of anatomical alignment [27]. Wu and colleagues reported a series of 28 bicondylar plateau fractures initially treated with lateral plating only that went onto either nonunion or malunion. They described open repair of deformity using an angled blade plate and compression with a minimum one-year follow-up on 25 patients. All had healed by four months, and 22 of 25 showed improved function after intervention [28].

---

## 12.5 Summary

Nonunion of the proximal tibia is relatively rare. The evaluation requires thorough assessment of deformity in all planes, evaluation for infection, and appraisal of metabolic causes of nonunion. There is little definitive outcomes data, but surgical intervention that includes correction of alignment, compression, and bone grafting will most often yield satisfactory functional results.

---

## 12.6 Case Discussions

**Case 1**

A very active 65-year-old physicist presenting one year after open treatment of bicondylar tibial plateau fracture .

**Case 2**

A 38-year-old female seven years removed from being struck by a bus. Her leg buckles when she attempts to walk. She has surprisingly good knee motion and no drainage. Laboratories were negative for infection or metabolic causes of nonunion.

**Case 3**

A 57-year-old female with significant COPD on corticosteroids failed closed treatment of this proximal tibial fracture in the setting of significant osteopenia.

**Case 4**

A 22-year-old male with proximal metaphyseal tibial nonunion after closed treatment. Metabolic workup revealed low vitamin D and low testosterone, which were treated. Compliance concerns led to selection of ring fixator and immediate weight bearing.

**Case 5**
A 28-year-old male with unstable proximal tibia metaphyseal fracture treated with lateral locked plate. Patient is referred at 4 months with progressive deformity and nonunion.

**Case 6** An active 82-year-old male with proximal tibial fracture treated with lateral locked plating, presented four months after index treatment with progressive deformity. Preoperative workup revealed vitamin D deficiency, which was treated.

**Case 7** An active 72-year-old female with bicondylar tibial plateau fracture treated initially with spanning external fixation and secondary ORIF. At one year, patient is referred with nonunion, varus deformity, and lateral compartment arthrosis.

---

# References


1. Choo KJ, Morshed S. Postoperative complications after repair of tibial plateau fractures. J Knee Surg. 2014;27(1):11–9.
[Crossref]

2. Papagelopoulos PJ, Partsinevelos AA, Themistocleous GS, Mavrogenis AF, Korres DS, Soucacos PN. Complications after tibia plateau fracture surgery. Injury. 2006;37(6):475–84.
[Crossref]

3. Borrelli J Jr, Prickett W, Song E, Becker D, Ricci W. Extraosseous blood supply of the tibia and the effects of different plating techniques: a human cadaveric study. J Orthop Trauma. 2002;16(10):691–5.
[Crossref]

4. Schatzker J, McBroom R, Bruce D. The tibial plateau fracture. The Toronto experience 1968–1975. Clin Orthop Relat Res. 1979;(138):94–104.

5. Moore TM, Patzakis MJ, Harvey JP. Tibial plateau fractures: definition, demographics, treatment rationale, and long-term results of closed traction management or operative reduction. J Orthop Trauma. 1987;1(2):97–119.
[Crossref]

6. King GJ, Schatzker J. Nonunion of a complex tibial plateau fracture. J Orthop Trauma. 1991;5(2):209–12.
[Crossref]

7. Barei DP, Nork SE, Mills WJ, Henley MB, Benirschke SK. Complications associated with internal fixation of high-energy bicondylar tibial plateau fractures utilizing a two-incision technique. J Orthop Trauma. 2004;18(10):649–57.
[Crossref]

8. Egol KA, Su E, Tejwani NC, Sims SH, Kummer FJ, Koval KJ. Treatment of complex tibial plateau fractures using the less invasive stabilization system plate: clinical experience and a laboratory comparison with double plating. J Trauma. 2004;57(2):340–6.
[Crossref]

9. Phisitkul P, McKinley TO, Nepola JV, Marsh JL. Complications of locking plate fixation in complex proximal tibia injuries. J Orthop Trauma. 2007;21(2):83–91.
[Crossref]

10. Rademakers MV, Kerkhoffs GM, Sierevelt IN, Raaymakers EL, Marti RK. Operative treatment of 109 tibial plateau fractures: five- to 27-year follow-up results. J Orthop Trauma. 2007;21(1):5–10.
[Crossref]

11. Ruffolo MR, Gettys FK, Montijo HE, Seymour RB, Karunakar MA. Complications of high-energy bicondylar tibial plateau fractures treated with dual plating through two incisions. J Orthop Trauma. 2015;29(2):85–90.
[Crossref]

12. Mikulak SA, Gold SM, Zinar DM. Small wire external fixation of high energy tibial plateau fractures. Clin Orthop Relat Res. 1998;356:230–8.
[Crossref]

13. Watson JT, Coufal C. Treatment of complex lateral plateau fractures using Ilizarov techniques. Clin Orthop Relat Res. 1998;353:97–106.
[Crossref]

14. Canadian Orthopaedic Trauma Society. Open reduction and internal fixation compared with circular fixator application for bicondylar tibial plateau fractures. Results of a multicenter, prospective randomized clinical trial. J Bone Joint Surg Am. 2006;88(12):2613–23.
[Crossref]

15. Weiner LS, Kelley M, Yang E, Steuer J, Watnick N, Evans M, Bergman M. The use of combination internal fixation and hybrid external fixation in severe proximal tibia fractures. J Orthop Trauma. 1995;9(3):244–50.
[Crossref]

16. Ahearn N, Oppy A, Halliday R, Rowett-Harris J, Morris SA, Chesser TJ, Livingstone JA. The outcome following fixation of bicondylar tibial plateau fractures. Bone Joint J. 2014;96-b(7):956–62.

17. Martinez A, Sarmiento A, Latta LL. Closed fractures of the proximal tibia treated with a functional

brace. Clin Orthop Relat Res. 2003;417:293–302.

18. Naik MA, Arora G, Tripathy SK, Sujir P, Rao SK. Clinical and radiological outcome of percutaneous plating in extra-articular proximal tibia fractures: a prospective study. Injury. 2013;44(8):1081–6.
[Crossref]

19. Ricci WM, Rudzki JR, Borrelli J Jr. Treatment of complex proximal tibia fractures with the less invasive skeletal stabilization system. J Orthop Trauma. 2004;18(8):521–7.
[Crossref]

20. Freedman EL, Johnson EE. Radiographic analysis of tibial fracture malalignment following intramedullary nailing. Clin Orthop Relat Res. 1995;315:25–33.

21. Krettek C, Stephan C, Schandelmaier P, Richter M, Pape HC, Miclau T. The use of Poller screws as blocking screws in stabilising tibial fractures treated with small diameter intramedullary nails. J Bone Joint Surg Br. 1999;81(6):963–8.
[Crossref]

22. Tornetta P 3rd, Collins E. Semiextended position of intramedullary nailing of the proximal tibia. Clin Orthop Relat Res. 1996;328:185–9.
[Crossref]

23. Lang GJ, Cohen BE, Bosse MJ, Kellam JF. Proximal third tibial shaft fractures. Should they be nailed? Clin Orthop Relat Res. 1995;315:64–74.

24. Kulkarni SG, Varshneya A, Kulkarni S, Kulkarni GS, Kulkarni MG, Kulkarni VS, Kulkarni RM. Intramedullary nailing supplemented with Poller screws for proximal tibial fractures. J Orthop Surg (Hong Kong). 2012;20(3):307–11.
[Crossref]

25. Kerkhoffs GM, Rademakers MV, Altena M, Marti RK. Combined intra-articular and varus opening wedge osteotomy for lateral depression and valgus malunion of the proximal part of the tibia. J Bone Joint Surg Am. 2008;90(6):1252–7.
[Crossref]

26. Toro-Arbelaez JB, Gardner MJ, Shindle MK, Cabas JM, Lorich DG, Helfet DL. Open reduction and internal fixation of intraarticular tibial plateau nonunions. Injury. 2007;38(3):378–83.
[Crossref]

27. Gardner MJ, Toro-Arbelaez JB, Hansen M, Boraiah S, Lorich DG, Helfet DL. Surgical treatment and outcomes of extraarticular proximal tibial nonunions. Arch Orthop Trauma Surg. 2008;128(8):833–9.
[Crossref]

28. Wu CC. Salvage of proximal tibial malunion or nonunion with the use of angled blade plate. Arch Orthop Trauma Surg. 2006;126(2):82–7.
[Crossref]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_13

# 13. Tibial Nonunions

Samuel E. Galle[1] and David P. Zamorano[1]

(1) Orthopedic Trauma Service, Orthopedic Trauma & Fracture Clinic, Saint Alphonsus—Boise Campus, 901 Curtis Road, Suite 501, Boise, ID 83706, USA

**David P. Zamorano**
**Email:** David.Zamorano@saintalphonsus.org

**Keywords** Tibia – Hypertrophic – Atrophic – Intramedullary nailing – Ilizarov – Masquelet – Bone morphogenic protein 7

## 13.1 Introduction

### 13.1.1 Tibial Shaft Fractures

Tibial shaft fractures are universal orthopedic injuries, as they are the most common long bone diaphyseal fracture [1]. According to the US National Center for Health Statistics, fractures of tibia, fibula, and ankle together have an annual incidence of 492,000 [2]. A significant portion of these injuries is attributed to road traffic injuries, falls, and various sporting activities, and the majority of these athletic injuries being attributed to soccer. Once sustained, tibia and fibula fractures result in 77,000 hospitalizations annually, accounting for 569,000 hospital days, 825,000 physician office visits, and considerable direct and indirect healthcare costs [3, 4]. These fractures are commonly treated with cast management, intramedullary fixation, plate osteosynthesis , or external fixation, depending on the initial presentation and severity of the fracture. Various authors have reported tibial fracture union in approximately 17 weeks time; nevertheless, some patients suffer from long-

term functional impairment regardless of union status [1].

## 13.1.2 Incidence of Nonunion

Nonunions are defined by the US Food and Drug Administration as the failure of fracture union by 9 months after injury or the lack of radiographic progress toward union over 3 consecutive months [5]. This definition is debated in the literature. Many authors, among them the senior author of this chapter, define nonunions simply as those fractures which have failed to progress to union from prior clinic visits or have little to no potential for further healing without additional treatment or intervention. Clinically, they present with persistent pain, motion, and residual swelling about the nonunion site. As tibial fractures are the most common long bone injury, tibial nonunions occur more frequently than any other long bone [5, 6]. This is probably due to high-energy mechanisms that are often associated with tibial fractures and the limited soft tissue envelope. Additionally, the tibia is prone to significant soft tissue injury as well as damage to the underlying vascular supply. The incidence of delayed unions and nonunions of the tibia has been reported as high as 48%. Heckman et al. reported an average incidence of 25% for all tibial delayed union and nonunions [7, 8].

Various authors have retrospectively reviewed tibial fracture cases for criteria that might define those most likely to progress to nonunion in the future. Fong et al. described fracture characteristics associated with tibial nonunions and found that fractures with less than 25% cortical contact were the most predictive of nonunion and subsequent re-operation. The authors also acknowledged that open fractures, presence of comminution, and oblique or segmental fractures were also associated with tibial nonunions as well [9]. Teraa et al. [10] found significantly more delayed unions and nonunions in segmental fractures than nonsegmental fractures of the tibia. Nicoll [11] analyzed treatments of 705 patients with tibial fractures and concluded that the most significant factors affecting healing were initial displacement, comminution, soft tissue wounds, infection, and distraction. In 2013, Yang et al. developed questionnaires applied to 56 patients with tibial fractures treated with intramedullary nailing and asked three fellowship-trained trauma surgeons to assess for likelihood of progression to nonunion at 3 months postoperatively. The authors found diagnostic accuracy of 74% with a sensitivity of 62% and specificity of 77%, indicating that nonunion prediction is improving based on the clinical criteria as described by Fong, Teraa, and

Nicoll but clearly this is an area of future clinical research to improve the accuracy of nonunion prediction [12].

### 13.1.3 Ramifications of Nonunion

Tibial nonunions can have a catastrophic effect on a patient. Limb length inequality, rotational and angular deformity, pain and instability can all result in the inability to work, placing further economic burdens on the patient [13, 14]. Brinker et al. retrospectively reviewed 243 tibial shaft nonunions in 237 consecutive patients and performed quality of life outcome measurements. The most commonly affected tibial segment was the distal third (49%), and infection was present in 18% of all cases. They noted that "the impact of tibial shaft fracture nonunion on physical health was comparable with the reported impact of end-stage hip arthrosis and worse than that of congestive heart failure" [15]. The authors concluded that tibial nonunions are devastating in nature and have a significant adverse effect on quality of life. In terms of economic cost, Anotonova et al. [16] reported on the economic burdens of tibial nonunions and found median total care cost for tibial nonunion patients almost doubled that of those who did not have a nonunion ($25,555.97 vs. $11,686.24, $P < 0.001$).

## 13.2 Causes of Nonunion

Tibial fractures that progress to nonunions can be grouped into one of two categories: (1) those that lack stability and (2) those that lack biology (Table 13.1). This is an important distinction as it guides the surgeon's treatment.

***Table 13.1*** Biological risks for nonunion

| |
|---|
| Open fracture |
| Age |
| Smoking |
| Alcohol |
| NSAIDs |
| Nutritional deficiency |
| Prior radiation |
| Endocrinopathies |
| |

## 13.2.1 Mechanical

Instability may cause excessive motion at the fracture site, which encourages stem cells to differentiate into fibroblasts, resulting in the formation of fibrous tissue formation and delayed union or nonunion. Factors that promote instability at the fracture site include bony comminution, inadequate or poor plate fixation, small diameter nails, poorly constructed external fixation constructs, and inadequate bony contact.

Comminuted tibial shaft fractures are typically treated with relative stability techniques including intramedullary nailing, bridge plating, and external fixation. When treating these fractures, it is important for the surgeon to choose the construct based on appropriate stiffness. This can be a difficult task and requires much experience with tibial nonunions and the fixation constructs available. In general, the use of larger diameter nails, stiffer plates, and multi-planer external fixation constructs can decrease the risk of nonunion in comminuted tibial shaft fractures.

Bony contact is also an important factor in providing stability to the fracture. As bony contact decreases, fracture stability will also decrease, predisposing to hypertrophic nonunion.

## 13.2.2 Biological

There are many factors that contribute to “biologic” nonunions. A common cause includes poor blood supply to the fracture often secondary to the soft tissue injury, surgical technique, or a combination of the two. Bishop et al. [17] described patient-related contributors of nonunions and included medical comorbidities, advancing age, smoking, alcohol abuse, nonsteroidal anti-inflammatories, nutritional deficiency, prior radiation treatment, genetic disorders, and various metabolic diseases.

### *13.2.2.1 Open Fracture*

The tibial shaft maintains a subcutaneous anatomical location for a substantial portion of its length especially along its medial border. Poor soft tissue coverage of the tibia has long been associated with a higher incidence of open fractures, which have a higher likelihood of nonunion. Rosenthal et al. retrospectively reviewed 104 open tibial fractures for the relationship

between initial wound presentation and potential for healing. Records were analyzed for some 71 patients: all Gustilo type I fractures united, two patients in type II continued to nonunion, and 13 patients in the type III fracture classification went on to nonunion. The authors concluded that there was a strong association between fractures that suffered nonunions and extensive soft tissue loss [18].

#### *13.2.2.2 Smoking*

While surgeons cannot always choose their patients in trauma, care should be taken in selecting patients for elective nonunion surgery. Cigarette smoking and nicotine have been implicated in inhibiting fracture healing and increasing the risk of delayed union or nonunion [19, 20]. The effects of smoking are related to its inhibitory effects on the formation of fibroblast-rich granulation tissue leading to impaired healing [21]. Smoke inhalation leads to low concentration of antioxidant vitamins and reactive oxygen species that cause cellular damage, particularly in osteoblasts, fibroblasts, and macrophages. Nicotine has been shown to increase platelet aggregation, to inhibit fibroblast function, and to decrease blood flow to extremities due to increased peripheral vasoconstriction [22].

A large number of studies document the effects of smoking and nicotine in various animal models. In the rabbit model, Donigan et al. studied the effects of transdermal nicotine on fracture healing in 22 mid-shaft tibial osteotomies treated with plate fixation. They noted that, although the nicotine-treated rabbits had similar areas of periosteal callus formation, these rabbits had significantly less torsional resistance and stiffness at 21 days postoperatively and three rabbits had gross nonunions [23]. Similar results reported by Raikin et al. [24] showed that nicotine-exposed rabbits had tibial healing that was 26% weaker resistant to three-point bending than those not exposed. In humans, the majority of research confirmed similar associations as that of the animal models; however, this has been mostly retrospective reviews rather than the understandably difficult prospective, randomized study. Castillo et al. as part of their prospective lower extremity assessment project of 268 tibial fracture patients revealed that current smokers had a higher incidence of nonunion at 24 months after injury compared to nonsmokers (24.1 vs. 9.9%, respectively). Smokers were also more than twice as likely to develop infection and 3.7 times as likely to develop osteomyelitis [25]. In a retrospective study, Adams et al. [26] compared 140

smoking and 133 nonsmoking patients. Mean time to union was 32 weeks compared to 28 weeks, respectively. Clearly, there is an association with smoking and delayed fracture healing, but further research is necessary to identify the exact molecular pathway and possible therapeutics to counteract its effects. Prior to any surgical intervention, smoking cessation should be emphasized to enhance the likelihood of healing. Urine and/or blood screening for nicotine and cotinine can be used to confirm patient's smoking status. Clinical experience has shown that blood levels of nicotine will return to normal within 2 weeks of cessation while urine will be positive for several weeks.

#### *13.2.2.3 Medications*

Nonsteroidal anti-inflammatory drugs (NSAIDs) are commonly cited in the literature as being associated with delayed unions and nonunions, while controversy remains regarding their effect on fracture repair [27]. The exact biochemical pathway is an area of further research, but many authors have theorized that these medications inhibit cyclooxygenase leading to less prostaglandin E2, which leads to less bone formation by osteoblasts [28, 29]. Zhang et al. [30] proposed a schematic model for cyclooxygenase-2, (COX-2) effects on bone repair after fracture using COX knock out mice, whereby decreased levels lead to decreased production of prostaglandin E2, which may lead to low levels of (bone morphogenic) protein. Simon and O'Connor expanded on this murine model and administered various doses of celecoxib, a selective COX-2 inhibitor, to explore the dose-dependent and time-dependent effects of this NSAID. The authors found impaired healing with increasing dosage radiographically, in torsional stability, and overall increased formation of nonunion [31]. Giannoudis et al. [32] retrospectively reviewed the effects of NSAIDs on femoral nonunions in 32 patients and noted a strong correlation. While this association has not been proven definitively in humans with a prospective randomized control trial, caution should be used when prescribing NSAIDs in the setting of tibial fractures, especially in those patients with impaired healing, e.g., smokers, diabetics, etc.

#### *13.2.2.4 Endocrinopathies*

Patients who present with a tibial nonunion without an obvious cause should

be worked up for an endocrinopathy. Vitamin D, vitamin C, calcium, thyroid hormone, and parathyroid hormone abnormalities have all been implicated in the formation of nonunions. Brinker et al. [33] analyzed 37 prescreened nonunion patients with the hypothesis that these idiopathic nonunions identified actually had underlying endocrine and metabolic abnormalities. They found that 83.8% of the 37 patients had some type of endocrinopathy with the most common being vitamin D deficiency. These authors proposed a diagnostic algorithm for identifying these patients for further workup by an endocrinologist as part of their study. Additional research may further elucidate the causal nature of various endocrinopathies and metabolic disorders and their relationship to nonunions, as well as potential medical treatments.

#### *13.2.2.5 Infection*

Infected tibial nonunions pose a complex clinical problem for surgeons and can lead to significant morbidity. In the setting of tibial fractures, infections are propagated from open wounds or introduced during surgical management. *Staphylococcus aureus* is the most commonly implicated organism and has been found in 65–70% of patients with long bone infections [34]. On the microscopic level, bacteria will form a biofilm or glycocalyx that significantly inhibits ability of the immune system to clear the infection. This leads to involucrum formation, which is reactive bone, as the body attempts to limit the spread of the infection. Shortly following is sequestrum formation, or necrotic bone, indicating a chronic infection with little ability to heal without intervention.

---

## 13.3 Evaluation and Diagnosis

### 13.3.1 History

Of the utmost importance in defining the scope of the problem is the history of the tibial fracture and prior treatment modalities that have failed to obtain fracture union. This includes mechanism of injury, prior open wounds, pain with weight bearing, feelings of instability, and any delayed wound healing. Patients who present with tibial nonunions have often had an extensive treatment history at multiple institutions. Previous records, including operative notes, injury and postoperative radiographs, and any pertinent

laboratory values, should be obtained from all previously treating physicians. Questions specific to infectious etiology are particularly important, covering wound drainage, prior cellulitis, constitutional symptoms, pertinent cultures/sensitivities, and previous antibiotic treatment regimens. A complete account of the patient's chronic illnesses is also important and will help to guide treatment algorithms. This should include the patient's nutritional status, smoking history, constitutional symptoms, and prior history of nonunion.

### 13.3.2 Physical Exam

The physical examination of all tibial nonunions begins with observation of the lower extremity for prior wounds, surgical incisions, erythema, gross deformity, and the general state of the surrounding soft tissue. Tenderness to palpation about the nonunion site should be noted and gross motion may be found as well. The surgeon should document the limb vascularity, limb lengths, and range of motion of the knee and ankle joints, as contractures may have occurred.

### 13.3.3 Laboratories

Important laboratory markers in the evaluation of tibial nonunions that can help guide the surgeon's treatment include erythrocyte sedimentation rate (ESR) , C-reactive protein (CRP) , and white blood cell (WBC) count . Unfortunately, many authors note that negative laboratory markers do not completely rule out indolent infection. In nonunions with reasonable stabilization, laboratory evaluation for metabolic and endocrine disorders should be obtained in consultation with an endocrinologist, as previously discussed. These markers include serum calcium, serum 25-hydroxy-vitamin D, thyroid-stimulating hormone, phosphorus, and alkaline phosphatase levels.

### 13.3.4 Radiographs

Radiographs on initial presentation should include the standard anteroposterior and lateral of the tibia/fibula to document the characteristics of the nonunion. Forty-degree internal and external oblique views and stress views may also be useful to better characterize the nonunion.

## 13.3.5 CT/MRI

Computed tomography (CT) and magnetic resonance imaging (MRI) of nonunions are important tools for defining the three dimensional extent of tibial pathology. CT scans can provide useful information regarding the number of cortices that have healed across a tibial fracture site with bridging callus formation. One study of CT scans for the presence of tibial nonunion found 100% sensitivity and 62% specificity [35]. MRI of tibial nonunions may delineate soft tissue infections from underlying osteomyelitis. Osteomyelitis appears as a low signal intensity of T1-weighted images and high signal intensity of T2-weighted sequences (Fig. 13.1a, b).

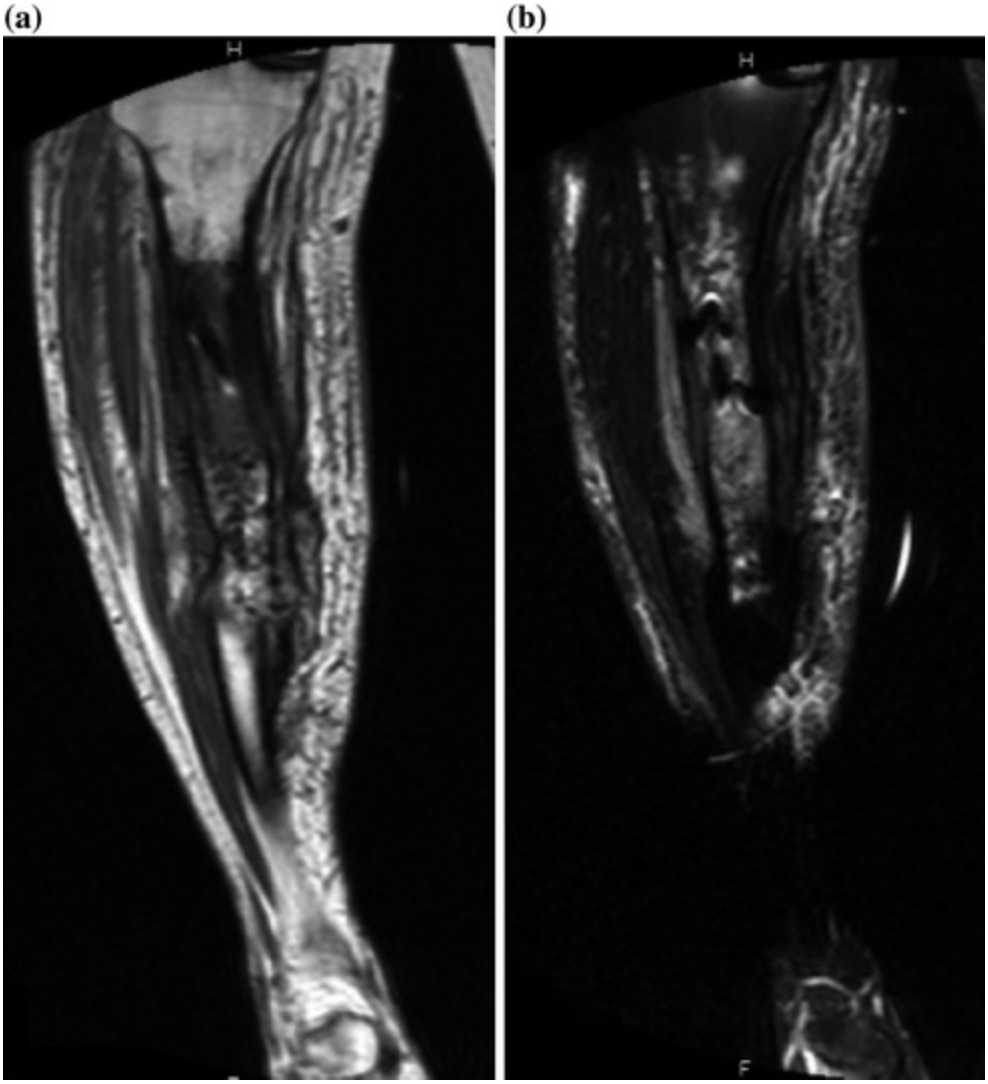



***Fig. 13.1*** Demonstration of osteomyelitis on magnetic resonance imaging . T1-weighted image are low signal and appear dark (**a**) while T2-weighted images show high signal (**b**). Images courtesy of

Animesh Agarwal, MD Animesh Agarwal, MD, Department of Orthopedics, University of Texas Health Science Center, San Antonio, Texas, USA

### 13.3.6 Nuclear Imaging

Various modalities are available and are used to evaluate infection as an etiology of the nonunion. The most commonly used nuclear medicine scans of nonunions include technetium-99 m, gallium-67 citrate, and indium-111-labeled leukocyte. Madsen recently reported a case report on the use of bone SPECT/CT imaging to detect sequestrum formation in a chronically infected tibial nonunion [36]. Further research is required to determine the clinical applicability of using SPECT/CT in the setting of tibial nonunions.

---

## 13.4 Treatment

The most important aspect of treating tibial nonunions is identifying and correcting the underlying cause of the nonunion. This may be a systemic issue, such as an endocrinopathy, or a localized pathology, for example an infection. Once the etiology of the nonunion has been addressed, the surgeon can continue his or her plan to repair the nonunion. Basic treatment modalities include improving fracture mechanics, restoring the local biology of the fracture, providing electrical or ultrasonic stimulation, and various combinations of these modalities. The surgeon should also consider factors such as the patient's functional level, occupation, and expectations when developing a treatment plan to ensure its eventual success for both the surgeon and the patient. Various treatment modalities will be discussed and the surgeon should select one based on their training, comfort level, and prior experience.

### 13.4.1 Based on Nonunion Type

#### *13.4.1.1 Hypertrophic*

In most cases of hypertrophic nonunion, fracture stabilization is the fundamental management concept and bone grafting is generally not necessary. Hypertrophic tibial nonunions present with callus formation about the fracture ends, leading to the very characteristic flared ends as the bone attempts to unite (Fig. 13.2). These nonunions tend to be well vascularized but are thought to lack the requisite mechanical stability for bone formation.

Once bony stability has been restored, motion at the fracture site is decreased and allows for capillary ingrowth with eventual enchondral ossification. Often this case presents as a tibial shaft that had been previously treated with an intramedullary nail. In such a case, exchange nailing with a larger nail can provide the necessary stability to promote bone healing. In cases where the nonunion had been treated nonoperatively or with an unstable external fixator, intramedullary nailing or compression plating is all that is required to obtain fracture healing. Figure 13.3 demonstrates a hypertrophic nonunion due to a lack of mechanical stability. A typical large callus has formed in the attempt to increase fracture stability. The nonunion was treated with exchange intramedullary nailing with a larger diameter nail. No bone grafting or other biological adjunct was used to achieve union.


***Fig. 13.2*** Hypertrophic nonunion . Broken distal interlock *(red arrow*) consistent with excessive motion at the fracture site. Note the abundant callus formation which is a hallmark of a hypertrophic nonunion


***Fig. 13.3*** 32-year-old male with a closed tibial shaft fracture (**a**). Closed intramedullary nailing with a 10 mm nail resulted in a hypertrophic nonunion (**b**). Exchange nailing was successfully performed using a 11.5 mm nail (**c**, **d)**

## *13.4.1.2 Atrophic*

Unlike hypertrophic nonunions, atrophic tibial nonunions present with poor callus formation, indicating little to no attempt at fracture healing (Figs. 13.4 and 13.5). Classically, these nonunions are thought to be poorly vascularized, but recent research has elucidated a more complicated understanding of atrophic nonunions. Matuszewski and Mehta recently described a case report of a 30-year-old patient who sustained a type IIIC tibial shaft fracture initially treated with vascular repair, soft tissue coverage and plating and yet unfortunately progressed to an aseptic, atrophic nonunion. The treating team

noticed pallor of the lower extremity and angiogram revealed stenosis of both the anastomosis sites. After angioplasty, the patient planned on further intervention but was delayed secondary to pregnancy. When she returned to clinic 5 months later, 15 months after initial treatment, the fracture site was radiographically healed without further intervention, implying the importance of vascular supply in the setting of atrophic nonunions [37]. Brownlow et al. analyzed 16 rabbits with atrophic nonunions at various time points to document the vascularity at the fracture site compared with controls. The authors found that at 1 week the control fracture sites were vascularized and the experimental fracture sites were nonvascularized, but this difference resolved by 8 and 16 weeks [38].


***Fig. 13.4*** Classic atrophic nonunion . Notice there is no evidence of bone healing. These types of nonunions typically require a biological stimulus to promote healing

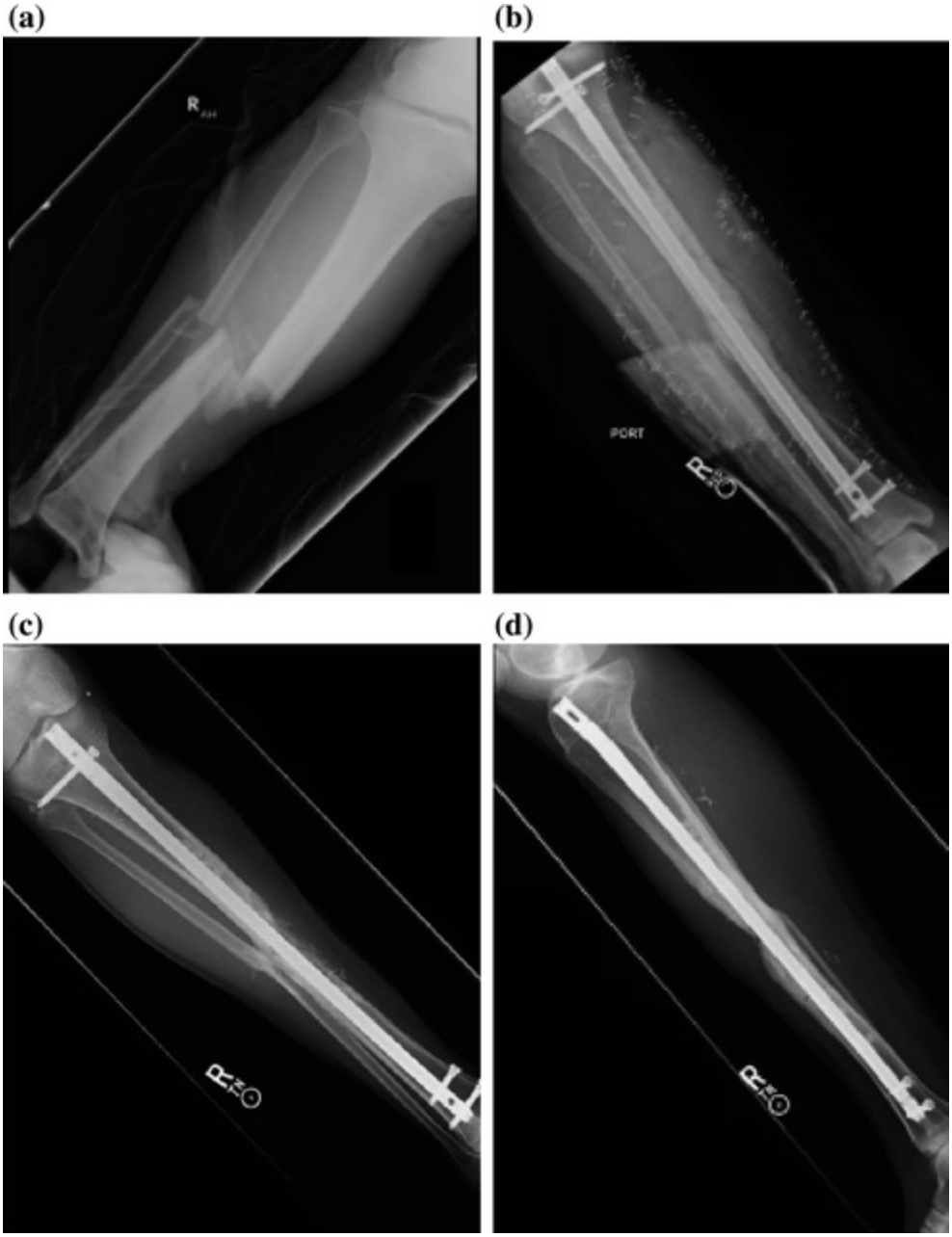



***Fig. 13.5*** 22-year-old male with a type IIIC open tibia (**a**). Free flap with intramedullary nailing and cement spacer (**b**) that was eventually bone grafted using a reamer-irrigator/aspirator technique. Nonunion eventually healed with small anteromedial defect (**c**, **d**)

Treatment goals should be focused on the underlying etiology of the nonunion, stimulating a healing response, and providing stable fixation if needed. Bone grafting with autogenous graft remains the gold standard but adjuncts such as bone morphogenic protein-2 and parathyroid hormone (PTH) can also be useful. Atrophic nonunions are most likely multifactorial

and present an area for further research.

### *13.4.1.3 Oligotrophic*

Oligotrophic nonunions are those that have characteristics of both atrophic and hypertrophic nonunions, as previously discussed. Management options follow those for hypertrophic nonunions as well as examination for possible causes of a biological lack of bony healing.

### *13.4.1.4 Infected*

Infected nonunions of the tibia can be a challenging problem. Multiple surgeries are usually required to get adequate debridement and eventually restore the function of the limb. Patzakis and Zalavras [39] summarized the basic tenets of care, which includes surgical debridement, antibiotics, fracture stabilization , adequate soft tissue coverage, and eventual restoration of bone defects.

Cierny et al. [40] described the basic classification schema of osteomyelitis based on anatomic types, e.g., medullary, superficial, localized, and diffuse, and patient characteristics based on underlying comorbidities (Fig. 13.6). Clinically, these patients present with ongoing pain, erythema, swelling, and possibly a draining sinus. Laboratory markers include ESR, CRP levels, and a WBC count may be elevated and can be used to diagnose and demonstrate clinical improvement after treatment. Computed tomography or MRI are more useful than plain radiographs in identifying affected areas, periosteal reactions, or abscess formations in the preoperative setting. Erdman et al. [41] demonstrated that MRI of patients suspected of having osteomyelitis had a sensitivity of 98% and a specificity of 75%.

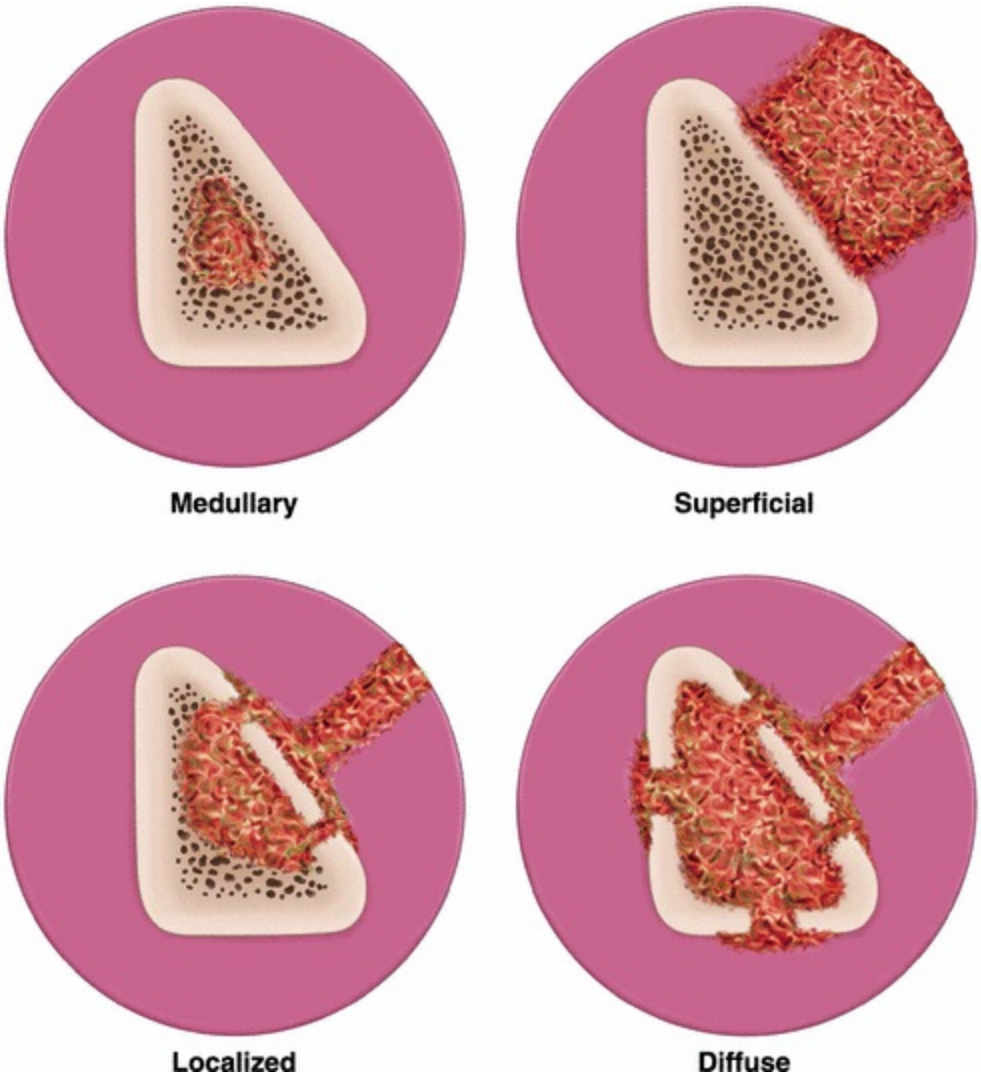



***Fig. 13.6*** Basic classification schema of osteomyelitis based on anatomic types. Modified from Cierny et al. [40], with permission

Intra-operative cultures from the sinus tract, purulent discharge, soft tissue, and curetted/debrided bone are imperative, as they can help determine a proper antibiotic regimen. Perry et al. [42] noted that superficial wound cultures and needle aspirations were insufficient to rule out infection perioperatively.

For the medullary and superficial cases of osteomyelitis (Cierny-Mader type I and II), general consensus treatment includes removal of metal implants and radical debridement of all involved bone and soft tissue. It is important that the surgeon does not sacrifice a thorough debridement for the hope of an easier reconstruction. While reconstructing large soft tissue and

bony defects can be difficult, an inadequate debridement will be doomed to failure. For the localized and diffuse cases (Cierny-Mader type III and IV), nonviable bone must be debrided fully and may require reconstruction at a later date. The complicated diffuse cases with extensive bony and soft tissue defects may result in amputation as the only viable treatment option, especially when presenting in patients with severe comorbidities. Amputation versus limb salvage is a clinical judgment based on patient comorbidities, soft tissue defects, bony involvement, neurovascular assessment, and the desires of the patient. In either case, a good support system for the patient is imperative for a successful outcome.

Fracture stability is of the upmost importance in treating infected tibial nonunions . The senior author was taught as a resident, and this still stands true that “an infected STABLE nonunion is better than an infected UNSTABLE nonunion.” Patzakis and Zalavras [39] have similarly recommended retaining the implants in infected nonunions in certain clinical situations, e.g., early diagnosis, known bacterial species, antibiotic sensitive species, etc. Implants that may be colonized or have failed should be replaced with either external fixation or intramedullary nailing; however, plate fixation may also be reasonable in certain settings. Megas et al. [43] treated nine patients with infected tibial nonunions and bone defects of 2–12 cm after intramedullary nailing with Ilizarov external fixation and reported a 100% union rate with a mean external fixation time of 187.4 days. Consideration should also be given to the placement of poly(methyl methacrylate) (PMMA) beads impregnated with heat-stable antibiotics such as tobramycin and vancomycin. Holtom and Patzakis [44] recommended approximately 2.4–4.8 g of tobramycin, or vancomycin, 2–4 g, per 40 g of PMMA cement to achieve local bactericidal conditions.

Adequate soft tissue coverage should also be obtained during the wound debridement of infected tibial nonunions. This is usually accomplished with a rotational muscle flap or a free muscle transfer, depending on the integrity of the local tissues and the size and location of the soft tissue defect. Muscle transfers are particularly useful for providing a new influx of vascular supply, which improves antibiotic dispersal and host immune system, preventing further microbial seeding. Figure 13.7 demonstrates the usefulness of muscle transfers to help eradicate osteomyelitis and heal a nonunion. A thorough debridement of the infected area is vital to a good outcome. Once the surgical debridement is complete, an antibiotic cement depot is inserted and the

patient receives intravenous antibiotics tailored to the specific bacteria that is cultured. After 6 weeks of antibiotics, the flap is elevated, the cement spacer is removed, and autogenous bone graft is placed in the defect.

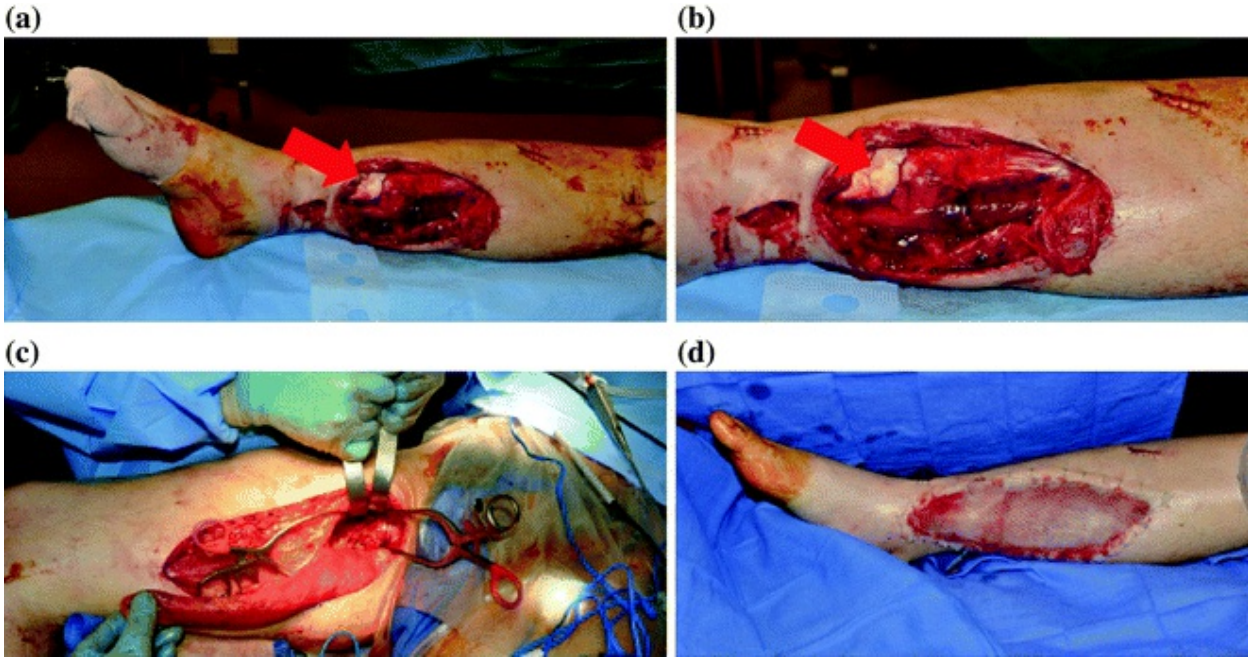



***Fig. 13.7*** 28-year-old female with a Gustilo-type IIIB open tibia and a segmental bone defect. Intramedullary nailing was performed with insertion of an antibiotic impregnated cement spacer (*red arrow*, **a**, **b**). Harvesting of a free gracilis muscle (**c**). Final coverage with split thickness skin graft (**d**). The patient would later go on to have removal of the spacer with autogenous bone grafting 6 weeks later. (Images courtesy of Garrett A. Wirth, MD, University of California–Irvine, Orange, California, USA)

## *13.4.1.5 Nonunion Location*

Peri-articular nonunions are a relatively uncommon occurrence but they can be difficult clinical problems to treat. Metaphyseal bone of the peri-articular region is well vascularized which provides the basic components for fracture healing. Nevertheless, nonunions do occur in this area and the soft metaphyseal bone may not provide the best implant fixation. Treatment regimens include fixed-angle plating, external fixation, and at times intramedullary nailing. Harvey et al. [45] reported on 17 proximal and 13 distal tibial nonunions using customized blade plate fixation, with 29 unions and five persistent nonunions after initial blade plate fixation. These authors found that blade plate fixation was able to achieve eventual union in 97.2% of peri-articular fractures. Gardner et al. performed a retrospective clinical

study on 16 patients with proximal tibial nonunions treated with deformity correction, bone grafting, and lateral plating. The authors found that all nonunions healed at an average of 4 months, Knee Society function scores improved significantly, and 88% were able to return to their prior activities [46]. Reed and Mormino reviewed functional outcomes after distal tibial metaphyseal nonunion fixation with blade plates. The authors found all 11 patients had healed and AOFAS scores improved from average scores of 29–89. Pain scores also improved from an average preoperative score of 14–36 postoperatively [47]. Alternatively, Richmond et al. [48] reported on 32 patients with distal tibial nonunions treated with intramedullary nailing and noted 91% union rate at an average of 3.5 months as long as there was enough room distally for two interlocking screws.

#### *13.4.1.6 Segmental Defects*

Segmental tibial nonunions are a clinically challenging problem to manage, as the body's natural ability to fill in bony defects is fairly limited [49]. Surgical options include acute shortening with possible future bone lengthening, autologous cancellous bone grafting, vascularized fibula cortical bone graft, and bone transport with an Ilizarov frame or over an intramedullary nail. Although there is no formal consensus on treating segmental tibial nonunions, many surgeons approach them with treatment guided by the size of the defect. Bone loss of less than 2 cm can be effectively treated with autologous bone graft ing. Defects between 2 and 6 cm may be treated with large autologous bone grafting, such as the Masquelet technique, or bone transport. The Masquelet technique involves a two-stage procedure starting with radical debridement and cement spacer placement, which induces an osteoinductive membrane, followed by autologous bone grafting after removal of the cement spacer [50]. This technique can be quite powerful. Our institution has had success with defects up to 9 cm in the tibia and even larger in the femur. Bone defects larger than 6 cm are often treated with bone transport, free vascularized fibular transfer or amputation. The Ilizarov bone transport technique is a useful tool and allows for bifocal or trifocal correction of large segmental defects. Sala et al. [51] reviewed results from 12 patients with post-infectious segmental tibial nonunions treated with Ilizarov bone transport (Taylor Spatial Frame) in a bifocal or trifocal method and noted 100% union rate in an average external fixation time of 418 days (range 300–600 days).

Stafford et al. [49] retrospectively reviewed 19 segmental tibial nonunions treated with the reamer-irrigator/aspirator (RIA) system (Synthes, Paoli, PA, USA) and a two-stage Masquelet technique for bone defects from 1 to 25 cm in length. At the final clinical follow-up at approximately 1 year postoperatively, 17 of the nonunions had achieved clinical union. Kundu et al. reported results of the Huntington's procedure, a tibialization of the fibula, for bone defects over 6 cm in size in 22 patients. The authors described clinical unions in 21 of the 22 patients with full unprotected weight bearing at an average of 16 months [52]. Figure 13.8 demonstrates the Masquelet technique in a 38-year-old female who presented from an outside institution 6 months out with a draining sinus over her anterior tibia. She underwent a staged procedure with irrigation and removal of all dead and infected bone, placement of an antibiotic spacer, and a free gracilis muscle transfer. She was placed on intravenous antibiotics for 6 weeks. After a 2 week antibiotic holiday, infectious laboratory markers were drawn and were normal. She was taken back to the operating room for removal of the antibiotic spacer and autologous bone grafting using RIA on her ipsalateral femur. A robust pseudomembrane was generated and preserved (see Fig. 13.8d).

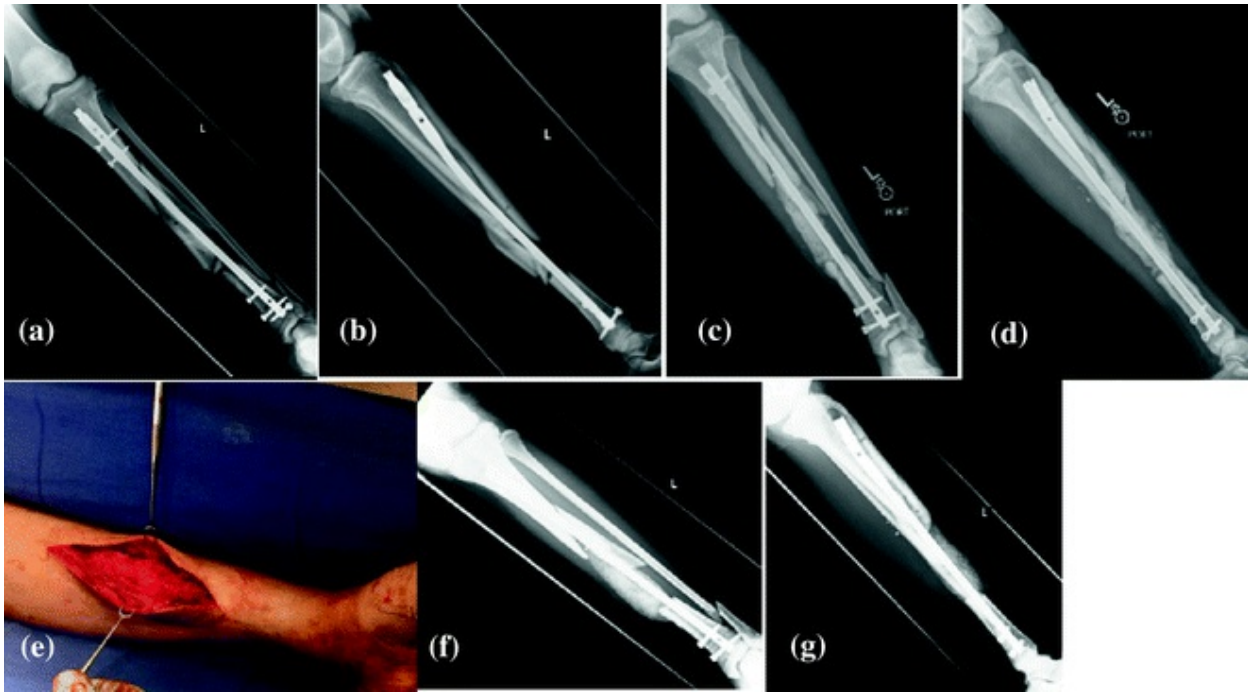



***Fig. 13.8*** 38-year-old female presented with a infected nonunion (**a**, **b**). Underwent debridement and placement of antibiotic cement spacer (**c**, **d**). Reamer-irrigator/aspirator performed at 8 weeks from ipsalateral femur. Bone grafting of the tibial defect (**e**). AP and lateral radiographs of tibia at 2 weeks

show position of graft (**f**, **g**)

### *13.4.1.7 Prior Failed Treatment*

Recalcitrant nonunions of the tibia are those that have undergone multiple operations and fail to achieve union. The etiology of these resistant nonunions may be multifactorial and are more difficult to treat. The soft tissue envelope and, therefore, the blood supply, of recalcitrant nonunions are often tenuous due to the initial trauma, chronic inflammation, and multiple surgical procedures. When treating these nonunions, it is important to consider the type of procedure previously performed, as there is little value of repeating a properly done surgery that has already failed. Instead, the surgeon must consider possibly using a combination of different modalities to achieve successful union. Desai et al. [53] reported on nine recalcitrant tibial nonunions that had previously undergone at least four operations and treated them with the RIA bone graft, human recombinant bone morphogenic protein 2 (BMP-2), and intramedullary nailing. The mean time to union, which included clinical and radiographic parameters, averaged 27.6 weeks (range 11–59), and all patients eventually healed.

## 13.4.2 Methods of Treatment

Tibial nonunions have a variety of nonsurgical and surgical treatment modalities. Regarding nonsurgical options, surgeons may pursue observation, functional bracing , external electrical or ultrasonic stimulation, or extracorporeal shock wave therapy [54]. Surgical management includes fibular osteotomy, compression plating, intramedullary nailing, external fixation, bone grafting, and distraction osteogenesis.

### *13.4.2.1 Functional Bracing*

In a small subset of tibial nonunions, the lack of clinical healing may be augmented by a weight-bearing cast or brace. Sarmiento and Latta suggested that weight-bearing causes a change in the fracture site biology that stimulates callus formation [55]. This is especially important with regard to patients who have undergone a nonweight-bearing period to maintain reduction but were then referred when the fracture did not heal promptly. Functional bracing is indicated in the setting of stable fracture patterns, acceptable alignment, and a compliant patient. Sarmiento et al. also reported

on 67 delayed and nonunited tibial fractures that were treated with functional bracing alone in nine patients, with fibular ostectomy in 48 patients, and with fibular ostectomy with bone grafting in ten patients. Of the 67, 91% went on to union at an average of 3–4 months. In the group treated exclusively with the functional brace, only one of nine failed to heal [56]. Overall this method tends to harbor low risk for the patient and allows for future treatment options should they be necessary.

### *13.4.2.2 Mechanical*

**Compression Plating**. Achieving compression across the nonunion site is paramount for proper healing, and the most basic tool to achieve it is the compression plate. Compression plating of tibial fractures continues to be a widely used technique for treating tibial nonunions, as it allows for immediate correction of any deformity, debridement of the nonunion, placement of bone graft, and direct compression. Compression with a plate can be achieved in many ways, including using an articulated tensioning device, compressing with a verbrugge clamp against a screw placed outside the plate, and even a two-screw technique with a farbeuf clamp. Caution should be used when relying on the plate compression holes to achieve compression as they do not generally achieve enough compression for nonunion repair. Bone grafting is also possible to augment this technique and the direct exposure to the nonunion site allows for simultaneous formal debridement. Postoperatively, the patient is able to begin early mobilization and rehabilitation. Figure 13.9 shows an atrophic nonunion in a patient with a history of neurofibromatosis. Direct compression plating and bone grafting were performed. As with all open techniques, plate osteosynthesis presents increased risk of wound complications, which is of particular importance in patients who have had prior surgeries and those with prior insult to the surrounding soft tissue. Piriou et al. [57] reported on 18 patients with tibial nonunions treated with decortication and medial plating and achieved approximately 94% union rate at an average of 108 days. Helfet et al. [58] presented 33 patients with tibial nonunions treated with tension-band plating, augmented with lag screw fixation and autogenous bone grafting, and reported 100% healing rate at an average of 4 months postoperatively. Lastly, Wiss et al. [59] found a 92% healing rate at 7 months with compression plating augmented by bone grafting on his 50 patients. Compression plating remains a valuable tool in the armamentarium of any orthopedic surgeon

when treating tibial nonunions.

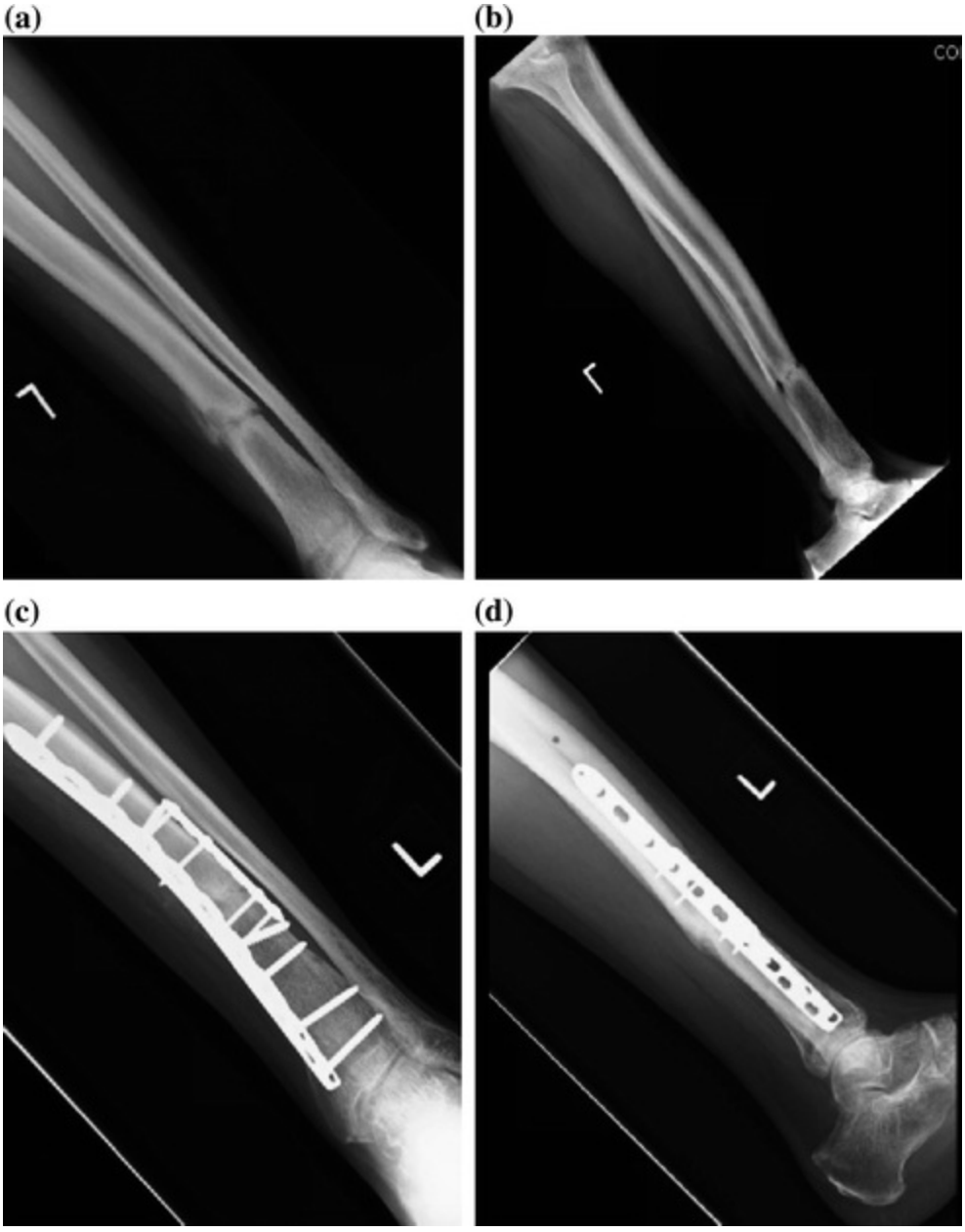



***Fig. 13.9*** 41-year-old male with a history of neurofibromatosis and a 1 year history of aseptic nonunion (**a**, **b**). Underwent compression plating and bone grafting with bone morphogenic protein (BMP)-2. Eventually healed 2 months after surgery (**c**, **d**)

**Intramedullary Nailing.** Intramedullary nailing of tibial nonunions provides an ideal combination of augmenting mechanical stability, dispersing of growth factors, and allows early active rehabilitation. Indications for this procedure continue to broaden and are similar to the indications for acute

fracture treatment, including prior closed and open diaphyseal injuries, segmental nonunions, and nonunions with significant bone loss.

With exchange nailing, reaming the medullary canal can promote angiogenesis and allow for a larger, more rigid, nail to be placed. This is especially important in hypertrophic nonunions where mechanical stability is lacking. Johnson and Marder discussed 20 patients, of whom 11 had hypertrophic nonunion s, treated with intramedullary nailing and found a healing rate of 100% with callus formation at an average of 11 weeks in the hypertrophic patients alone [60].

Compression with a nail can be achieved with “back slapping” or reverse impaction, utilizing the compression screw in many nail designs and by early weight bearing depending on the amount of cortical contact in the repaired nonunion. Clinical union rates range from 76 to 100% [61–63]. Zelle et al. retrospectively reviewed the results from 38 aseptic nonunions after initial unreamed nailing that underwent exchange reamed intramedullary nailing. The clinical union rate, as defined by painless weight bearing and bridging callus on three cortices, was 95% at an average time of 29 weeks with only three complications noted, one deep vein thrombosis and two hardware failures [64].

**External Fixation**. External fixation in the setting of tibial nonunions is a useful tool and is used primarily to manage infections and deformity in patients with a poor soft tissue envelope. However, external fixation can be used for the same indications as intramedullary nailing and compression plating as well. Ring fixators also have the advantage of immediate postoperative weight bearing. Harshwal et al. reported on 30 cases of tibial nonunions treated with mono-lateral external fixation and either corticotomy/bone transport for cases with >3 cm of shortening or compression/distraction for cases with <3 cm of shortening. These authors reported at an average of 8 months follow-up approximately 96.7% of cases achieved union in the tibial group alone [65]. Menon et al. [66] used circular fixators to achieve compression in nine tibial nonunion patients and retrospectively found 100% union rate at an average of 6.2 months. García-Cimbrelo and Martí-González [67] presented 82 patients treated with circular external fixators for patients with tibial nonunions with and without bone loss and noted an overall 93% healing rate. External fixation remains a powerful technique for the correction of tibial nonunions with many different characteristics, e.g., infected, bone loss, angular deformity, etc. Figure 13.10

demonstrates the usefulness of a ringed external fixator for management of tibial nonunions. The patient had an infected nonunion with a free muscle transfer.

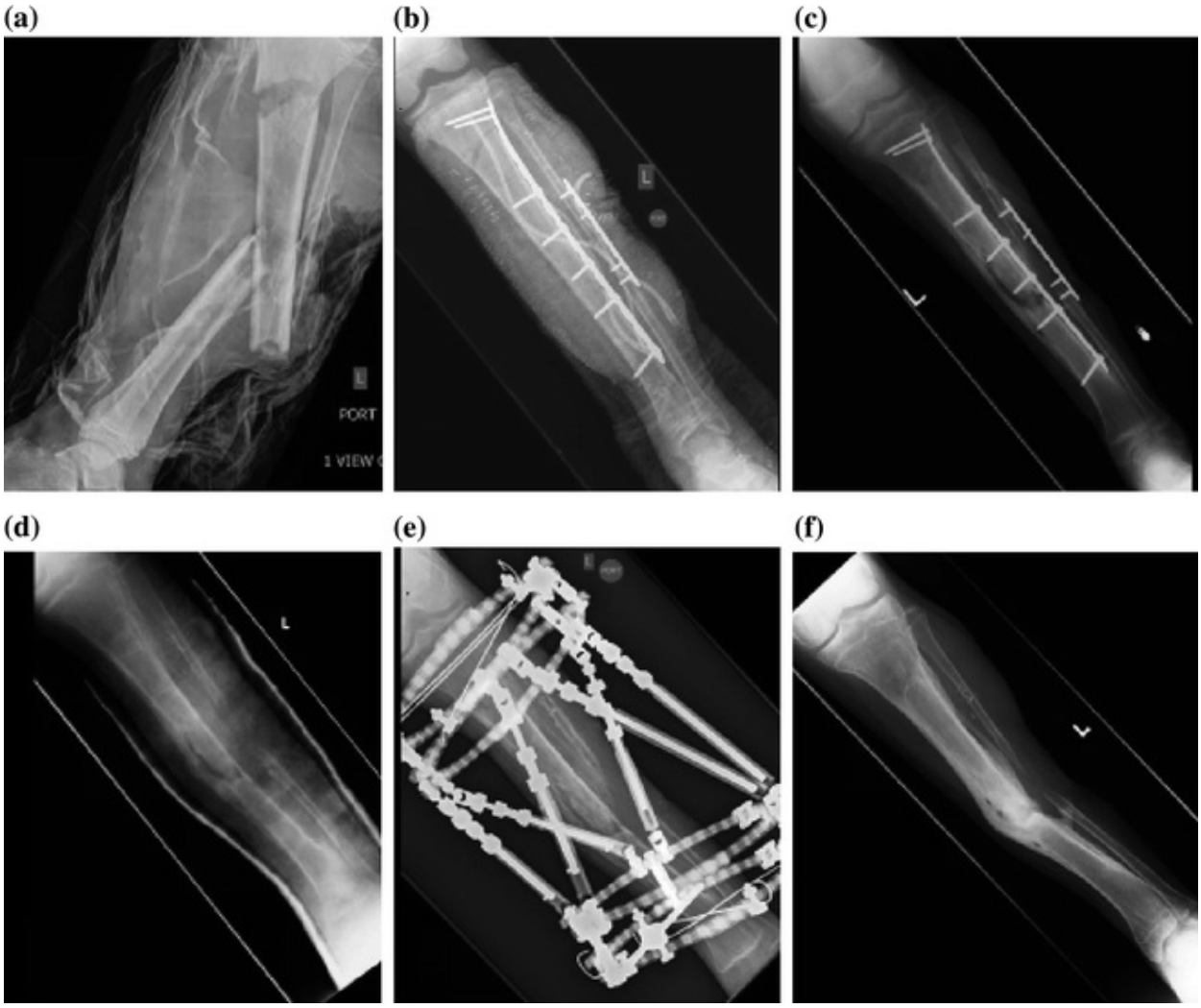



***Fig. 13.10*** 12-year-old male with a type IIIB open tibia fracture (**a**). Initially treated with a free muscle transfer and open reduction internal fixation (**b**). Developed a deep infection (**c**), which was debrided, and the implants were removed **(d)**. A ring external fixator was eventually placed (**e**) and the patient went on to heal his infected nonunion (**f**)

**Fibular Osteotomy**. Historically, fibular osteotomy has been used to increase the load transmitted through the tibial fracture site. The theory is based on the intact or healed fibula acting as a distracting strut that prevents tibia fracture apposition. The procedure is oftentimes used in conjunction with compression plating, exchange nailing, or external fixation in order to allow the force of weight bearing to be transmitted preferentially through the

tibia.

### 13.4.2.3 Biological

**Bone Grafting**. Iliac crest autograft is considered the gold standard for bone grafting of tibial nonunions across a variety of locations given its osteoconductive, osteoinductive, and osteogenic properties. The RIA has made possible the harvesting of large quantities of cancellous bone from the endosteal surface of the femur. This technique is appropriate for nonunions with up to 6 cm of bone loss and has a reported success rate of 88–95%.

Surgical approaches to the nonunion site include posterolateral or anterolateral, both of which provide adequate soft tissue coverage for graft incorporation. Takemoto et al. [49] performed analysis of different bone graft sites, including iliac crest, proximal humerus, and proximal tibia, for various BMPs and their receptors and found no significant difference at any of the three sites for all variables [68].

**Bone Graft Substitutes**. Bone graft substitutes are widely available today and are osteoconductive and osteoinductive in nature. They are designed to avoid harvest site morbidity; however, they are relatively expensive and have not been proven as clinically effective as cancellous bone grafting. Demineralized bone matrix (DBM) consists of allograft derived bone graft with osteoconductive properties. While DBM may hold osteoconductive properties by providing a scaffold for bony ingrowth, its osteoinductive characteristics have been inconsistent. DBM is available as putties, pastes, gels, and granules from a variety of different companies and is best used as a graft extender in conjunction with autograft bone. Ceramic bone substitutes include calcium sulfate and calcium phosphate, which similarly have osteoconductive properties. Both DBM and ceramics have been used to fill defects after nonunion formation. McKee et al. utilized medical grade alpha-hemihydrate calcium sulfate impregnated with tobramycin (Osteoset-T, Wright Medical Technology, Arlington, TN, USA) pellets to address voids in 25 patients with femoral, tibial, ulna, or humeral osteomyelitis. Each patient was treated with the pellets in addition to external and/or internal fixation, and the authors achieved 92% rate of infection eradication. By 6 months, postoperatively all pellets were noted to be resorbed and 71% of those patients treated with bone substitute without autograft achieved union [69].

**Growth Factors**. Bone morphogenic proteins, specifically, recombinant

human BMP-2 and BMP-7, have been utilized to augment tibial healing in a variety of studies. rhBMP-7, also known as osteogenic protein-1, has been studied specifically for tibial nonunions and has been approved by the US Food and Drug Administration solely under the “humanitarian device exemption” [70]. Friedlaender et al. [71] reported the results of a prospective clinical trial of tibial nonunions treated with intramedullary nailing with autologous bone graft or BMP-7 and found similar rates of union at 9 and 24 months. The authors concluded that BMP-7 was at least as effective as autologous bone graf ts in the setting of tibial nonunions treated with intramedullary nailing. Calori et al. performed retrospective cost analysis of 54 patients treated with BMP-7 or autologous bone grafting and noted clinical union in 89.3 and 76.9%, respectively, $P = 0.22$. The authors noted significantly higher costs associated with BMP-7 treatment and total costs incurred during BMP-7 usage [72]. Dehabreh et al. performed economic analysis of nonunion treatment across various anatomic locations prior to and after application of BMP-7 to augment fracture healing. The authors concluded that BMP-7 resulted in a 47% reduction in costs compared to persistent fracture nonunions treated otherwise [73]. BMP is not routinely used as a first-line treatment for nonunions at our institution; currently, BMP is reserved for recalcitrant nonunions that have failed previous attempts at union and for certain patients who are not candidates for autogenous bone grafting. Further research is necessary to identify the patient population that will benefit the most from this costly adjunctive procedure.

#### *13.4.2.4 Adjunct Treatments*

**Distraction Osteogenesis**. Modern distraction osteogenesis is based on the principles set forth by Gavriil Ilizarov. Ilizarov is credited for inventing a circular-type external fixator that allowed correction of deformity with six degrees of freedom [74]. His external fixation device or Ilizarov frame became the basis for bone regeneration from tension stress applied to a corticotomy site. With distraction osteogenesis, nonunions undergo increased perfusion, inflammatory response, and gain the ability to ward off infection (Fig. 13.11). Ilizarov argued treating infected nonunions with his method as “infection burns in the fire of regeneration.” Ring et al. retrospectively reviewed 27 patients comparing autologous bone grafting with the aforementioned Ilizarov technique. All patients in the bone grafting group went on to heal and nine of the ten patients treated with the Ilizarov technique

went on to clinical healing. The authors concluded that the Ilizarov technique was best suited for proximal or distal metaphyseal nonunions, and for those patients with large bone defects [75]. Dendrinos et al. reviewed 28 cases of infected tibial nonunions treated with distraction osteogenesis at a mean of 16 months after the original injury. All patients were cured of the infection. As to results achieved, 14 were considered excellent, eight good, one fair, and five poor [76].

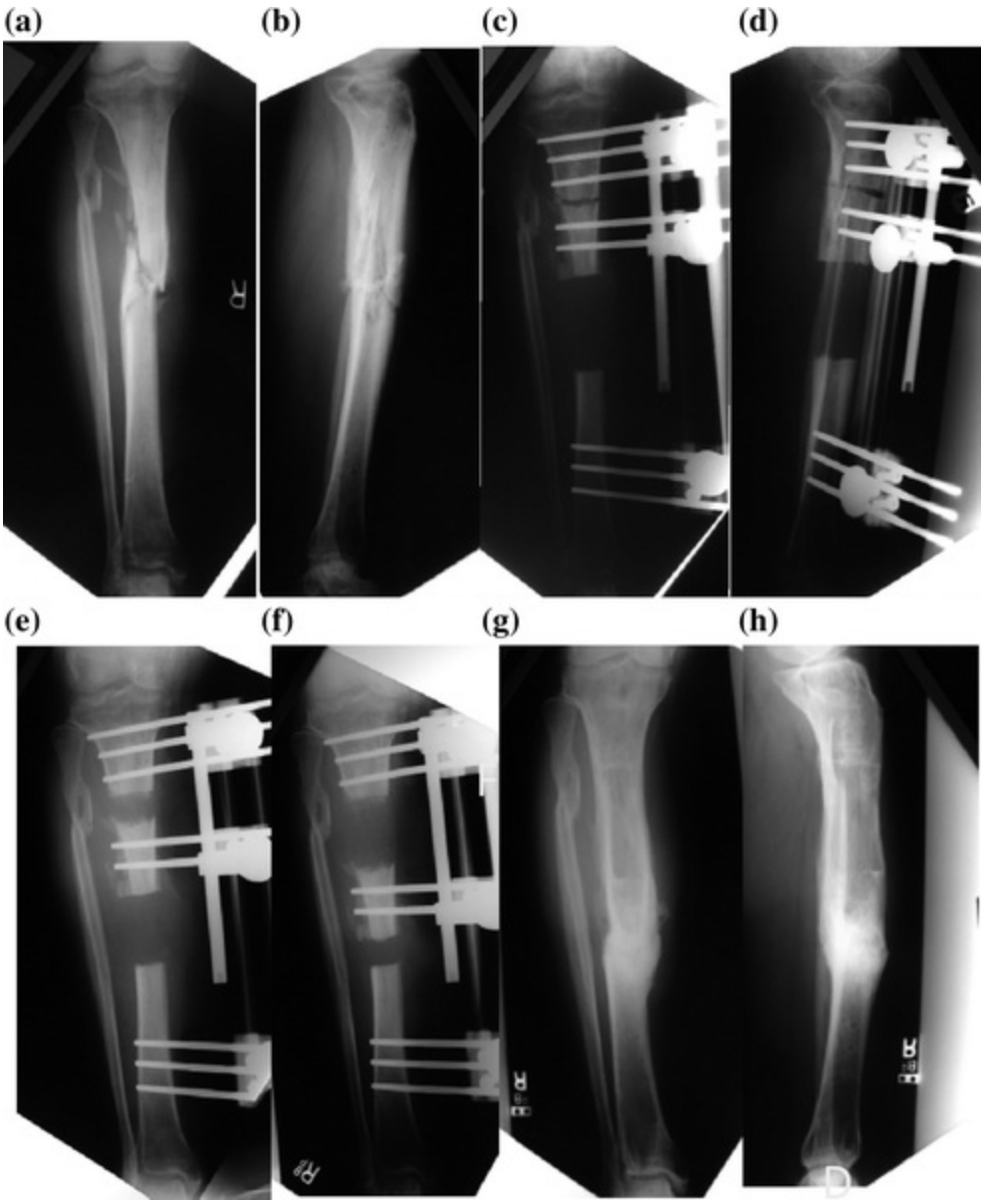



***Fig. 13.11*** 33-year-old-male status post falls from horse. Patient suffered a Gustilo-type IIIB open fracture. Presented with an infected nonunion (**a**, **b**). Debridement and a monolateral external fixator wa placed along with proximal corticotomy (**c**, **d**). Distraction osteogenesis was utilized (**e**, **f**) until final

healing (**g**, **h**). Images courtesy of Animesh Agarwal, MD Animesh Agarwal, MD, Department of Orthopedics, University of Texas Health Science Center, San Antonio, Texas, USA

**Structural Grafts** . In the treatment of tibial nonunions, the Huntington procedure is well-described technique for tibial defects greater than 12 cm. The first successful such procedure performed by Huntington in 1903 described ipsilateral transfer of the fibula to the tibia in a young boy [77]. Kassab et al. retrospectively reviewed the results of 11 tibial nonunions treated with the Huntington procedure and observed healing in eight of 11 patients at an average of 10.5 months. Of the three persistent nonunions, one patient had a 22 cm defect after tumor removal and the other two developed infections that led to subsequent amputations [78].

## *13.4.2.5 Electrical*

Electrical techniques include a variety of applications from invasive to noninvasive that all provide electromagnetic field stimulation to induce fracture healing. The exact mechanism of augmenting bone healing is not completely understood but proposed theories include increased mineralization and angiogenesis, increased DNA synthesis, and changes in osteoblast intracellular calcium levels [79]. These electrical treatments are applied with concurrent bony stabilization in the form of splinting or external fixation. Electromagnetic stimulation may be considered especially useful in those patients who are poor surgical candidates secondary to medical comorbidities or local soft tissue pathology that may not heal the postsurgical incision. Anglen [80] noted that electromagnetic techniques are not effective and contraindicated in the setting of fracture gaps greater than half the diameter of bone, synovial pseudarthrosis and unacceptable deformity.

Prospective randomized clinical trials demonstrating efficacy of these modalities is limited despite a body of literature supporting the technique in vitro. A comprehensive review performed by Griffen et al. [81] as part of the Cochrane Library concluded that the current body of research is inconclusive and insufficient to determine clinical practice. Additionally, there is no ability to correct rotation, angulation, or limb-shortening deformities.

## *13.4.2.6 Ultrasound*

Ultrasound techniques use high-frequency sound waves and include low-intensity pulsed ultrasound (LIPUS) , high-intensity focused ultrasound

(HIFUS) and extracorporeal shockwave therapies (ECSW) . All methods rely on bone growth in response to mechanical forces across the fracture site to encourage fracture union.

LIPUS generates high-frequency sound waves that induce mechanical pressure on bone to augment the healing process. These ultrasonic waves have been shown in animal models to increase callus formation and accelerate bone healing. Randomized control studies have been performed in acute fractures of the tibia, but they are lacking in the setting of established nonunions [82]. Of those cohort studies performed without controls, LIPUS union rate ranged from 81 to 100% with the caveat that some tibial nonunions may spontaneously heal without any intervention [83–89]. Given the current lack of good quality randomized studies, Watanabe et al. [89] concluded that while there are many potential benefits of LIPUS, including lack of harmful effects, ease of use, and possible use in compromised patients, further research is necessary to fully understand the indications and benefits of LIPUS.

Extracorporeal shock wave therapy is thought to increase bone mass, augment angiogenesis, and enhance mesenchymal stem cells differentiation to osteogenic cells lines. Elster et al. retrospectively reviewed 192 tibial nonunions treated with ECSW therapy and found 80.2% healing rate in an average of 4.8 months. The authors concluded that ESCW therapy was indeed a safe and reliable treatment modality for tibial nonunions to be used in conjunction with fracture immobilization and stabilization [90]. Further prospective research into the effects of ECSW therapy is necessary to make this adjunctive treatment option part of the standard of care for tibial nonunion management.

---

# References


1. Trafton PG. Tibial shaft fractures. In: Browner BD, Jupiter JB, Levine AM, Trafton PG, editors. Skeletal trauma: basic science, management, and reconstruction. 4th ed. Philadelphia: WB Saunders; 2009.

2. Praemer A, Furner S, Rice DP. Musculoskeletal conditions in the United States. Park Ridge, IL: American Academy of Orthopedic Surgeons; 1992.

3. AHRQ (Agency for Health Care Research and Quality). Introduction to the healthcare cost and utilization project (HCUP) state inpatient databases (SID). http://www.hcup-us.ahrq.gov/db/state/siddist/Introduction_to_SID.pdf. Accessed 22 April 2016.

4. Schmidt AH, Finkemeier CG, Tornetta P 3rd. Treatment of closed tibial fractures. Instr Course Lect. 2003;52:607–22.
[PubMed]

5. Brinker MR, O'Connor DP. Nonunions: evaluation and treatment. In: Browner BD, Jupiter JB, Levine AM, Trafton PG, editors. Skeletal trauma: basic science, management, and reconstruction. 4th ed. Philadelphia: W.B. Saunders; 2009. p. 615–708.
[Crossref]

6. Court-Brown CM, Keating JF, Christie J, McQueen MM. Exchange intramedullary nailing. Its use in aseptic tibial nonunion. J Bone Joint Surg Br. 1995;77(3):407–11.
[PubMed]

7. Canadian Institute for Health information (CIHI). National trauma registry: hospital injury admissions. Ottawa: Canadian Institute for Health Information; 2003.

8. Heckman JD, Sarasohn-Kahn J. The economics of treating tibia fractures: the cost of delayed unions. Bull Hosp Jt Dis. 1997;56(1):63–72.
[PubMed]

9. Fong K, Truong V, Foote CJ, Petrisor B, Williams D, Ristevski B, et al. Predictors of nonunion and reoperation in patients with fractures of the tibia: an observational study. BMC Musculoskeletal Disord. 2013;14:103.
[Crossref]

10. Teraa M, Blokhuis TJ, Tang L, Leenen LPH. Segmental tibial fractures: an infrequent but demanding injury. Clin Orthop Relat Res. 2013;471(9):2790–6.
[Crossref][PubMed]

11. Nicoll EA. Fractures of the tibial shaft: a survey of 705 cases. J Bone Joint Surg Br 1964;46-B:373–87.

12. Yang JS, Otero J, McAndrew CM, Ricci WM, Gardner MJ. Can tibial nonunion be predicted at three months after intramedullary nailing? J Orthop Trauma. 2013;27(11):599–606.
[Crossref][PubMed][PubMedCentral]

13. Alt V, Donell ST, Chhabra A, Bentley A, Eicher A, Schnettler R. A health economic analysis of the use of rhBMP-2 in Gustilo-Anderson grade III open tibial fractures for the UK, Germany, and France. Injury. 2009;40(12):1269–75.
[Crossref][PubMed]

14. Hak DJ, Saleh K. Socioeconomic burden of traumatic tibial fractures: nonunion or delayed union. Mescape; 2001. http://www.medscape.org/viewarticle/418523. Accessed 22 April 2016.

15. Brinker MR, Hanus BD, Sen M, O'Connor DP. The devastating effects of tibial nonunion on health related quality of life. J Bone Joint Surg Am. 2013;95(24):2170–6.
[Crossref][PubMed]

16. Antonova E, Le TK, Burge R, Mershon J. Tibia shaft fractures: costly burden of nonunions. BMC Musculoskeletal Disorders. 2013;14:42.
[Crossref][PubMed][PubMedCentral]

17. Bishop JA, Palanca AA, Bellino MJ, Lowenberg DQ. Assessment of compromised fracture healing. J Am Acad Orthop Surg. 2012;20:273–82.
[Crossref][PubMed]

18. Rosenthal RE, MacPhail JA, Ortiz JE. Non-union in open tibial fractures. J Bone Joint Surg. 1977;59(2):244–8.
[Crossref][PubMed]

19. Hernigou J, Schuind F. Smoking as a predictor of negative outcome in diaphyseal fracture healing. Int Ortho. 2013;37(5):883–7.
[Crossref]

20. Ristiniemi J, Flinkkilä T, Hyvönen P, Lakovaara M, Pakarinen H, Biancari F, Jalovaara P. Two-ring hybrid external fixation of distal tibial fractures: a review of 47 cases. J Trauma. 2007;62(1):174–83.
[Crossref][PubMed]

21. Sloan A, Hussain I, Maqsood M, Eremin O, El-Sheemy M. The effects of smoking on fracture healing. Surgeon. 2010;8(2):111–6.
[Crossref][PubMed]

22. Mosely LH, Finseth F. Cigarette smoking: impairment of digital blood flow and wound healing in the hand. Hand. 1977;9(2):97–101.
[Crossref][PubMed]

23. Donigan JA, Fredericks DC, Nepola JV, Smucker JD. The effect of transdermal nicotine on fracture healing in a rabbit model. J Orthop Trauma. 2012;26(12):724–7.
[Crossref][PubMed]

24. Raikin SM, Landsman JC, Alexander VA, Froimson MI, Plaxton NA. Effect of nicotine on the rate of strength of long bone fracture healing. Clin Orthop Relat Res. 1998;353:231–7.
[Crossref]

25. Castillo RC, Bosse MJ, MacKenzie EJ. Patterson BM; LEAP Study Group. Impact of smoking on fracture healing and risk of complications in limb-threatening open tibia fractures. J Orthop Trauma. 2005;19(3):151–7.
[Crossref][PubMed]

26. Adams CI, Keating JF, Court-Brown CM. Cigarette smoking and open tibial fractures. Injury. 2001;32(1):61–5.
[Crossref][PubMed]

27. Bo J, Sudmann E, Marton PF. Effect of indomethacin on fracture healing in rats. Acta Orthop Scand. 1976;47(6):588–99.
[Crossref][PubMed]

28. Murnaghan M, Li G, Marsh DR. Nonsteroidal anti-inflammatory drug-induced fracture nonunion: an inhibition of angiogenesis? J Bone Joint Surg Am. 2006;88(Suppl 3):140–7.
[PubMed]

29. Karachalios T, Boursinos L, Poultsides L, Khaldi L, Malizos KN. The effects of the short-term

administration of low therapeutic doses of anti-COX-2 agents on the healing of fractures: an experimental study in rabbits. J Bone Joint Surg Br. 2007;89(9):1253–60.
[Crossref][PubMed]

30. Zhang X, Schwarz EM, Young DA, Puzas JE, Rosier RN. O'Keefe. Cyclooxygenase-2 regulates mesenchymal cell differentiation into the osteoblast lineage and is critically involved in bone repair. J Clin Invest. 2002;109(11):1405–15.
[Crossref][PubMed][PubMedCentral]

31. Simon AM, O'Connor JP. Dose and time-dependent effects of cyclooxygenase-2 inhibition on fracture- healing. J Bone Joint Surg Am. 2007;89(3):500–11.
[PubMed]

32. Giannoudis PV, MacDonald DA, Matthews SJ, Smith RM, Furlong AJ, De Boer P. Nonunion of the femoral diaphysis. J Bone Joint Surg Br. 2000;82(5):655–8.
[Crossref][PubMed]

33. Brinker MR, O'Connor DP, Monla YT, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trauma. 2007;21(8):557–70.
[Crossref][PubMed]

34. Patzakis MJ, Wilkins J, Kumar J, Holtom P, Greenbaum B, Ressler R. Comparison of the results of bacterial cultures from multiple sites in chronic osteomyelitis of long bones: a prospective study. J Bone Joint Surg Am. 1994;76(5):664–6.
[Crossref][PubMed]

35. Bhattacharyya T, Bouchard KA, Phadke A, Meigs JB, Kassarjian A, Salamipour H. The accuracy of computed tomography for the diagnosis of tibial nonunion. J Bone Joint Surg Am. 2006;88(4):692–7.
[PubMed]

36. Madsen J. Bone SPECT/CT detection of a sequestrum in chronic-infected nonunion of the tibia. Clin Nucl Med. 2008;33(10):700–1.
[Crossref][PubMed]

37. Matuszewski PE, Mehta S. Fracture consolidation in a tibial nonunion after revascularization: a case report. J Orthop Trauma. 2011;25(2):e15–20.
[Crossref][PubMed]

38. Brownlow HC, Simpson AH, Kenwright JK. The vascularity of atrophic non-union gaps. J Bone Joint Surg [Br]. 1999;81-B(Suppl. III):331.

39. Patzakis MJ, Zalavras CG. Chronic posttraumatic osteomyelitis and infected nonunion of the tibia: current management concepts. J Am Acad Orthop Surg. 2005;13(6):417–27.
[Crossref][PubMed]

40. Cierny G 3rd, Mader JT, Penninck JJ. A clinical staging system for adult osteomyelitis. Contemp Orthop. 1985;10:17–37.

41. Erdman WA, Tamburro F, Jayson HT, Weatherall PT, Ferry KB, Peshock RM. Osteomyelitis: characteristics and pitfalls of diagnosis with MR imaging. Radiology. 1991;180(2):533–9.

[Crossref][PubMed]

42. Perry CR, Pearson RL, Miller GA. Accuracy of cultures of material from swabbing of the superficial aspect of the wound and needle biopsy in the preoperative assessment of osteomyelitis. J Bone Joint Surg. 1991;73(5):745–9.
[Crossref][PubMed]

43. Megas P, Saridis A, Kouzelis A, Kallivokas A, Mylonas S, Tyllianakis M. The treatment of infected nonunion of the tibia following intramedullary nailing by the Ilizarov method. Injury. 2010;41(3):294–9.
[Crossref][PubMed]

44. Holtom PD, Patzakis MJ. Newer methods of antimicrobial delivery for bone and joint infections. Instr Course Lect. 2003;52:745–9.
[PubMed]

45. Harvey EJ, Henley MB, Siontkowski MF, Agel J, Benirschke SK. The use of a locking custom contoured blade plate for peri-articular nonunions. Injury. 2003;34(2):111–6.
[Crossref][PubMed]

46. Gardner MJ, Toro-Arbelaez JB, Boraiah S, Lorich DG, Helfet DL. Surgical treatment and outcomes of extraarticular proximal tibial nonunions. Arch Orthop Trauma Surg. 2008;128(8):833–9.
[Crossref][PubMed]

47. Reed LK, Mormino MA. Functional outcome after blade plate reconstruction of distal tibia metaphyseal nonunions. J Orthop Trauma. 2004;18(2):81–6.
[Crossref][PubMed]

48. Richmond J, Colleran K, Borens O, Kloen P, Helfet DL. Nonunions of the distal tibia treated with reamed intramedullary nailing. J Orthop Trauma. 2004;18(9):603–10.
[Crossref][PubMed]

49. Stafford PR, Norris BL. Reamer-irrigator-aspirator bone graft and bi Masquelet technique for segmental bone defect nonunions: a review of 25 cases. Injury. 2010;41(Suppl 2):S72–7.
[Crossref][PubMed]

50. Masquelet AC, Begue T. The concept of induced membrane for reconstruction of long bone defects. Orthop Clin North Am. 2010;41(1):27–37.
[Crossref][PubMed]

51. Sala F, Thabet AM, Castelli F, Miller AN, Capitani D, Lovisetti G, Talamonti T, Singh S. Bone transport for postinfectious segmental tibial bone defects with a combined Ilizarov/Taylor spatial frame technique. J Orthop Trauma. 2011;25(3):162–8.
[Crossref][PubMed]

52. Kundu ZS, Gupta V, Sangwan SS, Kamboj P. Gap nonunion of tibia treated by Huntington's procedure. Indian J Orthop. 2012;16(6):653–8.
[Crossref]

53. Desai PP1, Bell AJ, Suk M. Treatment of recalcitrant, multiply operated tibial nonunions with the RIA graft and rh-BMP2 using intramedullary nails. Injury. 2010;41(Suppl 2):S69–71.

54. Anglen J. Update on the management of nonunion. American Academy of Orthopaedic Surgeons Web Site: Orthopaedic Knowledge Online 2012;10(7). http://orthoportal.aaos.org/oko/article.aspx?article=OKO_TRA039#article. Accessed 30 Mar 2016.

55. Sarmiento A, Latta LL. Closed functional treatment of fractures. Berlin: Springer; 1981. p. 1–58. [Crossref]

56. Sarmiento A, Burkhalter WE, Latta LL. Functional bracing in the treatment of delayed union and nonunion of the tibia. Int Ortho. 2003;27(1):26–9.

57. Piriou P, Martin JR, de Loubresse CG, Judet C. Tibial nonunion after intramedullary nailing for fracture: decortication and osteosynthesis by medial plating. Rev Chir Orthop Reparatrice Appar Mot. 2005;91(3):222–31.
[Crossref][PubMed]

58. Helfet DL, Jupiter JB, Gasser S. Indirect reduction and tension-band plating of tibial non-union with deformity. J Bone Joint Surg Am. 1992;74(9):1286–97.
[Crossref][PubMed]

59. Wiss D, Johnson DL, Miao M. Compression plating for non-union after failed external fixation of open tibial fractures. J Bone Joint Surg Am. 1992;74(9):1279–85.
[Crossref][PubMed]

60. Johnson EE, Marder RA. Open intramedullary nailing and bone-grafting for non-union of tibial diaphyseal fracture. J Bone Joint Surg Am. 1987;69(3):375–80.
[Crossref][PubMed]

61. Brinker MR, O'Connor DP. Exchange nailing of ununited fractures. J Bone Joint Surg Am. 2007;89(1):177–88.
[PubMed]

62. Hsiao CW, Wu CC, Su CY, Fan KF, Tseng IC, Lee PC. Exchange nailing for aseptic tibial shaft nonunion: emphasis on the influence of a concomitant fibulotomy. Chang Gung Med J. 2006;29(3):283–90.
[PubMed]

63. Court-Brown CM, Keating JF, Christie J, McQueen MM. Exchange intramedullary nailing. Its use in aseptic tibial nonunion. J Bone Joint Surg Br. 1995;77(3):407–11.
[PubMed]

64. Zelle BA, Gruen GS, Klatt B, Haemmerle MF, Rosenblum WJ, Prayson MJ. Exchange reamed nailing for aseptic nonunion of the tibia. J Trauma. 57(5):1053–9.

65. Harshwal RK, Sankhala SS, Jalan D. Management of nonunion of lower-extremity long bones using mono-lateral external fixator— report of 37 cases. Injury. 2014;45(3):560–7.
[Crossref][PubMed]

66. Menon DK, Dougall TW, Pool RD, Simonis RB. Augmentative Ilizarov external fixation after failure of diaphyseal union with intramedullary nailing. J Orthop Trauma. 2002;16(7):491–7.
[Crossref][PubMed]

67.

García-Cimbrelo E, Martí-González JC. Circular external fixation in tibial nonunions. Clin Orthop. 2004;419:65–70.
[Crossref]

68. Takemoto RC, Fajardo M, Kirsch T, Egol KA. Quantitative assessment of the bone morphogenetic protein expression from alternate bone graft harvesting sites. J Orthop Trauma. 2010;24(9):564–6.
[Crossref][PubMed]

69. McKee MD, Wild LM, Schemitsch EH, Waddell JP. The use of an antibiotic-impregnated osteoconductive bioabsorbable bone substitute in the treatment of infected long bone defects: early results of a prospective trial. J Orthop Trauma. 2002;16(9):622–7.
[Crossref][PubMed]

70. Schultz, DG. HP19992 OP-1 (TM) Implant. Letter to LaForte AJ. Office of Device Evaluation, Food and Drug Administration, Department of Health & Human Services, Rockville, MD. 17 Oct 2001. http://www.accessdata.fda.gov/cdrh_docs/pdf/h010002a.pdf. Accessed 29 Mar 2016.

71. Friedlaender GE, Perry CR, Cole JD, Cook SD, Cierny G, Muschler GF, et al. Osteogenic protein-1 (bone morphogenetic protein-7) in the treatment of tibial nonunions. J Bone Joint Surg Am. 2001;83-A(Suppl 1Pt 2):S151–8.

72. Calori MG, Capanna R, Colombo M, Biase PD, O'Sullivan C, Cartareggia V, Conti C. Cost effectiveness of tibial nonunion treatment: a comparison between rhBMP-7 and autologous bone graft in two Italian centers. Injury. 2013;44(12):1871–9.
[Crossref]

73. Dahabreh Z, Dimitriou R, Giannoudis PV. Health economics: a cost analysis of treatment of persistent fracture non- unions using bone morphogenetic protein-7. Injury. 2007;38(3):371–7.
[Crossref][PubMed]

74. Kanellopoulos AD, Soucacos P. Management of nonunion with distraction osteogenesis. Injury. 2006;37(Suppl 1):S51–5.
[Crossref][PubMed]

75. Ring D, Jupiter JB, Gan BS, Israeli R, Yaremchuk MJ. Infected nonunion of the tibia. Clin Orthop. 1999;369:302–11.
[Crossref]

76. Dendrinos GK, Kontos S, Lyritsis E. Use of the Ilizarov technique for treatment of non-union of the tibia associated with infection. J Bone Joint Surg Am. 1995;77(6):835–46.
[Crossref][PubMed]

77. Huntington TWVI. Case of bone transference: use of a segment of fibula to supply a defect in the tibia. Ann Surg. 1905;41(2):249–51.
[Crossref][PubMed][PubMedCentral]

78. Kassab M, Samaha C, Saillant G. Ipsilateral fibular transposition in tibial nonunion using Huntington's procedure: a 12 year follow up study. Injury. 2003;34(10):770–5.
[Crossref][PubMed]

79. Bassett CA. Fundamental and practical aspects of therapeutic uses of pulsed electromagnetic fields

(PEMFs). Crit Rev Biomed Eng. 1989;17(5):451–529.
[PubMed]

80. Anglen J. The clinical use of bone stimulators. J South Orthop Assoc. 2003;12(2):46–54.
[PubMed]

81. Griffen XL, Costa ML, Parsons N, Smith N. Electromagnetic field stimulation for treating delayed union or non-union of long bone fractures in adults. Cochrane Database Syst Rev. 2011 Apr 13; (4):CD008471.

82. Heckman JD, Ryaby JP, McCabe J, Frey JJ, Kilcoyne RF. Acceleration of tibial fracture-healing by non-invasive, low-intensity pulsed ultrasound. J Bone Joint Surg Am. 1994;76(1):26–34.
[Crossref][PubMed]

83. Mayr E, Frankel V, Rüter A. Ultrasound—an alternative healing method for nonunions? Arch Orthop Trauma Surg. 2000;120(1–2):1–8.
[Crossref][PubMed]

84. Rubin C, Bolander M, Ryaby JP, Hadjiargyrou M. The use of low-intensity ultrasound to accelerate the healing of fractures. J Bone Joint Surg Am. 2001;83-A(2):259–70.

85. Mizuno K, Yamano Y, Itoman M, et al. Effects of low-intensity pulsed ultrasound therapy for delayed unions and nonunions: a multi-center clinical study. Orthopaedic Surg Traumatol. 2003;46:757–65.

86. Nolte PA, van der Krans A, Patka P, Janssen IM, Ryaby JP, Albers GH. Low-intensity pulsed ultrasound in the treatment of nonunions. J Trauma. 2001;51(4):693–702.
[Crossref][PubMed]

87. Pigozzi F, Moneta MR, Giombini A, Giannini S, Di Cesare A, Fagnani F, Mariani PP. Low-intensity pulsed ultrasound in the conservative treatment of pseudoarthrosis. J Sports Med Phys Fitness. 2004;44(2):173–8.

88. Rutten S, Nolte PA, Guit GL, Bouman DE, Albers GH. Use of low-intensity pulsed ultrasound for posttraumatic nonunions of the tibia: a review of patients treated in the Netherlands. J Trauma. 2007;62(4):902–8.
[Crossref][PubMed]

89. Watanabe Y, Matsushita T, Bhandari M, Zdero R, Schemitsch EH. Ultrasound for fracture healing: current evidence. J Ortho Trauma. 2010;24(Suppl 1):S56–61.
[Crossref]

90. Elster EA, Stojadinovic A, Forsberg J, Shawen S, Andersen RC, Schaden W. Extracorporeal shock wave therapy for nonunion of the tibia. J Orthop Trauma. 2010;24(3):133–41.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_14

# 14. Distal Tibia and Ankle Nonunions

Kevin J. Pugh[1]

(1) Orthopedic Trauma and Reconstructive Surgery, Grant Medical Center, 285 E. State St. #500, Columbus, OH 43215, USA

**Kevin J. Pugh**
**Email:** kjportho@mac.com

**Keywords** Nonunion – Ankle – Malleoli – Hypertrophic nonunion – Atrophic nonunion – External fixation – Internal fixation

## 14.1 Introduction

Normal gait is dependent on normal function of the ankle joint [1, 2]. Disruption of the ankle mortise by fracture of the plafond, malleoli, or syndesmosis can lead to ankle instability or lack of articular congruity, both of which can lead to degenerative arthritis. Anatomic reduction and stable fixation of fractures about the ankle is required to restore function and to obtain union. Nonunions of the ankle lead to stiffness, dysfunctional gait, prolonged morbidity, and prolonged social stress on the patient. In addition, malreduced nonunions affect the mechanical axis of the limb, leading to dysfunction of the adjacent knee and subtalar joint.

## 14.2 Analysis of the Fracture

There is no one method of treatment for a nonunion. Instead, it is an analysis and correction of the cause of the nonunion and the unique features of each specific case. The fact that there is no one technique or implant to treat

nonunions is what makes them both challenging and satisfying to treat. The treatment plan, whether simple or involving several stages, is devised by following a set of principles as illustrated throughout this book. The principles of treating nonunions about the ankle will be illustrated in this chapter.

Injury to bone, whether it be due to a fracture, osteotomy, or attempted arthrodesis, initiates a predictable series of events that under normal circumstances should proceed to a union. Although a full discussion of the physiology of bone healing can be found in Chap. 2, to briefly summarize, the initial inflammatory process leads to cell recruitment and differentiation, the laying down of new bone matrix, and eventually to ossification, boney union, and remodeling of the immature construct. However, not all fractures are created equally. They differ in terms of the energy imparted, the size of the zone of injury, whether they are open or closed, the fracture pattern and comminution, the bone involved as well as the region on that bone, and the presence of articular involvement. Injuries to the ankle involve more than just the boney structures. The bone around the ankle is generally subcutaneous and often has poor soft tissue coverage. Thus, due to these characteristics, fractures about the ankle, unfortunately, often have significant associated soft tissue injuries. Perhaps the most important variable, and the least predictable, is the host. The medical and social history of the patient and his compliance can have a major effect on the success or failure of the treatment plan. A successful outcome requires a balance between the stability of the fracture treatment construct and the biological viability of the fracture site.

Unlike muscle, cartilage, and other connective tissue, bone does not heal with scar tissue. It is one of the few tissues in the body that heals with the same material—bone. Fractures, osteotomies, and arthrodesis constructs are thus repaired by a process of boney regeneration. A boney union is one that is repaired to the degree that it is mechanically able to function like denovo bone. The patient experiences no pain, and there is clinical stability at the fracture site. Clinical fracture unions are accompanied by radiographic signs of healing. In order to confirm a true union, both radiographic and clinical signs should be present. A delayed union is a fracture that, though making continual progress toward union, has not healed in the usual amount of time for a similar fracture. A nonunion is a fracture that will not heal. It has sustained an arrest of the repair process and has not shown radiographic or clinical progress toward healing for months. Nonunions may have some

clinical stability, as they will have cartilage or fibrous interposition instead of bone. Others will be atrophic, with little healing tissue, and have no clinical stability. Though nonunions cannot be predicted, some fractures are destined to go on to nonunion from the beginning of treatment.

## 14.3 Determination of Delayed or True Nonunion

The first issue in treatment is determining whether the fracture is merely delayed or a true nonunion [3]. A delayed union may go on to a successful outcome if given more time, while a true nonunion will require intervention to achieve union. This is not a trivial question to answer for the patient. Though most nonunions will be diagnosed if the surgeon waits long enough, it is imperative to identify fractures that are falling behind as soon as possible in order to shorten overall treatment time and to restore the patient back to full function. Government payers and many private insurance companies subscribe to clinical guidelines that incorporate a time factor into the definition of a nonunion. The United States Food and Drug Administration defines a nonunion as being “established when a minimum of nine months has elapsed since injury and the fracture site shows no visibly progressive signs of healing for a minimum of three months” [4]. This definition is not pragmatic and leads to prolonged morbidity, long periods of work-related impairment or socioeconomic stress, and the potential for narcotic abuse. As surgeons, we know that there are injuries that are at risk for nonunion based on the injury (open fracture, comminution, or bone loss), anatomic location (distal tibia and 5th metatarsal), or host (diabetic, smoker, and cancer patient). It has thus become more acceptable to label fractures as delayed or nonunions when the surgeon believes the fracture has little or no ability to heal. Delaying intervention for an arbitrary length of time before calling a fracture a nonunion results in more disability, more time off work, and greater psychological stress for the patient. As soon as slow healing is identified, there should be a frank discussion with the patient about the possibility of nonunion and the need for further future treatment. Most patients will opt for early intervention if it means an earlier return to work or recreational activities.

The causes of nonunion are multiple, and if identified should be addressed during treatment [4–8]. An inappropriate fracture treatment construct, whether unstable or too stiff, malpositioned or distracted, will lead

to poor results. The injury itself may result in comminution, bone loss, soft tissue injury, or stripping and avascularity. Infection can result in bone death with formation of a sequestrum, as well as osteolysis with loosening of implants and eventual instability at the fracture site. Host factors such as age, nutritional status, metabolic abnormalities [9], chronic disease, medicines, and smoking all play a role. In some patients, the cause is not identifiable, and thus idiopathic.

## 14.4 Classification of Nonunions

Unlike acute fractures, there is no single definitive classification system for nonunions. Nonunions can be classified on the basis of their anatomy, the presence or absence of infection, their biological potential, or their stiffness. Often more than one method of describing the nonunion will be helpful in determining a treatment plan.

Nonunions can be classified by their anatomic location. Diaphyseal nonunions have relatively less biological potential as they involve cortical bone, but are amenable to a wide variety of treatment methods, including nails, compression plating, and external fixation. The goal in this instance is to restore length and axial alignment while achieving fracture union. As the nonunion reaches the metaphyseal region, the goals remain the same. The potential for bone growth improves in the metaphysis, but the options for fixation are more limited. Fractures of the metadiaphyseal region of the tibia are particularly problematic. Peri-articular nonunions may also be associated with stiff, contracted, or arthritic joints that must be accounted for in the preoperative plan. Nonunions of the malleoli, with their ligamentous attachments, can lead to joint instability. Nonunions of the articular surface are particularly challenging. Defining the extent of the non-united segment may require multiple radiographs and computed tomography scans. Step-offs, gaps, and injury to the joint surface may lead to local or global articular arthritis. In the ankle, treatment may consist of open reduction and rigid fixation or arthrodesis.

Nonunions may be aseptic or infected. Though many authors have shown that bone constructs with adequate stability can heal in the face of infection, the general goal is to convert an infected nonunion into a non-infected nonunion, and then proceed with treatment of the fracture. Though many infected nonunions will have skin breakdown, open wounds, and drainage,

the diagnosis is not always obvious. Laboratory studies can be helpful, as can nuclear medicine studies. The patient should be counseled that treatment might take several staged procedures for hardware removal, debridement of dead bone, soft tissue coverage, and stabilization. A period of intravenous antibiotics-based thorough deep cultures is followed by definitive reconstruction. Depending on the extent of the infection and the amount of bone resected, this may require a period of months. Failed soft tissue coverage, failure to eradicate the infection, or failure to obtain boney union may lead to eventual amputation.

Weber and Čech [10] classified nonunions radiographically based on their biological potential. Hypertrophic nonunion s are characterized by abundant bone formation and are often referred to as having the appearance of an elephant foot. In general, they are stiff and relatively stable. Patients are often able to weight bear with pain on a hypertrophic nonunion. They have excellent blood supply and biological potential, and often require only the addition of stability for the fracture to unite (Fig. 14.1). Atrophic nonunions , on the other hand, have little biological potential. Atrophic nonunions are often the result of open fractures or previous surgical procedures that have caused a disruption of the normal vascular supply to the bone. They have had a cessation of the regeneration process, resorption of the bone ends, and in many instances, closure of the endosteal canal of the bone. These nonunions are mobile; patients usually are unable to bear weight and may require external immobilization for comfort. A special case of the atrophic nonunion is a true pseudarthrosis in which a false joint has been created between the two ends of the bone. These fractures need biological stimulation in addition to skeletal stability. Bone grafting and other adjuvants often play a role in their treatment. Oligotrophic nonunions are somewhere in between these two extremes. They have very little callous formation, but the bone ends are vital. They often require both biological and mechanical augmentation.

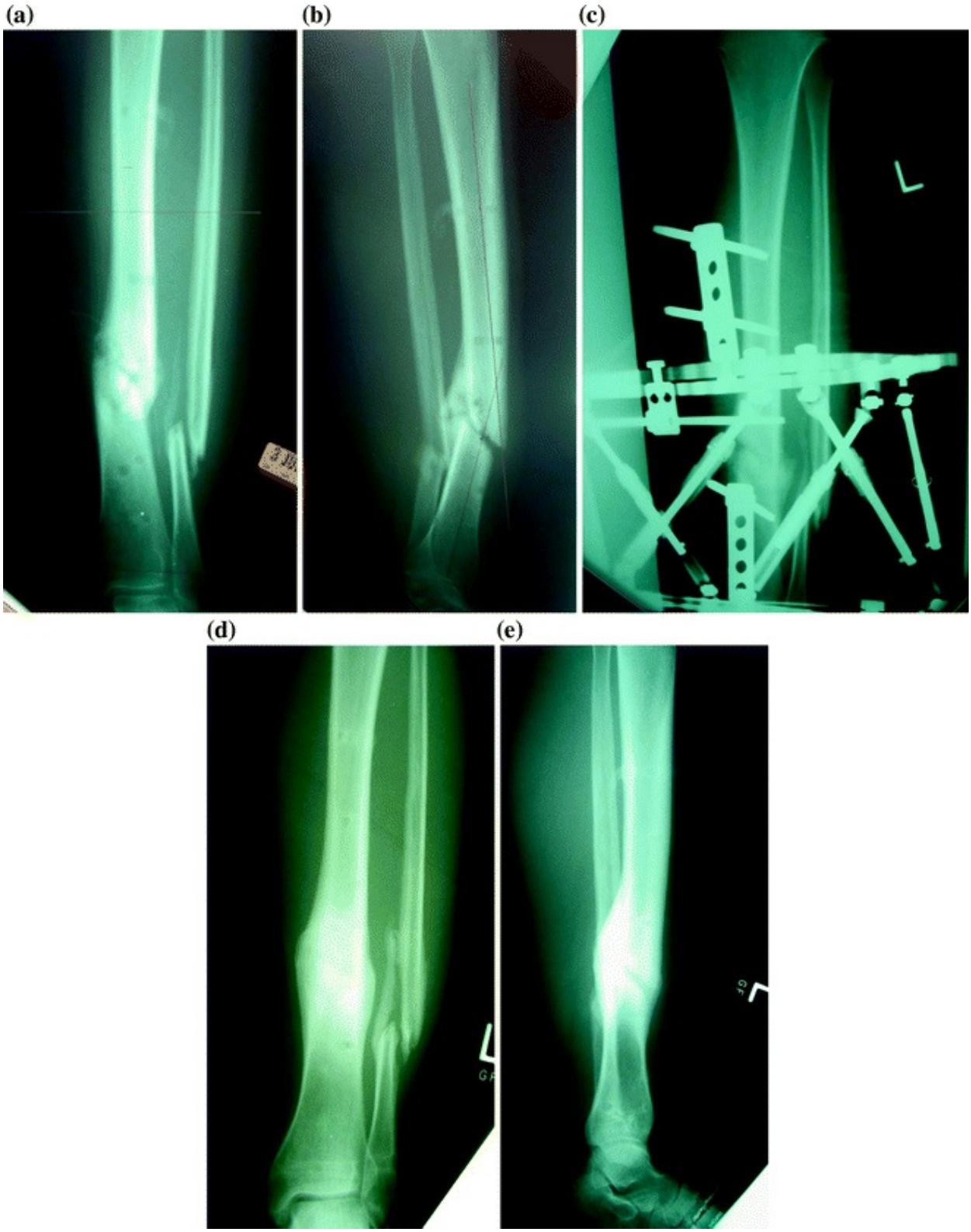



***Fig. 14.1*** 13-year-old girl hit by a light pole while waiting for the school bus. **a, b** Anteroposterior (AP) and lateral of the hypertrophic nonunion with deformity. **c** Application of a multiplanar external fixator to add stability, no fracture exposure, deformity correction. **d, e** AP and lateral of fracture union

after frame removal

Ilizarov described nonunions as stiff or mobile [11–13]. Stiff nonunions, those with less than 7 degrees of motion at the fracture site, are thought to be biologically viable and thus equivalent to the hypertrophic nonunion. They require stability to heal, are biologically viable, and can be a source of new bone formation if distracted. In many instances, they can be treated closed. Lax nonunions have more than 7 degrees of motion and are equivalent to the atrophic nonunion of Weber. They not only require stability to heal, but also require open debridement of the nonunion site to stimulate the bone and the addition of bone graft or other biological stimulus. They are not a source of new bone with pure distraction, but instead require compression.

The evaluation of the patient with a nonunion, just as with an acute injury, requires a thorough look at more than just the fracture pattern and the radiographs. One must determine the "personality of the fracture," as coined by Schatzker and Tile [14]. This involves a complete history of the events of the injury, the fracture, the host, the treating physician, and the institution at which the treatment will occur. Only with this kind of analysis can one do proper preoperative planning and optimize the chance for success.

## 14.5 Clinical Evaluation

A comprehensive history is essential, as a complete picture of the fracture and the host must be obtained. Was the initial injury open or closed? Was there a high-energy mechanism such as a motorcycle accident or a lower energy trip and fall? Were there any neurovascular issues at the time of initial injury or after treatment? A determination of the type and number of previous surgeries is essential, as is the presence and treatment of previous infection. If there is retained hardware at the fracture, old operative notes can be helpful in identifying the hardware type and manufacturer for planned removal. Have previous fractures healed in a timely fashion? Patients with recreational drug habits or other substance abuse may have compliance issues. Smokers are at risk because of the well-documented relationship between nicotine use and delayed healing. Patients using nicotine gum are not immune to this problem. The occupation of the patient is important, as treatment that requires a non-weight bearing gait will cause a longer period off work for a laborer than for a patient with a more sedentary occupation. The knowledge of the avocations

and hobbies of your patient are also important, as it rounds out the level of activity to which the patient must return. Hospital discharge planning often begins before surgery. The patient's living situation, amount of support from family or friends, their financial resources, the location of their home, and what type of dwelling in which they reside is helpful in planning successful aftercare.

A thorough musculoskeletal examination is also mandatory. Examination of the patient's other extremities will provide clues as to other disabilities that may play a role in mobility and later rehabilitation. Examination of the non-united segment includes an inspection for gross deformity and overall limb alignment. Gross limb length can be checked, and if the patient is ambulatory, the gait pattern should be examined. The fracture site should be checked for pain to manual stress, as well as the presence of gross or subtle motion. The stability and motion of adjacent joints should be examined. Ligamentous instability may require reconstruction as part of the treatment plan. If there is joint contracture or subluxation present, it should be determined if it is due to soft tissue contracture, heterotopic ossification, joint ankylosis, or a combination of factors.

The skin should be inspected for the presence, location, and healing status of previous open wounds and incisions. Adherent skin, especially in areas with subcutaneous bone such as the medial face of the tibia, the distal fibula, and the calcaneus, can be an obstacle. The presence or absence of lymphedema or venous stasis should be noted, as it may influence the choice of surgical approach. If previous external fixators have been in place, the condition of the old pin sites should be examined for signs of previous infection. A complete neurovascular examination should be carried out. Patients with suspected dysvascular limbs should be sent for more thorough testing, including transcutaneous oxygen tension and ankle-brachial indices. Existing nerve deficits can be examined tested by electromyography to determine the likelihood of recovery.

Radiographic evaluation includes true anteroposterior and lateral films of the problem limb segment, orthogonal to the "normal" portion of the limb. Radiographic signs of a nonunion can be subtle but include the absence of bridging trabeculae, sclerotic fracture edges, persistent fracture lines, and broken or displaced hardware. If deformity or limb length issues are suspected, additional work-up is required. Standard full-length alignment films should be obtained, as well as alignment films centered on each area in

question, i.e., tibia or ankle. Deformities must be fully characterized in all six axes so that correction can be planned. Comparison films of the contralateral leg are helpful in determining the normal alignment of the patient, and population normals can be used if the problem is bilateral. Computed tomography scans with reconstructions can be helpful in analyzing subtle nonunions, but can be hard to interpret with fracture fixation devices in place. Plain tomography can be very helpful in these instances, but is increasingly unavailable. If infection is suspected, a combined bone scan and tagged white cell study can help differentiate bone turnover from active infection. Magnetic resonance imaging can be helpful in evaluating a bone for infection, or looking at ligaments in adjacent joints, but are not commonly used in the evaluation of nonunions.

Laboratory studies can round out the clinical picture of the patient. In addition to routine preoperative chemistries and blood counts, patients suspected of infection should have their erythrocyte sedimentation rate and C-reactive protein checked. Patients suspected of malnutrition should have a complete nutritional panel drawn, including liver enzymes, total protein and albumin levels, and calcium, phosphate and vitamin D levels.

The last part of developing the “personality of the fracture” is a critical self-examination of the surgeon and the treating facility. Surgeons should honestly examine whether they have the training, skill, patience, and experience necessary to treat a complex nonunion. Even the most gifted surgeon requires help, and the appropriate consultants must be available from plastic and vascular surgery, internal medicine, and infectious disease. The hospital is the final element. Is the correct equipment in the house or available to be brought in? Is experienced nursing and surgical assistance available? Can the anesthesia staff care for the needs of the patient?

At the end of the evaluation, the surgeon should create a complete problem list in anticipation of preoperative planning [15]. An attempt should be made to define the cause of the nonunion and reverse it. Soft tissue defects, either existing or anticipated, must be covered. The consults required should be listed and obtained. Infected nonunions require debridement, temporary stabilization, and conversion to a non-infected nonunion, with eventual staged reconstruction. Constructs with mechanical instability should be made stable; those with a gap require strategies to restore bone loss; those with deformity require a better reduction or length; and those with vascularity require a better soft tissue environment and biological stimulation.

Using this problem list, a detailed preoperative plan should be drawn out in detail in all but the simplest of conditions. Putting the case on paper, often with multiple methods or implants, allows one to foresee possible obstacles to success, to define the sequential steps in the operation, to select the appropriate patient positioning as well as to ensure the availability of equipment and implants, and to make the procedure in the operating room the execution of a plan instead of a surgical adventure (Tables 14.1 and 14.2).

***Table 14.1*** Treatment suggestions: distal tibia and ankle nonunions

| Classification | Objective | Treatment | Suggestions | Problems |
|---|---|---|---|---|
| Hypertrophic nonunion | Provide stability | Plate, external fixation, nail with polar screws | Does not require grafting | Must provide adequate stability |
| Atrophic/oligotrophic nonunion | Provide stability and biological stimulus | Bone graft or appropriate substitute, stable fixation | Thorough debridement or excision of nonunion | Failure to provide biology and stability |
| Nonunion with deformity | Treat nonunion and deformity | Deformity correction, stability and biology | Formal deformity analysis | Failure to restore mechanical axis |
| Metaphyseal nonunion | Maintain axial alignment | Plate, external fixation, nail with polar screws | Provide adequate fixation, build external fixator to foot if needed | Prevention of deformity |
| Malleolar nonunion | Restore joint stability | AO techniques | Restore ankle mortise, stress views in OR | Failure to restore joint stability |
| Articular nonunion | Restore articular surface | Rigid internal fixation | Arthrodesis if surface is not reconstructable | Cartilage injury, poor prognosis |

***Table 14.2*** Treatment strategy: distal tibia and ankle nonunions

| Treatment method | Clinical indication |
|---|---|
| Plate and screw fixation | Metaphyseal, malleolar, or articular nonunion, no infection, adequate soft tissue |
| Intramedullary nail | Metaphyseal location, may require polar screws for stability, no infection |
| Multiplanar external fixation | Larger deformity, leg length deficiency, infection, bone defect, poor soft tissue, joint subluxation |
| Acute correction | Small or no deformity, no lengthening, adequate soft tissues, nonunion requires open approach |
| Gradual correction | Larger deformity, leg length deficiency, infection, bone defect, poor soft tissue, joint subluxation |

Some nonunions need no treatment at all. Patients with normal alignment, normal function, and no pain may not require surgical treatment. This is most common with small fractures of the posterior malleolus or at the tips of the medial or lateral malleolus. Nonunions of the metaphysis are usually painful or involve deformity, while those of the articular surface predispose the patient to arthritis and require treatment. In all cases, surgery is contraindicated where the morbidity of the treatment exceeds the expected benefit in function .

Hypertrophic and stiff nonunions require only stability to promote union [11, 16]. Stable constructs minimize motion, allow compression, and minimize shearing at the fracture site. A stable fracture construct, made up of the patients bone and the fixation device, allows stable vascular ingrowth and the progression of fracture healing. This may involve the addition of blocking screws to improve the stability of a nailing construct in the distal tibial metaphysis, plate fixation , or compression with an external fixator .

## 14.6 Atrophic Nonunions

Atrophic and oligotrophic nonunions require both stability and biology. Patients with bone loss and infection fall into this category as well (Fig. 14.2). These fractures require the most preoperative planning, often involving several stages, as the physiological environment is inadequate to promote healing. Medical problems must be treated, while vascular surgery and plastic surgery consults may be necessary to correct soft tissue problems. These fractures must be opened and the bone ends debrided back to healthy viable tissue. All nonviable scar tissue must be removed, and the endosteal canal of the bone must be opened, either with a curette or a drill bit.

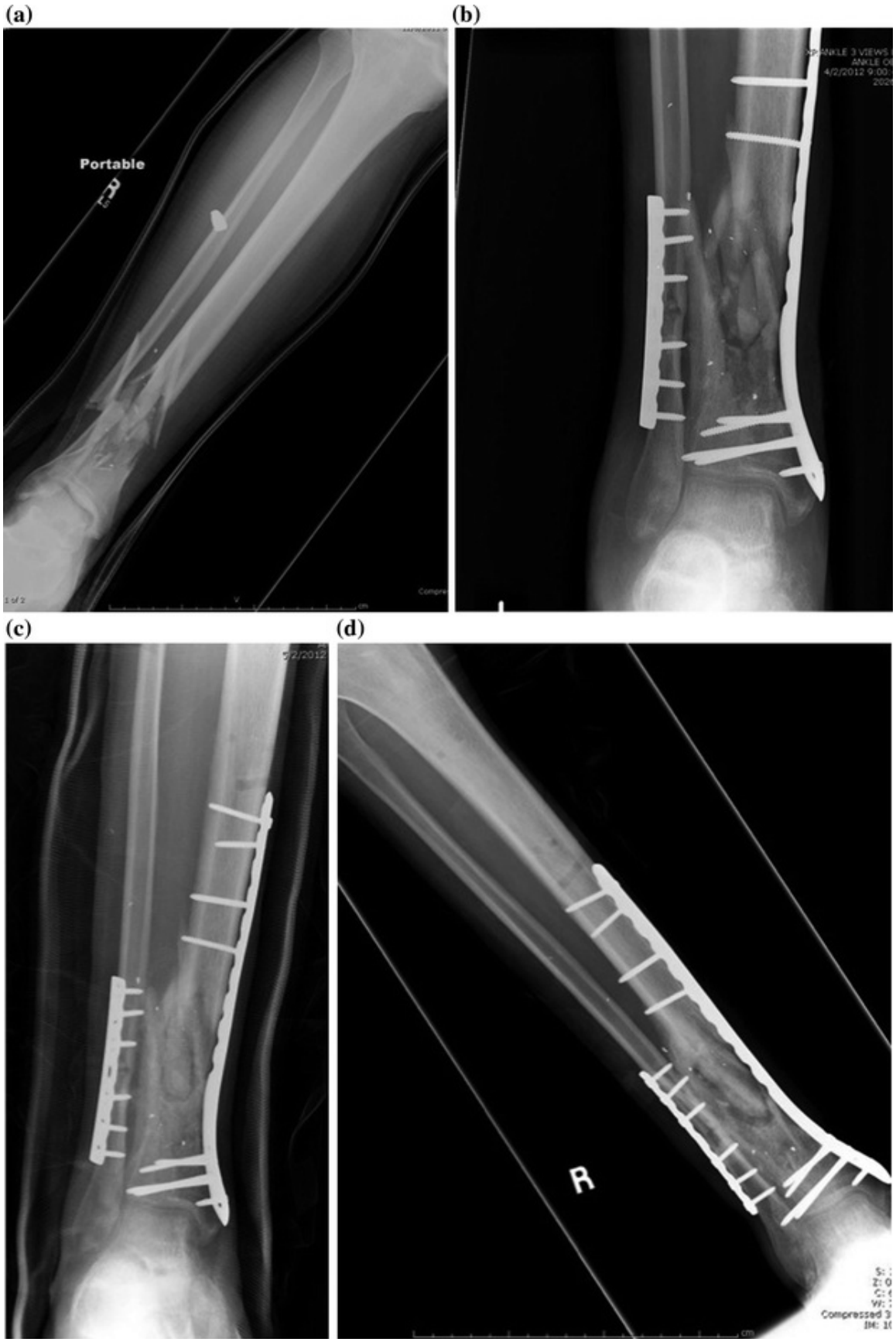

(a)
(b)
(c)
(d)
Portable
R

*Fig. 14.2* 30-year-old man after high-velocity gunshot wound. **a** Initial injury. **b** Three months after initial percutaneous plating, impending nonunion due to bone loss **c** Anteroposterior after bone grafting with reamer irrigator aspirator. **d** Fracture union 3 months after grafting

Atrophic fractures also require the addition of boney stimulus in the form of bone grafting [17–21]. Though the gold standard remains autogenous cancellous bone from the iliac crest, there are many other methods available to the orthopedic surgeon. Autogenous graft may come in the form of local bone from the proximal tibia or calcaneus and endosteal bone harvested from the femur or the tibia with a reamer-irrigator-aspirator or with other patient-derived material such as bone marrow aspirate. There are many commercially available bone graft substitutes in the marketplace from osteoconductive ceramics to osteoinductive growth factors, each with a specific use. Some patients will require more of an osteoconductive scaffold, while others will require a true osteogenic graft substitute. The challenge to the surgeon is to cut through the extensive marketing noise and to select the product, combination of products, or method that will solve unique clinical needs of each specific patient.

## 14.7 Infected Nonunions

Infected nonunions (Fig. 14.3) require a thorough debridement with intraoperative cultures. Existing colonized hardware must be removed. Dead bone and devitalized soft tissue must be debrided back to a healthy fracture bed. The endosteal canal must be opened to improve blood supply. In some cases, this means resecting the infected nonunion as if it were a malignancy. Dead space and areas of bone loss may be filled with antibiotic impregnated methacrylate beads or block spacers. These methods preserve space for bone grafting, elute local antibiotics, and allow the formation of vascularized membranes as popularized by Masquelet [22, 23]. Most infected nonunions are treated in a staged fashion. The first stage is to convert the infected nonunion to an aseptic atrophic nonunion by creating a viable fracture environment, obtaining deep cultures, managing dead space, and providing temporary stability. The second stage is definitive treatment of the atrophic fracture.


(a)
(b)
(c)
(d)

***Fig. 14.3*** 40-year-old diabetic 8 weeks after pilon fracture, grossly infected. **a** Mortise on presentation, medial wound visible. **b** After debridement of infected distal tibia, hardware removal, temporary spanning external fixator, and antibiotic spacer. **c** After flap coverage, nail, reamer-irrigator-aspirator graft. Salvage of joint with arthrodesis. **d** Final union

## 14.8 Traditional Plating Techniques

Nonunions of the distal tibia and ankle are very amenable to treatment with traditional plating techniques [24], following the principles advocated by the Arbeitsgemeinschaft für Osteosynthesefragen (Association for the Study of Internal Fixation) or AO group. Plating can be used to provide minimally invasive bridging fixation and stability to a well-aligned hypertrophic nonunion in a previously non-operatively treated distal fibula or tibial metaphysis. However, this method generally requires judicious fracture exposure for removal of hardware and direct reduction of the fragments. In nonunions with a questionable soft tissue envelope, this requirement can limit the available surgical approaches and fracture access. AO technique allows for rigid fixation of articular fragments and is thus required in nonunions involving the articular surface as well as the malleoli and syndesmosis. Plating also allows for absolute or relative stability constructs of the metadiaphysis [25]. Locking screw technology and anatomically contoured plates allow fixation of increasing smaller segments of the distal tibia. Plating techniques are suitable for use in nonunions with smaller and acutely correctable deformities, as well as in those with only small length discrepancies. As deformities become larger, especially in length, they are stiffer and more difficult to correct; plating becomes more of a challenge.

## 14.9 External Fixation and Intramedullary Nailing

Simple uniplanar external fixation is useful for the temporary stabilization of an infected nonunion in preparation for definitive reconstruction. Uniplanar constructs, usually with two tibial half pins and a transfixation pin through the calcaneus joined by bars in a delta configuration, provide relative stability at a distance after removal of hardware and fracture debridement in an infected case, or one that may require soft tissue coverage. In preparation for the second stage of treatment, patients are more comfortable, the fracture is grossly aligned, and the skin is accessible to examine wounds or flaps. These

frames are generally removed at the time of definitive reconstruction.

Multiplanar external fixation [26, 27] is generally reserved for definitive fixation of nonunions of the distal tibial metaphysis. These devices are available in a unibody design with fixation to the patient using half pins in the tibia and foot, or a circular ring configuration using thin wires and half pins for fixation. Traditional circular frames are built specifically for each patient, but may require multiple revisions in patients requiring step-wise correction of accompanying deformity. Newer fixator designs incorporate a Web-based computer program that may decrease the need for multiple rebuilds. Multiplanar external fixators are very stable and can allow earlier weight bearing during treatment. The major advantage to these devices in distal tibia nonunions is the ability to simultaneously correct multi-axis deformity, lengthen the bone if necessary, and modulate the fracture dynamically in compression and distraction. They can also be used to span the ankle joint, allowing simultaneous correction of foot deformities, ankle subluxation, or contracture (Fig. 14.4). In many instances, they can be combined with rigid screw fixation of the joint to solve multi-focal problems .

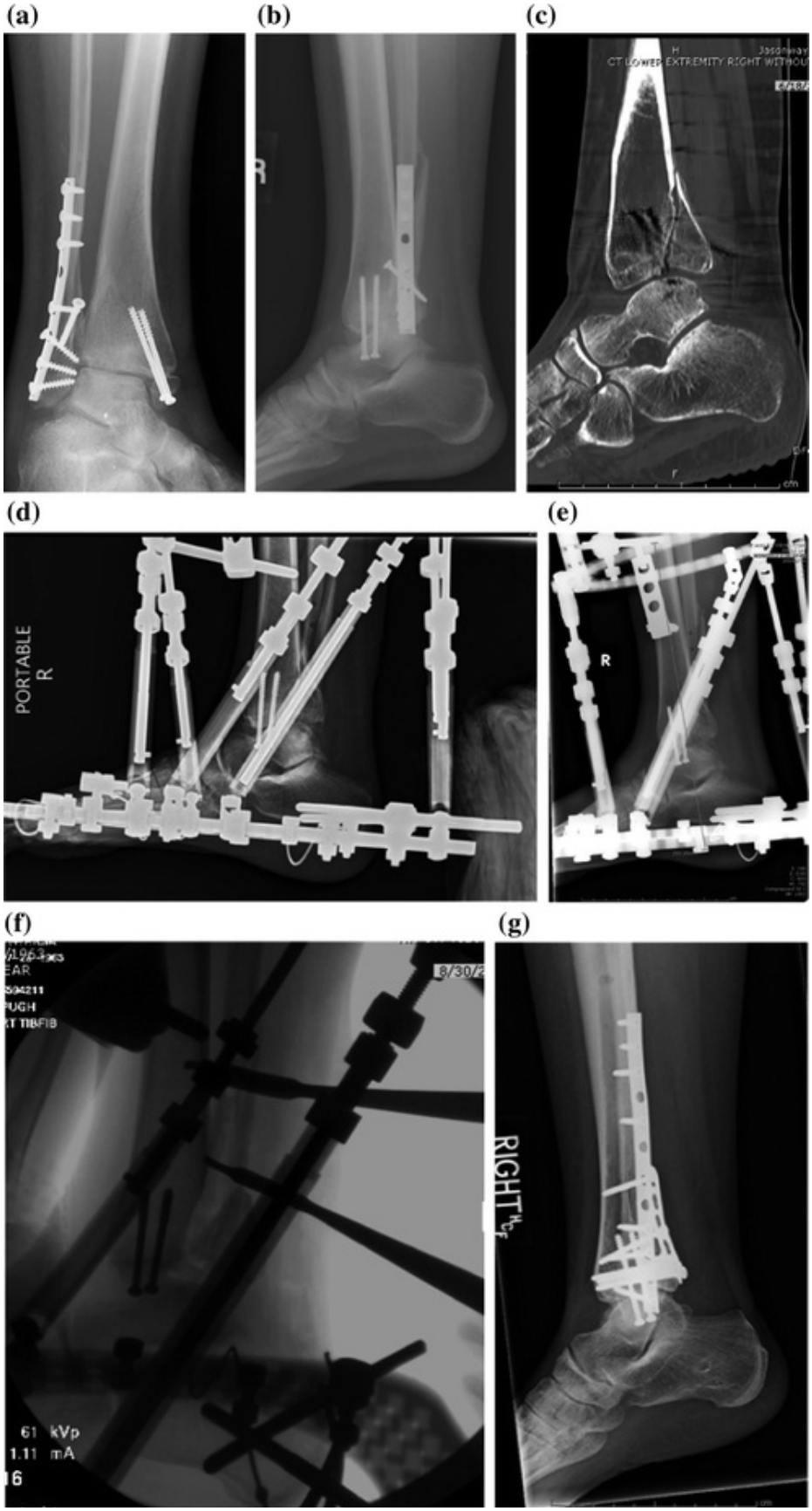

(a)
(b)
(c)
(d)
(e)
(f)
(g)
PORTABLE
R
R
RIGHT
61 kVp
1.11 mA

***Fig. 14.4*** 40-year-old patient eight weeks after treatment for a trimalleolar ankle fracture **a** mortise, **b** lateral and **c** computed tomography of malunited medial malleolus, non-united lateral and posterior malleolus with posterior ankle subluxation **d** application of multiplanar external fixator for joint distraction and reduction **e** after correction of joint subluxation **f** intraoperative view of repair of posterior malleolus **g** twenty-four month follow up with joint reduction and fracture union

Intramedullary nailing is generally not a primary method in the treatment of distal tibia and ankle nonunions [28]. Metadiaphyseal fractures of the tibia treated initially with a nail and which go on to nonunion are often revised to another form of fixation to add stability. Because of the size mismatch between the canal diameter and the nail, nailing does not increase stability unless used in combination with blocking screws or supplementary plates. A common application of a nail in this setting is in the salvage of the unreconstructable ankle with a pantalar fusion. Nails can also be used to accelerate frame removal in fractures with slow healing regenerate or bone graft (Fig. 14.5).

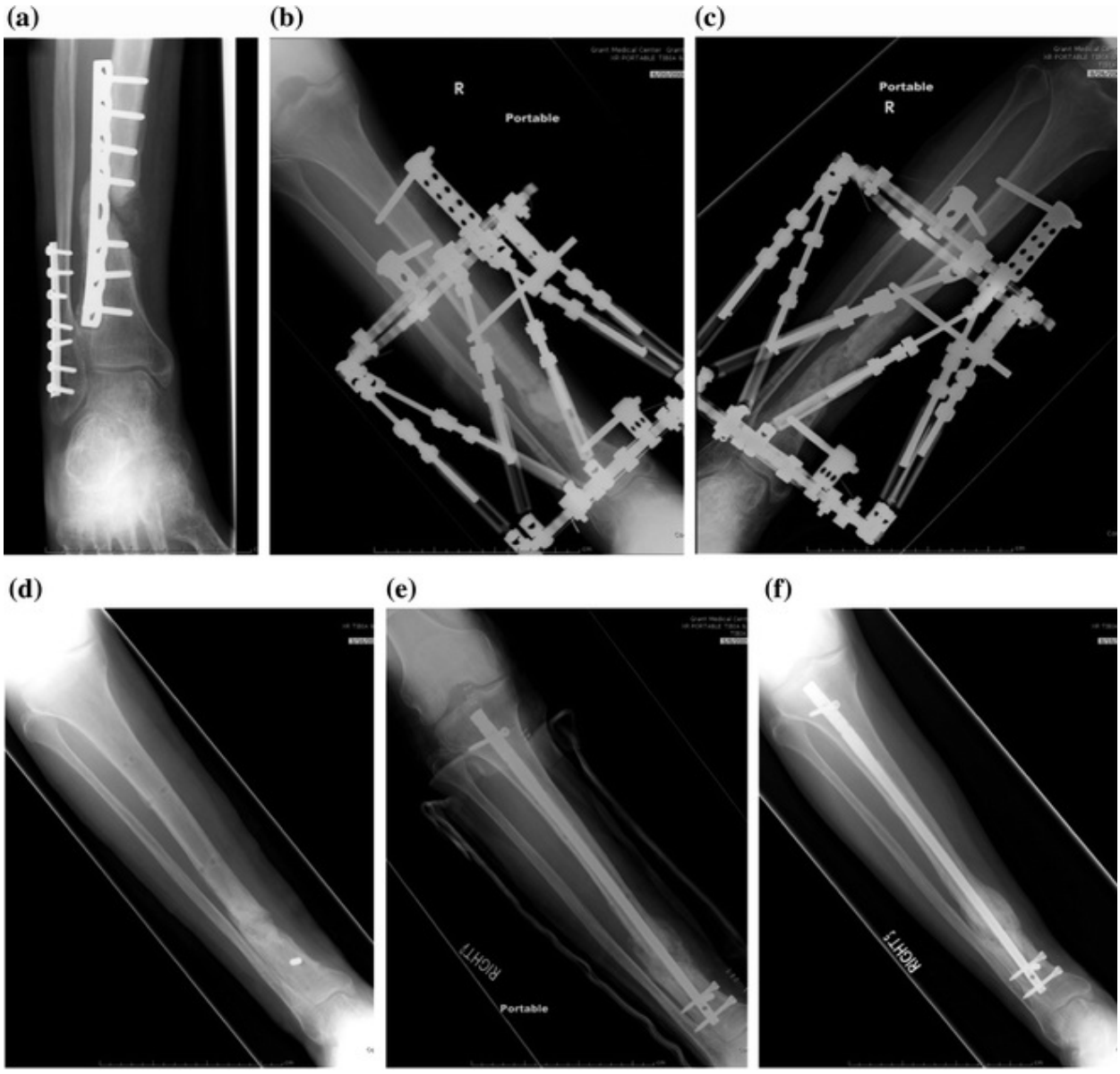



***Fig. 14.5*** 70-year-old man after distal tibia fracture while on cruise (repaired in the Middle East). **a** Fracture at presentation. Erythrocyte sedimentation rate and C-reactive protein elevated. **b** Hardware removed, antibiotic spacer, external fixation. **c** Antibiotic spacer removal, bone grafting. **d** Nonunion in external fixator. External fixator removed, frame holiday. **e** Salvage of metaphyseal nonunion with intramedullary nailing. **f** Fracture union

Arthodesis is a viable option in ankle and distal tibia nonunions in patients with joint stiffness, an articular surface that is not reconstructable, or with a poor capacity to heal. Lag screw fixation, blade plating, and external fixation can all be utilized successfully to achieve a solid fusion. If the subtalar joint remains mobile, it may be spared. If it is stiff, painful, or contracted, the ankle may be salvaged with a fusion of both ankle and

subtalar joints.

Amputation is always part of the informed consent process, especially in patients who have had multiple procedures for stability and soft tissue coverage and are still not united. Some of these patients will have been undergoing limb reconstruction for several months without result and can view this discussion as an admission of failure. These patients often have severe economic and social pressures and pain control issues, and are facing the prospect of months of continued treatment. An amputation is not a failure for the patient, but a way to cut their losses and to get on with life. A well-done below knee amputation will remove the problem and allows the patient to be on their feet in a number of weeks instead of months.

---

# References


1. Mann RA. Biomechanics of the foot. Instr Course Lect. 1982;31:167–80.
[PubMed]

2. Kotwick JE. Biomechanics of the foot and ankle. Clin Sports Med. 1982;1(1):19–34.
[PubMed]

3. Bhandari M, Fong K, Sprague S, Williams D, Petrisor B. Variability in the definition and perceived causes of delayed unions and nonunions: a cross-sectional, multinational survey of orthopaedic surgeons. J Bone Joint Surg Am. 2012;94(15):e1091–6.
[Crossref][PubMed]

4. Haverstock BD, Mandracchia VJ. Cigarette smoking and bone healing: implications in foot and ankle surgery. J Foot Ankle Surg. 1998;37(1):69–74; discussion 8.

5. Niikura T, Lee SY, Sakai Y, Nishida K, Kuroda R, Kurosaka M. Causative factors of fracture nonunion: the proportions of mechanical, biological, patient-dependent, and patient-independent factors. J Orthop Sci. 2014;19(1):120–4.
[Crossref][PubMed]

6. Hernigou J, Schuind F. Smoking as a predictor of negative outcome in diaphyseal fracture healing. Int Orthop. 2013;37(5):883–7.
[Crossref][PubMed][PubMedCentral]

7. McKee MD, DiPasquale DJ, Wild LM, Stephen DJ, Kreder HJ, Schemitsch EH. The effect of smoking on clinical outcome and complication rates following Ilizarov reconstruction. J Orthop Trauma. 2003;17(10):663–7.
[Crossref][PubMed]

8. Schmitz MA, Finnegan M, Natarajan R, Champine J. Effect of smoking on tibial shaft fracture healing. Clin Orthop Relat Res. 1999;365:184–200.
[Crossref]

9. Brinker MR, O'Connor DP, Monla YT, Earthman TP. Metabolic and endocrine abnormalities in patients with nonunions. J Orthop Trauma. 2007;21(8):557–70.
[Crossref][PubMed]

10. Weber BG, Čech O. Pseudoarthrosis: pathology, biomechanics, therapy, results. Berne, Switzerland: Hans Huber Medical Publisher; 1976.

11. Ilizarov GA. The principles of the Ilizarov method. Bull Hosp Jt Dis Orthop Inst. 1988;48(1):1–11.
[PubMed]

12. Ilizarov GA. The tension-stress effect on the genesis and growth of tissues. Part I. The influence of stability of fixation and soft-tissue preservation. Clin Orthop Relat Res. 1989;238:249–81.

13. Ilizarov GA. The principles of the Ilizarov method. 1988. Bull Hosp Jt Dis. 1997;56(1):49–53.
[PubMed]

14. Schatzker J, Tile M. The rationale of operative fracture care. 3rd ed. Berlin: Springer; 2005.

15. Wiss DA, Stetson WB. Tibial nonunion: treatment alternatives. J Am Acad Orthop Surg. 1996;4(5):249–57.
[Crossref][PubMed]

16. Müller ME. Treatment of nonunions by compression. Clin Orthop. 1965;43:83.
[PubMed]

17. Dawson J, Kiner D, Gardner W 2nd, Swafford R, Nowotarski PJ. The reamer-irrigator- aspirator as a device for harvesting bone graft compared with iliac crest bone graft: union rates and complications. J Orthop Trauma. 2014;28(10):584–90.
[Crossref][PubMed]

18. Masquelet AC, Benko PE, Mathevon H, Hannouche D, Obert L. French Society of Orthopaedics and Traumatic Surgery. Harvest of cortico-cancellous intramedullary femoral bone graft using the reamer-irrigator-aspirator (RIA). Orthop Traumatol Surg Res. 2012;98(2):227–232.

19. Pape HC, Evans A, Kobbe P. Autologous bone graft: properties and techniques. J Orthop Trauma. 2010;24(Suppl 1):S36–40.
[Crossref][PubMed]

20. Quintero AJ, Tarkin IS, Pape HC. Technical tricks when using the reamer irrigator aspirator technique for autologous bone graft harvesting. J Orthop Trauma. 2010;24(1):42–5.
[Crossref][PubMed]

21. Schmidmaier G, Herrmann S, Green J, Weber T, Scharfenberger A, Haas NP, et al. Quantitative assessment of growth factors in reaming aspirate, iliac crest, and platelet preparation. Bone. 2006;39(5):1156–63.
[Crossref][PubMed]

22. Masquelet AC, Begue T. The concept of induced membrane for reconstruction of long bone defects. Orthop Clin N Am. 2010;41(1):27–37; table of contents.

23. Pelissier P, Masquelet AC, Bareille R, Pelissier SM, Amedee J. Induced membranes secrete growth

factors including vascular and osteoinductive factors and could stimulate bone regeneration. J Orthop Res. 2004;22(1):73–9.
[Crossref][PubMed]

24. Khurana S, Karia R, Egol KA. Operative treatment of nonunion following distal fibula and medial malleolar ankle fractures. Foot Ankle Int. 2013;34(3):365–71.
[Crossref][PubMed]

25. Carpenter CA, Jupiter JB. Blade plate reconstruction of metaphyseal nonunion of the tibia. Clin Orthop Relat Res. 1996;332:23–8.
[Crossref]

26. Kabata T, Tsuchiya H, Sakurakichi K, Yamashiro T, Watanabe K, Tomita K. Reconstruction with distraction osteogenesis for juxta-articular nonunions with bone loss. J Trauma. 2005;58(6):1213–22.
[Crossref][PubMed]

27. Feldman DS, Shin SS, Madan S, Koval KJ. Correction of tibial malunion and nonunion with six-axis analysis deformity correction using the Taylor spatial frame. J Orthop Trauma. 2003;17(8):549–54.
[Crossref][PubMed]

28. Richmond J, Colleran K, Borens O, Kloen P, Helfet DL. Nonunions of the distal tibia treated by reamed intramedullary nailing. J Orthop Trauma. 2004;18(9):603–10.
[Crossref][PubMed]

© Springer Science+Business Media LLC 2018
Animesh Agarwal (ed.), Nonunions, https://doi.org/10.1007/978-1-4939-7178-7_15

# 15. Special Techniques for Nonunions Associated with Traumatic Bone Loss

Mark A. Lee[1] and Michael P. Leslie[2]

(1) Department of Orthopedic Surgery, University of California, Davis, 4860 Y Street, Suite 3800, Sacramento, CA 95817, USA
(2) Department of Orthopedics and Rehabilitation, Yale School of Medicine, New Haven, CT 06520, USA

**Mark A. Lee**
**Email:** mark.lee@ucdmc.ucdavis.edu

**Keywords** Bone loss – Nonunion – Masquelet – Distraction osteogenesis – Tantalum – Vascularized bone graft

## 15.1 Introduction

The reconstruction of long bone defects is often a major challenge in limb salvage regardless of the etiology of bone loss . There is limited high-grade evidence on the efficacy of multiple techniques for bone regeneration and especially on comparative outcomes of different management techniques. The literature is rife with limited case series and single surgeon experiences that do not provide evidence-based treatment recommendations. Nonetheless, bone loss remains a common problem for clinicians, and multiple approaches are utilized depending on surgeon experience and resources. To a certain extent, defect size dictates many of the treatment approaches. Small, stabilized defects (2–3 cm) are frequently treated with acute cancellous autograft application. While this may be effective in favorable (i.e., well

vascularized) healing sites, this is not typically used for larger defects (greater than 4 cm). In large defects, the healing is unpredictable, and significantly, larger bone graft volumes are necessary which leads to concerns about graft absorption [1]. Therefore, in large defects, specialized approaches are required. The most common classic techniques are vascularized free bone transfer and Ilizarov bone transport. Both of these techniques require specialized training or equipment and a high level of surgical expertise in conjunction with postsurgical resources and support. Significant patient compliance and cooperation are required, and very large defects require protracted treatment times. Despite these limitations, these are powerful techniques for bone regeneration , and the results can be remarkable. In many situations, neither vascularized transfer nor bone transport is optimal or available, and novel techniques are being utilized. The induced membrane technique (Masquelet ) is increasingly utilized in bone defects to extend the application of cancellous grafting to larger defect sizes. This technique has been well reviewed in the literature [2–4] and is utilized with increasing frequency for massive defects. Even more novel approaches to defects include the use of spinal cages (for graft containment and structure support) and noncustom porous tantalum implants (for structural support and defect substitution). These more unique approaches await systematic evaluation but do provide solutions in recalcitrant cases.

## 15.2 Distraction Osteogenesis

The concept of distraction osteogenesis as a method of skeletal reconstruction can be traced back to as early as 1905. This has been done with a variety of methods including osteotomy and immediate traction, external fixation, internal fixation with either intramedullary rods or extramedullary osteosynthesis, and fine wire fixation. Significant bone loss occurs in a minority of fractures (0.4%) but that is significantly higher in cases of open fracture and when planned intervention demands resection of large segments of bone [5]. Each case of bone loss carries an individual character that is comprised of the patient demographics and comorbidities along with the injury itself. In the presence of an acute open fracture, the key concepts include soft tissue compromise and instability. In the case of tumor, the demands of cancer treatment and medical compromise of the patient have significant impact on the planned intervention for bone loss. Infection and

nonunion include both concepts of soft tissue compromise along with inflammation and bone loss. The ultimate treatment for any of these situations would include resection and bone grafting with a source that provides cortical stability and rapid integration into the human skeleton without risk of infection or rejection. Unfortunately, there are no current interventions that can achieve these idealistic goals. However, the use of distraction osteogenesis remains the closest to this potential gold standard. With the use of corticotomy and distraction, the donor source risk is minimized as the graft is a similar shape and has a robust soft tissue envelope providing biology to the healing environment.

Distraction osteogenesis refers to the formation of new bone between two ends of vascularized bone that are gradually distracted [6]. This can be accomplished through multiple methods of distraction. This concept was first introduced in 1905 by Codivilla, who performed the first successful limb lengthening by osteotomy and immediate transcalcaneal traction [7]. In 1913, Ombredanne reported the fist use of external fixation for distraction. This was improved upon by Putti in 1921 who utilized a monolateral fixator at a rate of 2–3 mm/day as opposed to the 5 mm/day used by the sentinel author [8]. These concepts have been applied to many different clinical scenarios with variable success and complication. After World War II, Ilizarov began to develop the concept of distraction through the use of fine wire attached to circular frames. This imparted stability and allowed for limb salvage for many limbs that would have otherwise undergone amputation (Fig. 15.1). This was done out of necessity as he faced a community of patients in Siberia where antibiotics were scarce, osteomyelitis was common, and amputation led to poor outcomes [6, 9]. The additional capacity to correct deformity while concurrently treating bone loss remains unparalleled; however, the technical challenges for the surgeon and the practical difficulties for the patient continue to limit the use of this technique. The current section discusses the use of fine wire circular fixation, unilateral rail distraction, distraction over intramedullary devices, and distraction with plate osteosynthesis as unique treatments for bone loss.

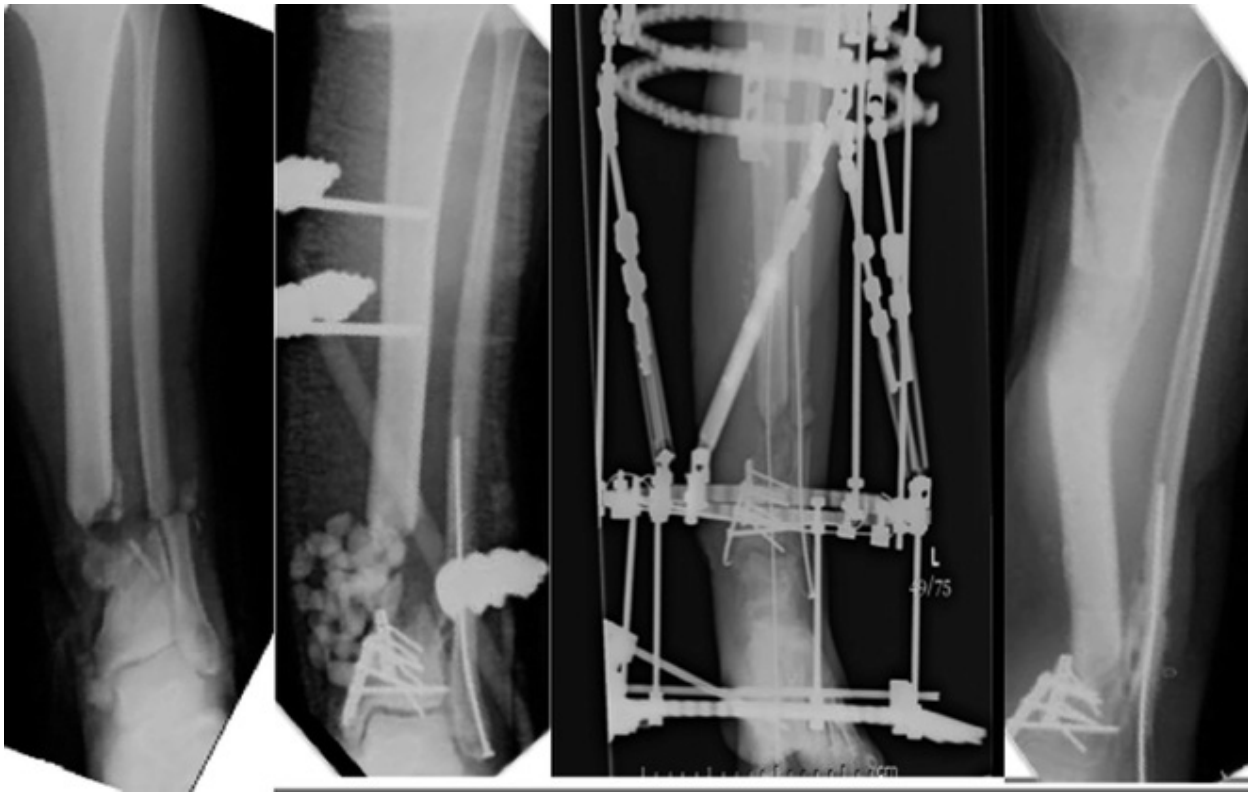


***Fig. 15.1*** 31-year-old male who suffered a moped accident with an isolated complex open intraarticular distal tibia and fibula fracture. He underwent staged management and with debridement and spanning external fixation, followed by open reduction internal fixation of the articular block and application of antibiotic impregnated beads until he healed a free latissimus flap. 5 cm of bone loss was then healed using a distraction osteogenesis technique with a proximal corticotomy in a multiplanar external fixator. The patient went on to consolidate the regenerate and heal the docking site without need for bone grafting, despite severe noncompliance with care. He currently walks without assistive device and has since had his distal tibial hardware removed due to a late infection due to shoewear breakdown of the free flap

## 15.2.1 Fine Wire Circular Fixation

External fixation has distinct advantages with respect to the ability to avoid direct instrumentation at sites of infected nonunions and also with the ability to slowly correct deformity, which potentially can limit the risk of injury to structures at risk. Fine wire circular fixation remains a powerful tool for both the correction of deformity and the application of distraction forces that allow for deposition of new bone. The most critical components linked to this remain to be the handling of the soft tissues during treatment (Fig. 15.2). The surgeon might choose the use of fine wire circular fixation in the setting of a nonunion that involves bone loss and angular deformity. All external fixator systems allow for multiple planes of freedom, but the use of fine wire circular

fixation is the only system that allows for both elastic control and dynamic control that respect bone biology. When an in-line or even multiplanar fixator is utilized with half pin fixation alone, there is not just control of length imparted but a distinct lack of control of angulation. This lack of control is considered “parasitic” to bony healing as it is uneven and nonbiologic. With the use of fine wire fixation, the stability that is imparted will allow for healing by secondary intention and callous formation but will at the same time limit the “parasitic” lack of control of angulation [10].

***Fig. 15.2*** Clinical photograph of a 38-year-old male who suffered a motorcycle collision with a complex Gustilo and Anderson type IIIB open proximal tibia fracture with 10 cm of proximal tibial bone loss. This patient required careful debridement, open reduction internal fixation, and massive autologous bone grafting using a Masquelet technique after a free flap successfully healed. He ambulates without assistive device at 2 years post-reconstruction

The more popularized understanding of fine wire fixation is that it can be used in conjunction with independent distraction–compression devices that will allow for multiplanar correction of deformity by application of compression in one plane and distraction in another.

The use of fine wire circular fixation has been successfully utilized in many clinical series as outlined above to achieve restoration of skeletal alignment and length. The cost and complexity associated with these types of systems can, however, be burdensome and has lead many surgeons to unilateral frames due to the ability to achieve skeletal success and simplify the process for both the surgeon and the patient.

In this technique, the nonunion site is debrided of all nonviable tissue and bone after the removal of any preexisting internal fixation devices. A unilateral frame can then be applied in a monofocal or bifocal method. In the

monofocal method, compression and distraction is initiated at the fracture site to stimulate osteogenesis. Distraction can then also be done at the nonunion site to restore leg length. If a bifocal method is done, the distraction is achieved outside of the nonunion site.

This is a widely used technique in all long bones. Harshwal et al. recently presented a series of 37 patients (7 femur and 30 tibias) all treated for nonunion within the first 8 months of the injury. Rate of union was reported at 91%. Minimal complications were noted, primarily those of pin-tract infections. These results are consistent with those reported by other authors [4, 11, 12].

## 15.2.2 Distraction Over Intramedullary Nails

### *15.2.2.1 Intramedullary Device Plus External Fixation*

Given the technical difficulties of controlling transport segments during distraction osteogenesis with purely external fixation , fine wire, or Schanz pin devices, the idea of guidance of the transport over intramedullary devices has become appealing. In addition, the angular deformities introduced by the use of a unilateral rail fixator alone, in conjunction with the inability to be fully weight bearing, have demanded the ability to guide a correction over an intramedullary device.

In a recent series, Gulabi altered the original descriptions of other authors to utilize acute compression and distraction osteogenesis. These patients were all tibial diaphyseal fractures with bone loss. Custom intramedullary nails were utilized with multiple locking hole options. In this technique, the bone loss site is cleared and a distant metaphyseal corticotomy is made that liberates a transport segment. The bone loss segment is shortened up to 5 cm, and the corticotomy site is compressed. The transport then proceeds at 2 mm/day, and when docking is achieved, the site is bone grafted from the iliac crest. Their results demonstrated radiographic union, no angular deformity, a moderate amount of pin site infections, and a 0.4 external fixation index (number of months external fixator system worn divided by centimeters of distraction) [13].

### *15.2.2.2 Telescopic Intramedullary Restoration of*

## *Length*

The problems associated with lengthening over an intramedullary nail are consistent with external fixation problems in general. These include pin-tract infections, scarring, pain, and patient comfort. In order to obviate these problems, several entirely intramedullary devices have been developed with the goal of using an internal lengthening mechanism to provide distraction osteogenesis. The intramedullary skeletal kinetic distractor (ISKD , Orthofix Inc., McKinney, TX, USA, and the PRECICE intramedullary nail (Ellipse Technologies, Irvine, CA, USA) utilize novel techniques of lengthening from within the canal (Fig. 15.3).

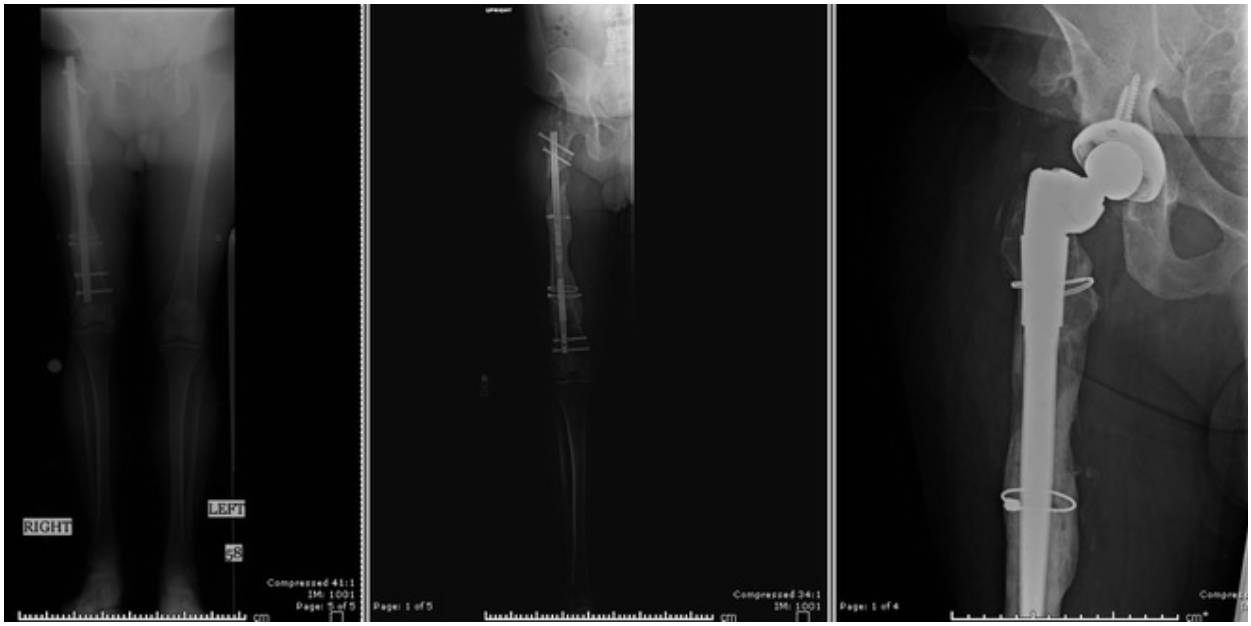



***Fig. 15.3*** 55-year-old male who underwent en bloc resection of the femur for malignant fibrous histiocytoma 20 years prior to presentation. The intercalary allograft femur had healed with limb foreshortening that lead to extensive back pain and hip arthritis. Staged management included restoration of standing balance with intramedullary nail extraction and application of an intramedullary telescopic nail with proximal corticotomy through native metaphysis. At 6 months, post-op patient was pain free at the *upper thigh* and underwent a total hip arthroplasty with concomitant removal of hardware at 1 year

The ISKD Nail utilizes two internal rotating clutches to advance a threaded rod within the nail that is attached to the distal segment beyond an osteotomy with interlocking bolts. This provides distraction that is based on typical activities of daily living that provide stimulus through 3–9 degrees of rotation through the osteotomy site. There have been many challenges with

this device including a lack of absolute control of distraction. This can be due to variable activities of patients, but can lead to a rate of distraction that is suboptimal, either too fast or slow [14, 15].

The PRECICE nail uses an externally applied magnetic device to control the lengthening. The proposed advantages to this include the ability to not only monitor the lengthening but also change the prescription of lengthening based on optimal conditions and the regenerate response time. There is less clinical evidence regarding this device but results appear similar to the ISKD with unique difficulties encountered [16, 17].

With respect to critical cortical defects and nonunion, these devices can be utilized for either compression of a fracture site or distraction osteogenesis. If a defect is predicted, this can be used to compress the fracture and then to perform an osteotomy and distract healthy bone to attain regenerate.

### 15.2.3 Distraction with Plate Osteosynthesis

The use of intramedullary nails in conjunction with external fixator distraction can be complicated by pin site infection that can develop into an intramedullary infection due to the proximity of the pins and the nail. It is also limited by the ability to apply transport to a proximal or distal fracture. Oh et al. [18] recently reported the use of locking plate stabilization with external fixator generated distraction osteogenesis. In their series of ten patients, a similar technique of corticotomy is performed, and after a latency period, distraction proceeded with 1 mm/day. When the docking site is achieved, the transport segment is stabilized with screw fixation through the plate, the docking site is grafted, and the external fixator is removed. All patients achieved radiographic union, and complications involve pin site infections only. Theoretically, these patients might be at higher risk for fracture of regenerate bone, although this has not occurred for them at the time of publication. The primary advantage is the ability to stabilize the transport segment and remove the external fixator despite a lack of radiographic union. The disadvantage is theoretically the lack of loadbearing the plate can contribute. However, the advantages of being able to apply this technique to skeletally immature patients, large amount of bone available for placement of external fixation, and decreased time to removal of external fixation can outweigh these disadvantages (Fig. 15.4).

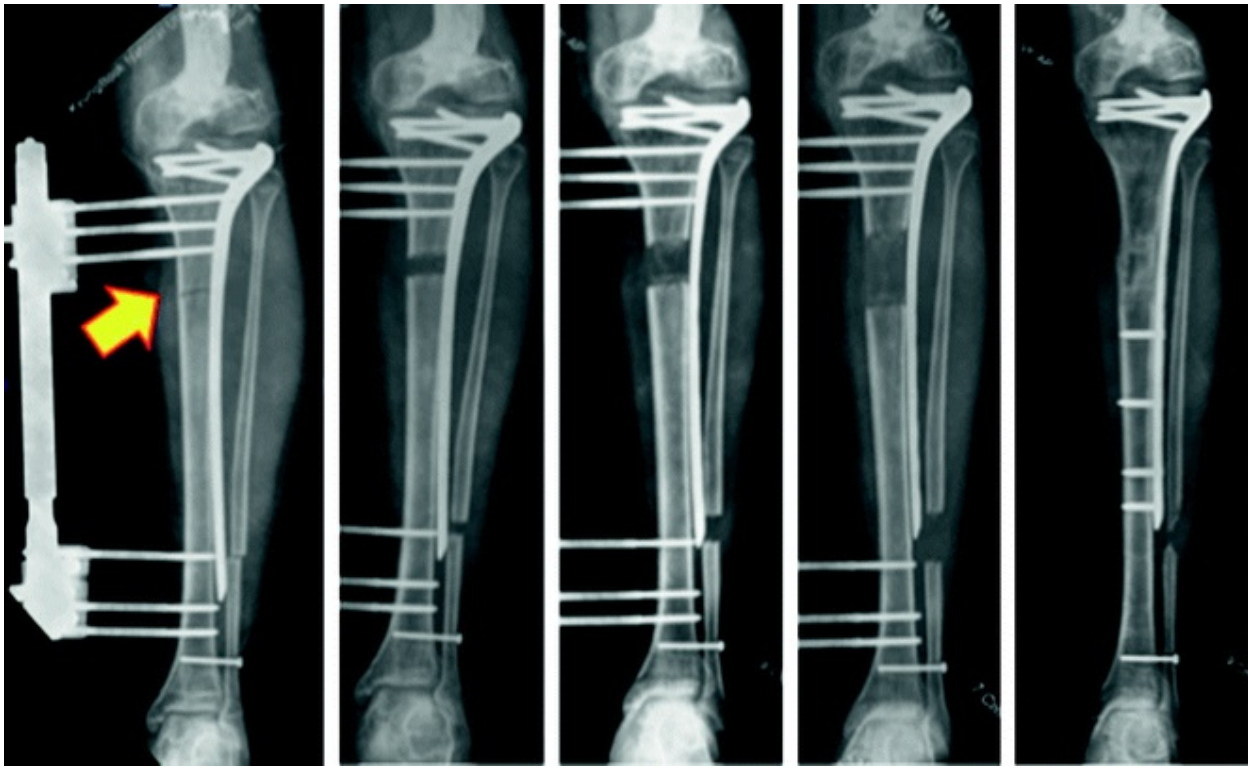


***Fig. 15.4*** 14-year-old male who underwent resection for osteosarcoma with limb foreshortening and flexion contracture of the knee. Distraction with plate osteosynthesis utilized with proximal tibial corticotomy highlighted (*yellow arrow)* to the *left*. External fixator removed at 7 weeks and consolidate locked into the plate construct distally. Allowed for 4.6 cm of distraction in 63 days of external fixation. Consolidation of regenerate noted to be complete by 4 months *on the right*. (Courtesy of Chang-Wug Oh, MD, Kyungpook National University Hospital, Daegu, Korea)

## 15.3 Masquelet Technique

The induced membrane technique is a unique alternative to acute bulk grafting. This technique was originally utilized for regeneration of diaphyseal defects, but use has been expanded to metaphyseal defects as well. Professor Masquelet developed the technique in early 1984 and soon after initiated a clinical study to demonstrate its efficacy [2] .

### Key Features

- A bioactive membrane is created by placement of a Poly(methyl methacrylate) (PMMA) block into a clean, debrided defect (Fig. 15.5).

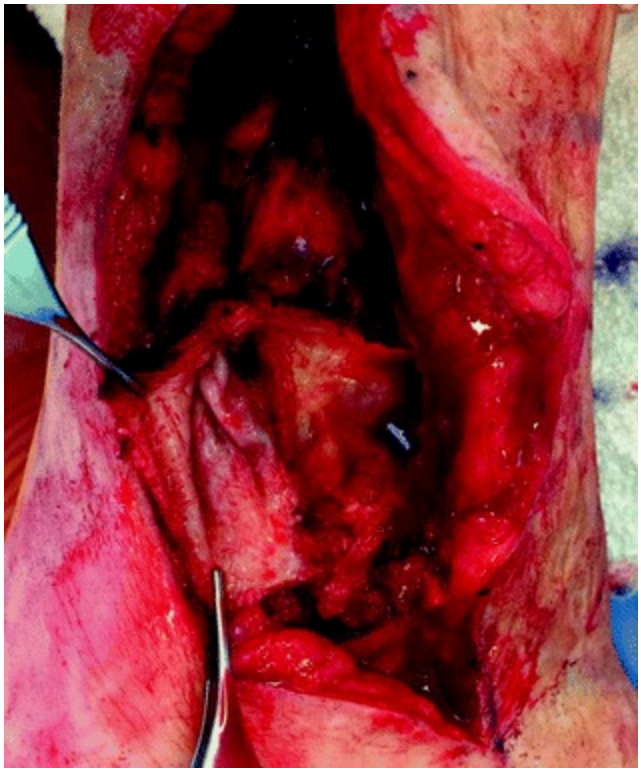


***Fig. 15.5*** The forceps are holding the induced membrane which has been opened longitudinally and provides vascularized pouch for graft material

- The blood supply around the induced membrane is left intact or optimized by free tissue transfer.
- The induced membrane is incised, and the PMMA block is carefully removed, leaving the membrane intact as a protective and supportive grafting bed.
- Slow consolidation is observed, and weight bearing is restricted until union [2].

## 15.3.1 Membrane

The induced membrane is believed to be a unique property of this technique and critical to its success. Extensive animal evaluations in both small and medium animal models have demonstrated the membrane is made of a type I collagen-heavy matrix and fibroblastic cells. The membrane itself has tissue level organization with an inner aspect of epithelial-like fibroblasts and collagen bundles that run parallel to the surface of the membrane. This tissue is well vascularized and contains a high concentration of vascular endothelial

growth factor. Typically, a solid block of PMMA is used to produce the spacer; this induces a mild foreign-body inflammatory response with giant cells and macrophages. The inflammatory response slowly decreases over time following spacer implantation may disappear by 6 months following bone grafting. Tissue from these membranes has been analyzed using molecular techniques including immunohistochemistry, and these studies demonstrate expression of proteins associated with induction of new bone formation. Thus, many feel that these membranes are bioactive. In addition, the induced membrane also acts to eliminate soft tissue interposition into defects and created a protective cavity to accept bone graft. The shape and size of the healed bone graft are defined by the membrane [2, 19–22].

## 15.3.2 Technique

By definition, this is a two-stage technique. The first stage is akin to a tumor debridement with aggressive removal of nonviable bone, scar, and any damaged or nonviable local soft tissues. The bone debridement cannot be limited since frequently bone necrosis at the fracture edges has progressed significantly proximal to the defect. After debridement/resection, the remaining bone ends should be healthy with a viable bleeding bed (Fig. 15.6). In the setting of a severe soft tissue deficit or wound problem, standard dead space management techniques using PMMA bead strands can be used, while the preliminary wound management is performed. Open wounds can be managed with negative pressure therapy or bead pouch depending on the individual patient need. Once the soft tissue bed is clean and mature, the definitive solid spacer can be placed with simultaneous muscle coverage.

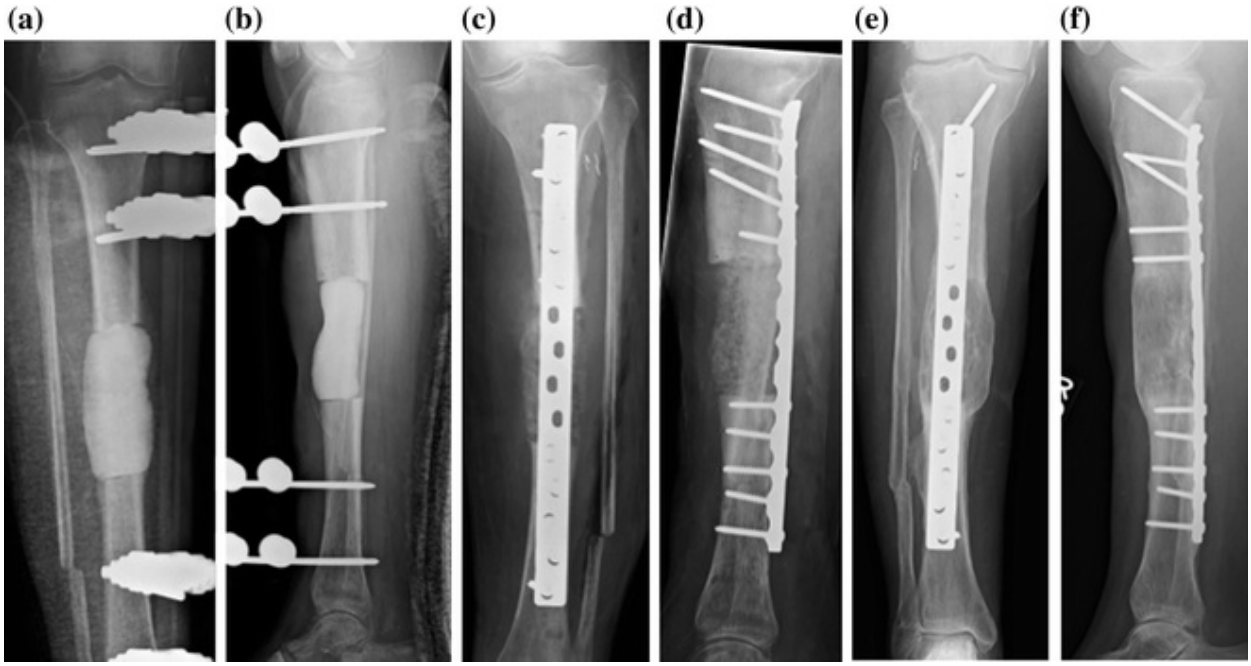



***Fig. 15.6*** Diaphyseal infection undergoes aggressive resection and debridement. **a, b** The defect is filled with PMMA, and preliminary stabilization is achieved with external fixation. **c, d** Classically, the ends of the bone are over wrapped with PMMA. At 8 weeks, the wound is filled with cancellous autograft and BMP and formal plate fixation is utilized. **e, f** At 6 months, the regenerate is completely healed and the patient is weight bearing

When feasible, intramedullary reaming is performed ot aid in the debridement of the intramedullary canal and to stimulate an endosteal healing response. For optimum membrane induction and better stability of the construct, the cement should be placed inside the canal (when feasible) and over the edges of the native bone (wrapping) and should fill the space of defect. While external fixation was utilized in the original technique, more stable forms of internal fixation are typically utilized, even intramedullary nails. Use of intramedullary nails can decrease required graft volumes and provide long-term stability in these slowly healing constructs. Finally, optimal soft tissue blood supply is requisite around the induced membrane zone. Free tissue transfer is far optimal to a tight primary wound closure especially in the mid-to-distal tibia.

## 15.3.3 Outcomes

The original Masquelet series of 35 patients with upper and lower extremity segmental defects that measured 4–25 cm in length reported a 100% healing rate. Most of these were treated with external fixation and many had free

flaps. The mean time to full weight bearing was 8.5 months [23]. While this series is impressive, it likely does not represent contemporary use of the technique. Subsequent reports have included the use of bone morphogenetic protein (BMP) , reamed intramedullary grafts, and multiple modes of internal fixation—for most of these techniques, ultimate union rates hover around 90% [2, 24, 25]. While many of these publications report good results, overall the level of evidence for this technique remains low since these are mostly retrospective case series or small prospective noncomparative studies.

### 15.3.4 New Considerations

The timing of bone grafting into the membrane has been recently evaluated [26]. While contemporary approaches demonstrate large variability in timing of secondary cancellous grafting into the membrane bed, most surgeons delay 6 weeks or more after placement of the spacer. A closer evaluation of one of the original animal studies demonstrated the time course of growth factor expression from induced membrane samples with quantitative and qualitative immunohistochemistry [20]. Maximum BMP-2 levels were seen at 4 weeks post-procedure with decrease over subsequent weeks. These data may suggest that the optimal time of membrane bioactivity is earlier than suspected. Samples of human induced membrane tissue were assayed for multiple time points. One-month-old membrane samples had the highest expression of VEGF, IL-6, and Col-1, whereas two-month-old membranes expressed <40% of the levels of the one-month-old membranes [26]. This study suggests a time-dependent decrease in bioactivity of the membrane and may suggest a role for earlier secondary grafting. So in the absence of definitive evidence for specific timing, grafting can be safely performed as soon as the wounds have healed well without evidence of residual infection and systemic antibiotic therapy is near complete (4–6 weeks). There is likely little benefit to protracted delays (greater than 8 weeks) to secondary graft application.

---

## 15.4 Cage Technique

In 2002, Ostermann published the first reports of extending the indication for use of titanium mesh cages to restore bony continuity [27]. These devices are routinely utilized in spine surgery to augment the use of nonstructural

allograft. They have demonstrated adequate ability to achieve bony union in conjunction with bone graft [28, 29]. The goal of utilizing the titanium cage is that cancellous allograft and demineralized bone matrix products offer advantages of no donor-site morbidity and ease of application. The difficulty in utilization of nonstructural allograft bone is that it does not reliably lead to bony union in gaps greater than 3 cm, those of critical cortical defects. The addition of the titanium mesh cage extends the application of the allograft material by imparting additional stability.

The technique involves either plate or intramedullary nail stabilization. It can be performed either acutely, on a delayed basis or in a nonunion setting. In each case, the cage is premeasured in accordance with the diameter of the bone and also the length of the defect to be spanned. The cage is prepared with a packing that consists of cancellous bone graft, and if an intramedullary nail is to be used, the guide wire is passed through the middle to ensure that there is no mechanical blockade to passage. Standard intramedullary nailing techniques can then be utilized including reaming over a guide wire (Fig. 15.7). Ostermann, Attias, and Cobos all reported success in small series with minimal complication, most notably in leg length discrepancy [27, 30–32].

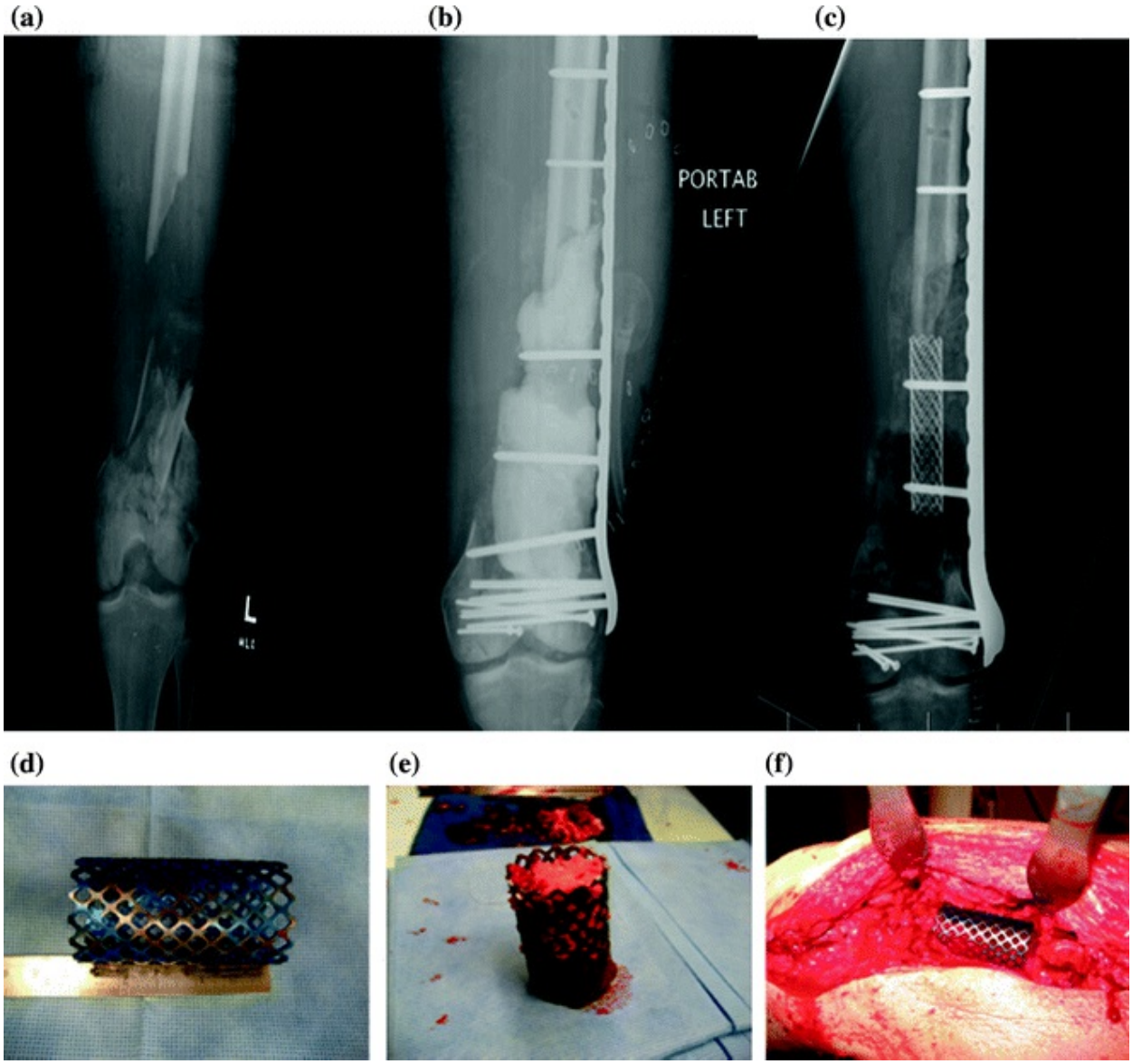



***Fig. 15.7*** 31-year-old male motorcyclist who suffered complex intraarticular distal femur fracture with extensive bone loss. **a**–**c** Patient underwent initial Masquelet technique after extensive debridement and bone grafting was assisted by the integration of a titanium mesh cage supported by screw fixation through the plate and massive autologous and allogeneic bone graft. **d**–**f** The cage, demonstration of packing the cage with bone graft, and a clinical photograph demonstrate the technique. (The cage images courtesy of Brian J. Cross, DO, Broward Health Medical Center, Plantation FL, USA)

In some situations, plate osteosynthesis might be the preferred method. Attias recommended plate osteosynthesis in the setting of nerve exploration or when intramedullary nailing might be suboptimal such as a proximal or distal metaphyseal segments. The same methodology of preparation was performed in the single case report using this method, and the cage was

implanted and compressed into the bone ends of a humeral fracture associated with a gunshot wound. They suggested the use of orthogonal plating to impart greater stability and allow for early motion [30].

## 15.5 Metal Tantalum for Defects

The use of metal alloys for structural substitution is an atypical technique and reserved for situations where regeneration is unfeasible, unlikely, or the patient declines other techniques. Any of these applications would certainly be considered “off-label” techniques since none of the currently available tantalum devices are intended for trauma applications.

### 15.5.1 Material

Tantalum is a transition metal (atomic number 73; atomic weight 180.05) that remains relatively inert in vivo. Porous tantalum is an open-cell tantalum structure of repeating dodecahedrons with an appearance similar to cancellous bone has been developed for clinical applications. (Zimmer-Biomet, Trabecular Metal Technology, Inc., Parsippany, NJ, USA). The basic structure of this porous tantalum metal yields a high volumetric porosity, a low modulus of elasticity, and relatively high frictional characteristics [33]. This frictional characteristic makes immediate stable interfaces with bone feasible and allows the potential for early or immediate weight bearing (Fig. 15.8) [34].

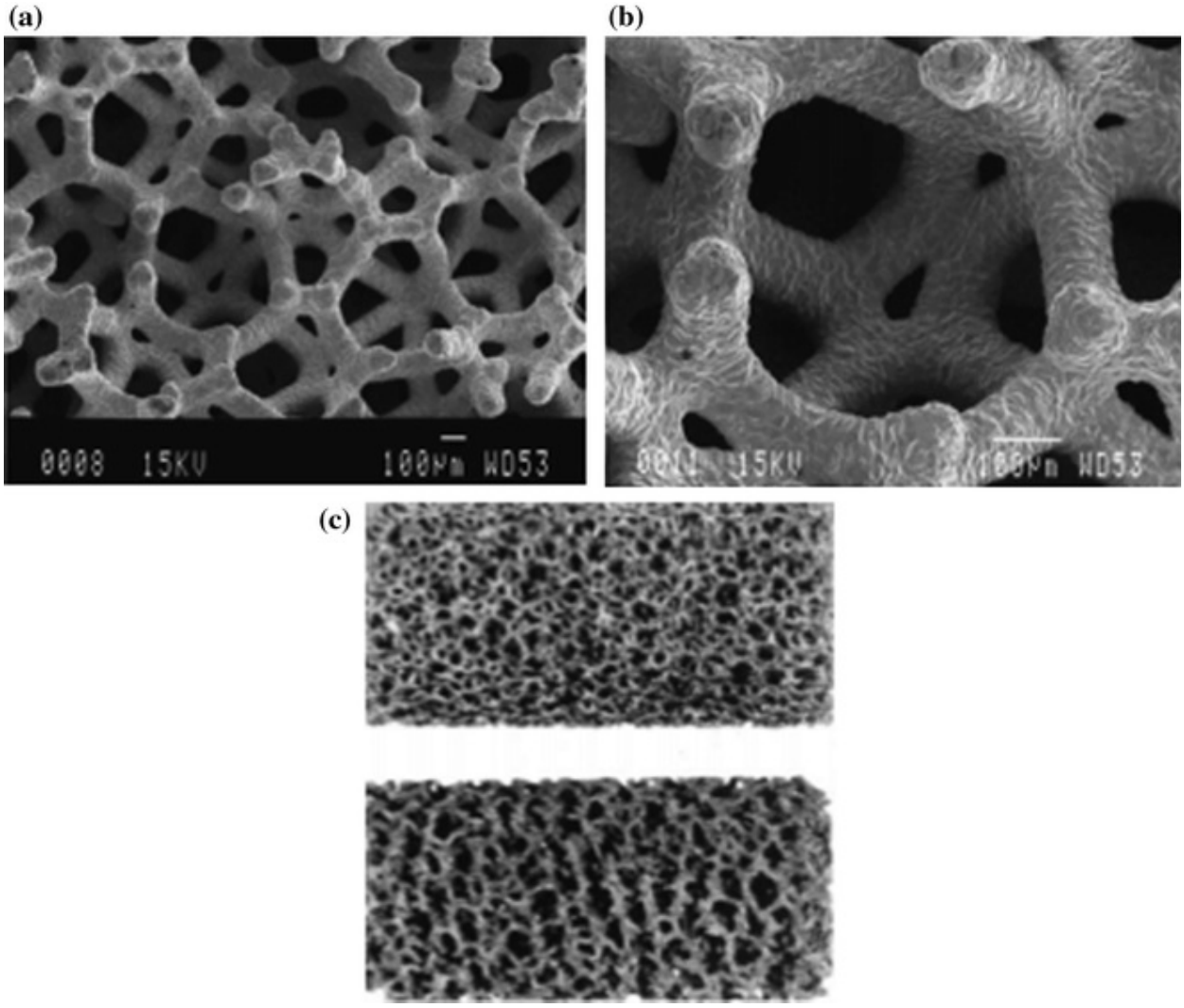



***Fig. 15.8*** **a** Scanning electron micrograph of porous tantalum showing the cellular structure formed by the tantalum struts. There is the occasional smaller opening or portal that interconnects with the larger pores or cells. **b** Higher power scanning electron micrograph of a single pore illustrating the surface microtexture on the struts caused by crystal growth during the process of tantalum deposition. **c** Photographs showing transcortical implants with small and large pore sizes (From Bobyn et al. [34] with permission of The British Editorial Society of Bone & Joint Surgery)

Porous tantalum structures utilized for orthopedic implants have a porosity of 75–85% compared to CoCr sintered beads (30–35%) [35]. The rigidity of porous tantalum increases with decreasing porosity. Current tantalum implants maintain a rigidity similar to the human fibula [36]. These characteristics optimize the biocompatibility of these implants.

In addition to its high biocompatibility, the frictional characteristics and the rigidity similarity to native bone make porous tantalum an intriguing candidate for defect management. With tantalum implants in structural

defects, stable implantation, structural support, and limited local stress shielding are feasible.

## 15.5.2 Bone Ingrowth Potential

The porosity of current tantalum implants has been designed to optimize bone ingrowth potential [37]. A recent in vivo study sought to evaluate the interaction between human osteoblasts and porous tantalum and convincingly demonstrated that porous tantalum is a good substrate for the attachment, growth, and differentiated function of human osteoblasts.

The current tantalum implants used for defects are designed for bone defect management around joint replacements. While not primarily designed for trauma, many of the shapes have been adaptable to the shape of common diaphyseal and meta-diaphyseal defects (Fig. 15.9).

***Fig. 15.9*** Multiple different tantalum implants that can be fashioned for critical bone loss substitution

## 15.5.3 Applications

Tantalum can be used for reconstruction of diaphyseal defects of large size. Our experience has been primarily in knee arthrodesis in conjunction with an intramedullary device for stabilization. This size defect will require multiple implants used end to end but allows for full defect reconstruction and immediate weight bearing (Fig. 15.10).

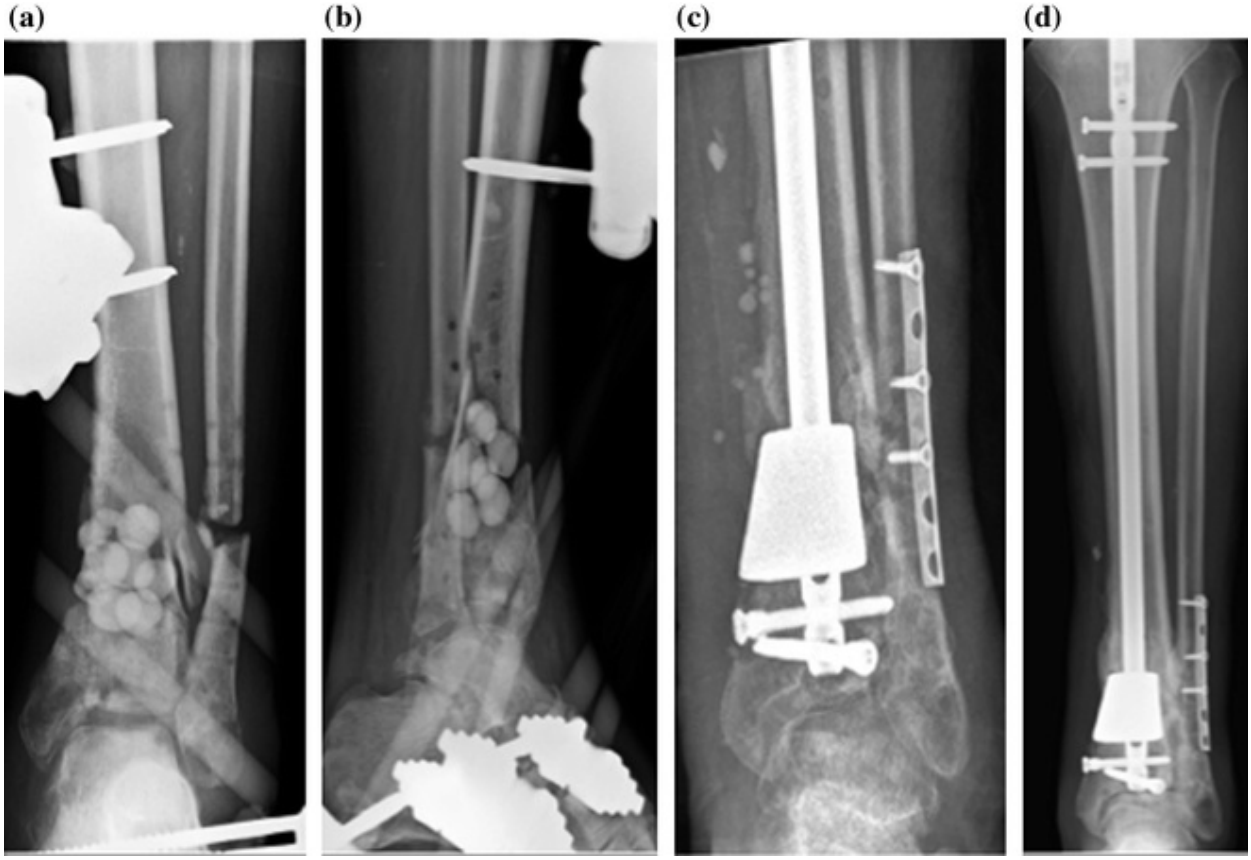



***Fig. 15.10*** This patient had purulent infection after ORIF distal tibia fracture with articular extension. **a, b** He underwent radical resection of infection and had antibiotic beads placed after moderate bone resection was required. **c** After 6 weeks of intravenous antibiotics and replacement of beads with a solid spacer, he had removal of his antibiotic spacer, placement of an intramedullary nail through a tantalum spacer, and autogenous cancellous grafting. **d** At 5 months, he is free of infection with full incorporation of tantalum implant

**Metaphyseal**. We have used tantalum most frequently in the setting of metaphyseal bone loss—for both complete and incomplete defects. Metaphyseal reconstructions can be done with either a plate of an intramedullary device for stabilization. Defects can be modified to accept the flat ends of the implant, and load can be applied. The most common indication for tantalum is critical size bone defect in patients who have

declined traditional approaches to bone regeneration (bulk grafting or distraction osteogenesis), patients who cannot comply with limited weight bearing postsurgically, and patients with poor bone regeneration potential (elderly, systemically ill). *Patients must be informed that this is an off-label application*.

**Outcomes**. There are no published outcomes of using tantalum for defect reconstruction. There are ongoing concerns about risk of infection with the use of tantalum in traumatic wounds and the required resection in the setting of a fulminant infection. However, in theory, this approach is not significantly different than the currently utilized induced membrane technique, which utilizes a PMMA spacer. If an infection were to occur, there is likely formation of a vascularized scar response around the implant that could ultimately be grafted.

---

## 15.6 Tissue Transfer

Except for distraction osteogenesis, the other techniques described above involve the use of bulk bone grafting that provides an avascular healing zone that requires creeping substitution with cells migrating from the intact bone through the matrix. The risk of nonunion, fracture of the transplanted bone, and overall poor microarchitecture of the healed environment places the patient at high risk. This can be obviated by distraction osteogenesis or vascularized tissue/bone transfer. The maintenance of periosteal and endosteal blood supply allows for healing and remodeling through both the vascular pedicle and the local supporting vasculature with osteoblast induction .

**Fibula**. The vascularized fibula (pedicled or free) is the most well-studied of all vascularized bone grafts in long bone loss. The fibula as a bony anatomic unit is quite versatile as it is similar to the radius and ulna in shape and size, can be used intramedullary in the humerus and can even be medialized to substitute for the tibia. The vascular supply is from the peroneal artery and veins, which provide a dual endosteal and periosteal supply from both the nutrient artery and the musculo-periosteal vessels [38]. It can be utilized as a purely osseous, or with the overlying skin and muscle depending on the amount of type of bone and soft tissue loss associated with the injury. One of the disadvantages of

utilization of the vascularized fibula is the small caliber of the bone, which can be compensated for with the double barrel technique that allows for the long fibula donor (which can be a maximum of 26 cm in length). Modifications such as this allow for broad application of the graft with the only limitation being the technical nature of the surgical harvest and implantation requiring a skilled microvascular surgeon [38, 39]. Outcomes of use of the vascularized fibula in the upper extremity demonstrate excellent incorporation at 3 months. In the lower extremity the fibula can be applied to the foot and ankle in standard fashion if there is an adequate location for anastomosis outside the zone of injury, but in the tibia different techniques might be used (Fig. 15.11). Medialization of the fibula to substitute for segmental bone loss can be performed primarily or with Ilizarov techniques, but must at all times account for the condition of the soft tissues [40] .

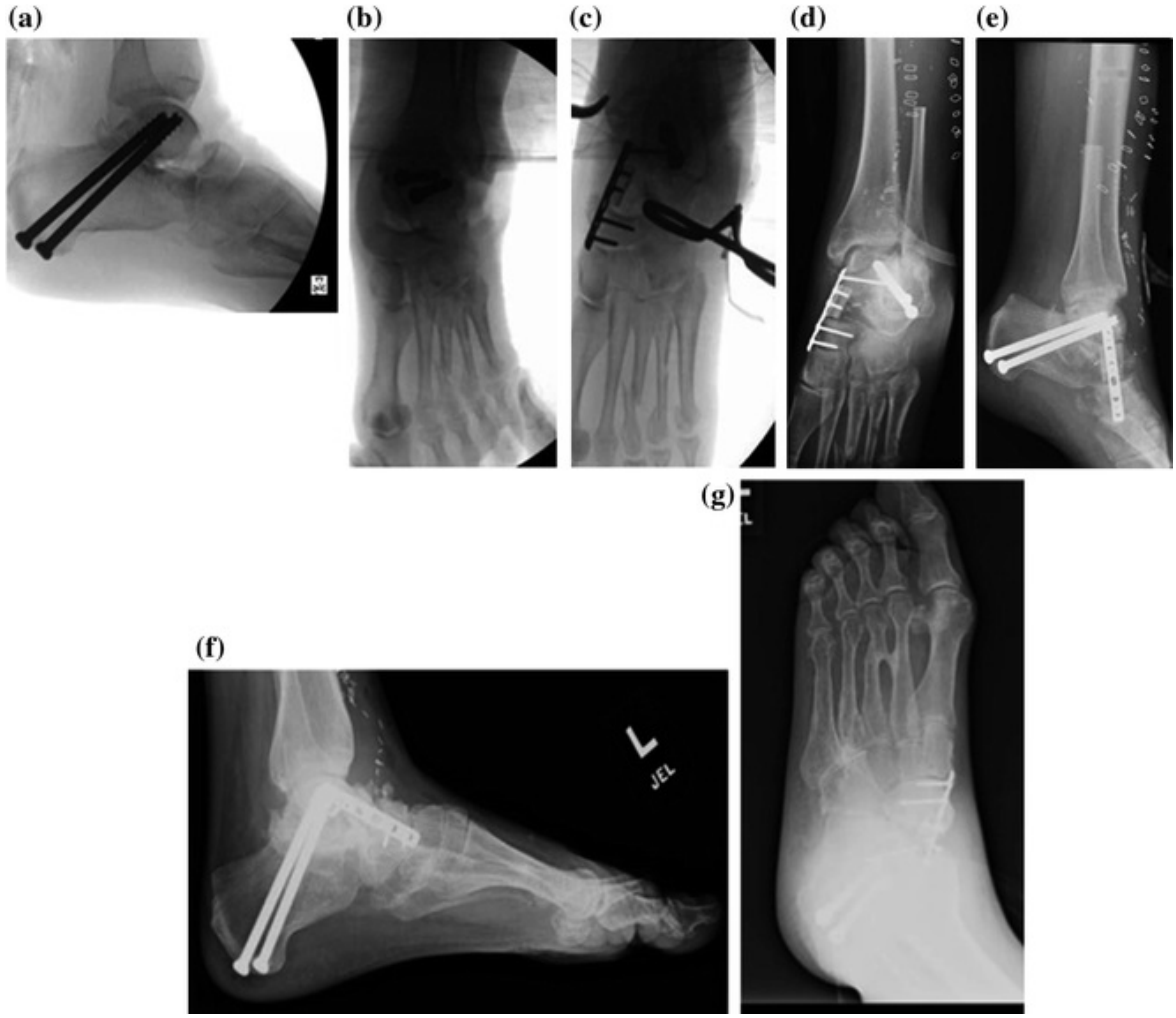


***Fig. 15.11*** 55-year-old female injured in a fall from a burning building, with a complex open fracture dislocation of the hindfoot. Complete traumatic loss of the talar head at the talonavicular joint. **a**–**e** Primary subtalar arthrodesis performed along with vascularized free fibula transfer to achieve both soft tissue coverage and a talonavicular arthrodesis. **f**–**g** Radiographs at 1.5 years demonstrate bony healing with a nonantalgic gait and complete return to function

**Rib**. Defects of the clavicle are rare but difficult to heal lesions that can be associated with longstanding nonunions. They occur in patients who often have had multiple failed procedures and there is no single answer to the reconstruction of these defects. Traditionally tricortical iliac crest with compression has been the standard of care with variable success rates. The advantage of this technique is the relative simplicity and ability to reconstructs small defects to equalize the affected clavicle to the length of the contralateral side. Larger defects may benefit from both a vascularized bone graft and compression. A free pedicled transfer is less than ideal in this region of the body. Free transfer of a vascularized rib pedicle has been utilized for mandibular, maxillary and extremity defects (tibial, calcaneal and humeral) [41]. This graft has also been studied in a rotational manner for the clavicle where a serratus anterior flap is taken with the seventh and eighth rib and tunneled under the pectoral musculature then embedded into the debrided clavicle with compression fixation. This is done in a double barreled fashion that allows for adequate strength [42]. In a few case studies this has demonstrated long term success and although technically challenging this technique can provide both mechanical and biologic advantages for the patient .

**Medial Femoral Condyle**. The medial femoral condyle has been extensively studied for small defects as it is taken as a cortico-periosteal graft that is supplied by the descending genicular artery. Traditionally this was described as a thin, non-biomechanically strong graft that is easily isolated and transferred into a site of defect with cancellous bone harvested most commonly from the iliac crest. The harvest site is highly reliable and it provides an answer for small areas of necrotic or missing bone (Fig. 15.12). The average size of flap is 5 cm in length; however; recent studies have demonstrated the potential harvest site to be as much as 13 cm [43, 44].

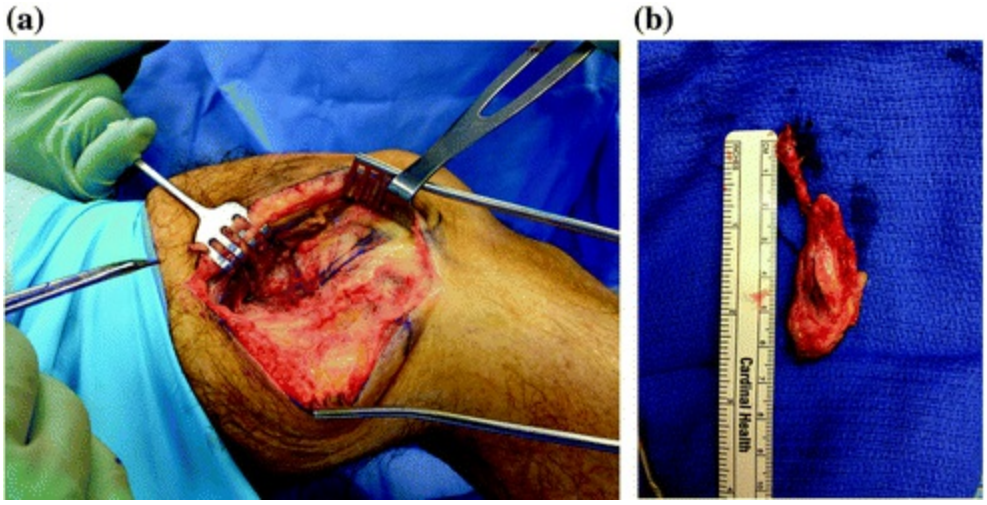



***Fig. 15.12*** The medial femoral condyle-free vascularized graft is ideal for small areas of necrotic bone. The harvest site is easily identifiable (**a**) and yields a graft that can be interposed into areas of bony loss (**b**), particularly useful in the distal aspects of the extremities. (Image courtesy of John S. Reach, Jr., MSc, MD, Yale University School of Medicine, New Haven CT, USA)

Other sources of vascularized bone transport will likely be identified in the future. There have been limited reports of others including a vascularized pelvic flap for calcaneal substitution [45]. These reports are limited case series or single case reports, but all identify the value of a vascularized graft particularly in the post-radiation, recalcitrant nonunions, and necrotic bone loss patients.

## 15.7 Summary

Nonunion care requires significant thought and precision with respect to achieving a sterile zone of injury along with an adequate understanding of the causative factor in a failure of bony regeneration. The occurrence of bone loss is not at all uncommon with respect to nonunion, and when critical cortical defects occur beyond 4 cm, special techniques must be employed to achieve complete reconstruction and return to function for patients.

For massive defects, especially in the setting of current or prior infection, distraction osteogenesis remains as the technique of choice for regeneration.

For large diaphyseal or metaphyseal defects, especially with no evidence of infection and stable fixation constructs, an induced membrane technique with cancellous grafting can be safely utilized. Metal cages can be used during reconstruction to contain the cancellous graft, provide stability benefit,

and potentially improve graft efficiency.

More unique approaches can be utilized in more challenging cases. Metal substitution is an alternative when patients decline other approaches to critical size defects or cannot comply with weight bearing limitations. Vascularized bone transfer is an alternative at centers that have microvascular expertise and in settings where the local blood supply will likely not support vigorous osteogenesis.

BMP with allograft cancellous chips remains an alternative for small to medium size defects, especially in the setting of a diaphyseal defect treated with an intramedullary rod. However, the quality of bone regenerate created by this approach and potential local inflammatory consequences limit the use of BMPs as a first-line approach.

---

# References


1. Stevenson S, Emery SE, Goldberg VM. Factors affecting bone graft incorporation. Clin Orthop Relat Res. 1996;324:66–74.
[Crossref]

2. Masquelet AC, Begue T. The concept of induced membrane for reconstruction of long bone defects. Orthop Clin North Am. 2010;41(1):27–37.
[Crossref][PubMed]

3. Taylor BC, French BG, Fowler TT, Russell J, Poka A. Induced membrane technique for reconstruction to manage bone loss. J Am Acad Orthop Surg. 2012;20(3):142–50.
[Crossref][PubMed]

4. Harshwal RK, Sankhala SS, Jalan D. Management of nonunion of lower-extremity long bones using mono-lateral external fixator–report of 37 cases. Injury. 2014;45(3):560–7.
[Crossref][PubMed]

5. Keating JF, Simpson AH, Robinson CM. The management of fractures with bone loss. J Bone Joint Surg Br. 2005;87(2):142–50.
[Crossref][PubMed]

6. Aronson J. Limb-lengthening, skeletal reconstruction, and bone transport with the Ilizarov method. J Bone Joint Surg Am. 1997;79(8):1243–58.
[Crossref][PubMed]

7. Codivilla A. On the means of lengthening, in the lower limbs, the muscles and tissues which are shortened through deformity. 1904. Clin Orthop Relat Res. 1994;301:4–9.

8. Putti V. The operative lengthening of the femur. 1921. Clin Orthop Relat Res. 1990;250:4–7.

9. Green SA. Ilizarov method. Clin Orthop Relat Res. 1992;280:2–6.

10. Catagni MA. Treatment of tibial nonunions. In: Bianchi Maiocchi A, editor. Treatment of fractures, nonunions, and bone loss of the tibia with the Ilizarov method. 4th ed. Milan: Medi Surgical Video; 2007. p. 1–8.

11. De Bastiani G, Aldegheri R, Renzi BL. Dynamic axial fixation. A rational alternative for the external fixation of fractures. Int Orthop. 1986;10(2):95–9.
[Crossref]

12. Marsh JL, Nepola JV, Meffert R. Dynamic external fixation for stabilization of nonunions. Clin Orthop Relat Res. 1992;278:200–6.

13. Gulabi D, Erdem M, Cecen GS, Avci CC, Saglam N, Saglam F. Ilizarov fixator combined with an intramedullary nail for tibial nonunions with bone loss: is it effective? Clin Orthop Relat Res. 2014;472(12):3892–901.
[Crossref][PubMed][PubMedCentral]

14. Mahboubian S, Seah M, Fragomen AT, Rozbruch SR. Femoral lengthening with lengthening over a nail has fewer complications than intramedullary skeletal kinetic distraction. Clin Orthop Relat Res. 2012;470(4):1221–31.
[Crossref][PubMed]

15. Lee DH, Ryu KJ, Song HR, Han SH. Complications of the intramedullary skeletal kinetic distractor (ISKD) in distraction osteogenesis. Clin Orthop Relat Res. 2014;472(12):3852–9.
[Crossref][PubMed][PubMedCentral]

16. Kirane YM, Fragomen AT, Rozbruch SR. Precision of the PRECICE internal bone lengthening nail. Clin Orthop Relat Res. 2014;472(12):3869–78.
[Crossref][PubMed][PubMedCentral]

17. Schiedel FM, Vogt B, Tretow HL, Schuhknecht B, Gosheger G, Horter MJ, et al. How precise is the PRECICE compared to the ISKD in intramedullary limb lengthening? Reliability and safety in 26 procedures. Acta Orthop. 2014;85(3):293–8.
[Crossref][PubMed][PubMedCentral]

18. Oh CW, Apivatthakakul T, Oh JK, Kim JW, Lee HJ, Kyung HS, et al. Bone transport with an external fixator and a locking plate for segmental tibial defects. Bone Joint J. 2013;95-B(12):1667–1672.

19. Viateau V, Bensidhoum M, Guillemin G, Petite H, Hannouche D, Anagnostou F, et al. Use of the induced membrane technique for bone tissue engineering purposes: animal studies. Orthop Clin North Am. 2010;41(1):49–56.
[Crossref][PubMed]

20. Pelissier P, Masquelet AC, Bareille R, Pelissier SM, Amedee J. Induced membranes secrete growth factors including vascular and osteoinductive factors and could stimulate bone regeneration. J Orthop Res. 2004;22(1):73–9.
[Crossref][PubMed]

21. Viateau V, Guillemin G, Bousson V, Oudina K, Hannouche D, Sedel L, et al. Long-bone critical-size defects treated with tissue-engineered grafts: a study on sheep. J Orthop Res. 2007;25(6):741–9.

[Crossref][PubMed]

22. Viateau V, Guillemin G, Calando Y, Logeart D, Oudina K, Sedel L, et al. Induction of a barrier membrane to facilitate reconstruction of massive segmental diaphyseal bone defects: an ovine model. Vet Surg. 2006;35(5):445–52.
[Crossref][PubMed]

23. Masquelet AC, Fitoussi F, Begue T, Muller GP. Reconstruction of the long bones by the induced membrane and spongy autograft. Ann Chir Plast Esthet. 2000;45(3):346–53.
[PubMed]

24. Stafford PR, Norris BL. Reamer-irrigator-aspirator bone graft and bi Masquelet technique for segmental bone defect nonunions: a review of 25 cases. Injury. 2010;41(Suppl 2):S72–7.
[Crossref][PubMed]

25. McCall TA, Brokaw DS, Jelen BA, Scheid DK, Scharfenberger AV, Maar DC, et al. Treatment of large segmental bone defects with reamer-irrigator-aspirator bone graft: technique and case series. Orthop Clin North Am. 2010;41(1):63–73.
[Crossref][PubMed]

26. Aho OM, Lehenkari P, Ristiniemi J, Lehtonen S, Risteli J, Leskela HV. The mechanism of action of induced membranes in bone repair. J Bone Joint Surg Am. 2013;95(7):597–604.
[Crossref][PubMed]

27. Eck KR, Bridwell KH, Ungacta FF, Lapp MA, Lenke LG, Riew KD. Analysis of titanium mesh cages in adults with minimum two-year follow-up. Spine (Phila Pa 1976). 2000;25(18):2407–15.

28. Hertlein H, Mittlmeier T, Piltz S, Schurmann M, Kauschke T, Lob G. Spinal stabilization for patients with metastatic lesions of the spine using a titanium spacer. Eur Spine J. 1992;1(2):131–6.
[Crossref][PubMed]

29. Ostermann PA, Haase N, Rubberdt A, Wich M, Ekkernkamp A. Management of a long segmental defect at the proximal meta-diaphyseal junction of the tibia using a cylindrical titanium mesh cage. J Orthop Trauma. 2002;16(8):597–601.
[Crossref][PubMed]

30. Attias N, Lehman RE, Bodell LS, Lindsey RW. Surgical management of a long segmental defect of the humerus using a cylindrical titanium mesh cage and plates: a case report. J Orthop Trauma. 2005;19(3):211–6.
[Crossref][PubMed]

31. Attias N, Lindsey RW. Case reports: management of large segmental tibial defects using a cylindrical mesh cage. Clin Orthop Relat Res. 2006;450:259–66.
[Crossref][PubMed]

32. Cobos JA, Lindsey RW, Gugala Z. The cylindrical titanium mesh cage for treatment of a long bone segmental defect: description of a new technique and report of two cases. J Orthop Trauma. 2000;14(1):54–9.
[Crossref][PubMed]

33. Levine BR, Sporer S, Poggie RA, Della Valle CJ, Jacobs JJ. Experimental and clinical performance of porous tantalum in orthopedic surgery. Biomaterials. 2006;27(27):4671–81.

[Crossref][PubMed]

34. Bobyn JD, Stackpool GJ, Hacking SA, Tanzer M, Krygier JJ. Characteristics of bone ingrowth and interface mechanics of a new porous tantalum biomaterial. J Bone Joint Surg Br. 1999;81(5):907–14.
[Crossref][PubMed]

35. Bobyn JD, Toh KK, Hacking SA, Tanzer M, Krygier JJ. Tissue response to porous tantalum acetabular cups: a canine model. J Arthroplasty. 1999;14(3):347–54.
[Crossref][PubMed]

36. Heiner AD, Brown TD, Poggie RA. Structural efficcay of a novel porous tantalum implant for osteonecrosis grafting. Trans Orthop Res Soc. 2001;26:480.

37. Bobyn JD, Pilliar RM, Cameron HU, Weatherly GC. The optimum pore size for the fixation of porous-surfaced metal implants by the ingrowth of bone. Clin Orthop Relat Res. 1980;150:263–70.

38. Soucacos PN, Kokkalis ZT, Piagkou M, Johnson EO. Vascularized bone grafts for the management of skeletal defects in orthopaedic trauma and reconstructive surgery. Injury. 2013;44:S70–5.
[Crossref][PubMed]

39. Soucacos PN, Korompilias AV, Vekris MD, Zoubos A, Beris AE. The free vascularized fibular graft for brisdging large skeletal defects of the upper extremity. Microsurgery. 2001;31:190–7.
[Crossref]

40. Rahimnia A, Fitoussi F, Pennecot G, Mazda K. Treatment of segmental bone loss of the tibia by tibialisation of the fibula: a review of the literature. Trauma. 2011;16(4):154–9.
[Crossref]

41. Bhathena HM, Savant DN, Kavarana NM, Parikh DM, Sanghvi VD. Reconstruction with different free flaps in oro-facial cancer patients. Acta Chir Plast. 1996;38(2):43–9.
[PubMed]

42. Brenner P, Zwipp H, Rammelt S. Vascularized double barrel ribs combined with free serratus anterior muscle transfer for homologous restoration of the hindfoot after calcanectomy. J Trauma. 2000;49(2):331–5.
[Crossref][PubMed]

43. Pelzer M, Reichenberger M, Germann G. Osteo-periosteal-cutaneous flaps of the medial femoral condyle: a valuable modification for selected clinical scenarios. J Rec Microsurgery. 2010;26(5):291–4.
[Crossref]

44. Iorio ML, Masden DL, Higgins JP. The limits of the medial femoral condyle corticoperiosteal flaps. J Hand Surg Am. 2011;36(10):1592–6.
[Crossref][PubMed]

45. Kurvin LA, Volkering C, Kebler SB. Calcaneus replacement after total calcanextomy via vascularized pelvis bone. Foot Ankle Surg. 2008;14:221–4.
[Crossref][PubMed]

# Index

## A

# B



# C

# D

# E



# F

- Forearm nonunion
  - anatomy
    - bone
    - DRUJ
    - interosseous membrane
    - muscle
    - proximal radioulnary joint
  - anticipated outcomes/recurrence
  - case study
  - patient variables
  - risk factors
  - surgical treatment
    - approach and exposure
    - bone preparation
    - essentials of exposure
    - operating room setup
    - preoperative planning
    - technical points
- Fracture healing
  - acute inflammation
  - assessment
    - clinical criteria
    - fracture union
    - radiologic scores
  - biomechanics
    - biologic healing response
    - fracture gap and hydrostatic pressure
    - interfragmentary strain
    - role in skeletal repair
    - timing
  - comorbidities
    - aging
    - endocrine disorders
    - habits
    - medications
    - metabolic bone disease
  - direct

# H



# I

# L

# M

# N

# O

# P

# R



# S

# T

# U



# V



# W



# X



# Y

Young and active patients, recent study