Sports Injuries of the Foot and Ankle

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A Focus on Advanced Surgical Techniques

Springer

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Foreword

[AUT]

This book, *Sports Injuries of the Foot and Ankle: A Focus on Advanced Surgical Techniques* by the Leg, Ankle and Foot (LAF) Committee of ISAKOS, is *quintessential ISAKOS*. This is a comprehensive book that should be in the library of every surgeon who operates on injuries to the foot and ankle, athlete or not. It is edited by an internationally acclaimed group of sports foot and ankle surgeons, Gian-Luigi Canata from Italy, Pieter D'Hooghe from Qatar, Ken Hunt from the USA, Giuseppe Longo from Italy, and Gino Kerkhoff from the Netherlands, and includes contributions from esteemed and internationally recognized experts from *all over the world*—true international authorities on the management of foot and ankle problems in athletes. The work and effort put into this book by the editors and authors, along with beautiful original illustrations, will make it the new standard by which foot and ankle surgical technique books will be measured.

This book represents some of what is best about ISAKOS—international experts, leaders in the field, not just in their respective country, but recognized all over the world, coming together to share thoughts, ideas, and concepts, to help advance the field and to make the world just a bit closer. Collaboration, whether at the biennial meeting, at committee meetings, and at interim offsite meetings, with research, state-of-the-art papers, and publications, like this one, is the heart of ISAKOS. It is a brotherhood (for lack of a better gender neutral term), friendship, and sort of family.

The authors provide a comprehensive look at the different parts of the foot and ankle from a variety of perspectives. They introduce newer, cutting-edge techniques, along with the standard "tried and true" surgeries. They span the gamut of techniques, from the basics to complex, in a comprehensive, well-illustrated methodology. Each technique is described in detail to help the orthopedic surgeon perform it accurately and safely.

But more than just a surgical technique book, they discuss important topics such as the anatomy, biology, surgical outcomes, and footwear considerations. They also discuss newer areas in evolution, such as tissue engineering, and the ever important area of rehabilitation following these surgeries.

Drs. Canata, D'Hooghe, Hunt, Longo, and Kerkhoff, as members of the Leg, Ankle and Foot Committee of ISAKOS, have brought together a talented and respected group of foot and ankle specialists from the LAF Committee and other ISAKOS members and must be commended for the exceptional quality of this book and be congratulated for a job well done. This book will

be valuable to all surgeons who care for foot and ankle problems, particularly those who care for athletes with foot and ankle problems.

With the help and guidance of João Espregueira-Mendes, the head of the Publications Committee, it has been a huge endeavor. I am honored that it has been initiated and completed during my tenure as president. The authors, and editors, are to be congratulated for a Herculean effort and a book that sets a new standard.

Marc R. Safran
ISAKOS
Stanford, CA, USA
2017–2019

Preface

This book is an update on current techniques for the treatment of foot and ankle injuries and conditions in the athletic patient. It is meant to serve as a current and comprehensive review of the state of the art, with an international perspective. Each chapter is written by orthopedic surgeons expert in the field, sharing their experience treating specific injuries and conditions, cutting-edge surgical procedures, and injury management strategies. Several different techniques are described step-by-step, easing the reader to thoroughly understand what the surgeon is doing getting information on details.

ISAKOS is devoted to its mission of disseminating knowledge to the world of orthopedics and sports medicine. Through committees like the Leg, Ankle, and Foot (LAF) Committee represented herein, ISAKOS continuously works to help its membership, and the orthopedic and sports medicine communities it serves, to strive to improve the art, optimize the delivery of care worldwide, and seek at all times the best interest and outcomes of the patient.

The editors extend a sincere thanks to all the authors for their outstanding contributions and to ISAKOS for its steadfast and unwavering support for this project.

Torino, Italy — Gian Luigi Canata
Doha, Qatar — Pieter d'Hooghe
Aurora, CO — Kenneth J. Hunt
Amsterdam, Netherlands — Gino M. M. J. Kerkhoffs
Rome, Italy — Umile Giuseppe Longo

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Dhanapal Palanisamy, Gabriele Schroeder, and Springer for their professional work and dedication.

Our families for their enduring support and patience.

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Part I

Ligament Injuries

Acute Ankle Ligament Injuries

1

Kenneth J. Hunt and Peter Lawson

1.1 Introduction

Acute lateral ligament sprains of the ankle are a common injury for patients and athletes and a burdensome healthcare issue for hospitals. While in general a relatively well-understood and treatable injury, lateral ankle sprains and recurrent injuries are very common and predictive and prognostic factors are still not entirely understood. In the treatment of these injuries, it is important for providers to have a clear understanding of injury mechanisms and identify patients at risk for recurrent injury and chronic instability. In order to select appropriate treatment strategies, the provider must understand not only the severity of the injury but also the mechanisms and contributing factors that lead to lateral ankle ligament injuries and chronic instability. As new surgical and rehabilitation techniques evolve, understanding of both natural and injury mechanics is critical.

1.2 Epidemiology

Ankle sprains continue to be a prevalent and costly healthcare issue with estimates suggesting that ankle sprains account for 7–10% of emergency department admissions [1]. By other estimates, injury to the lateral ligaments of the ankle joint can account for about 1 in 10,000 people a day [2]. Generally, lateral ankle sprains are much more common than syndesmotic and medial ankle sprains [3].

Rates of incidence vary across gender, race, and age—black and white adolescent females are recognized as the populations most at risk for ankle sprains. Racially, black patients and white patients have shown incidence rates three times greater than Hispanic patients [4]. Generally, females have shown to be at a higher risk for ankle sprain injury than males, reporting 13.6 vs. 6.94 ankle sprain injuries per 1000 exposures [3]. However, there is some evidence that suggests that among patients 15–24 years, males present with higher rates of ankle sprains than females, but among patients older than 30 years, females have higher incidence rates than males [4]. It is important to take into consideration that while lateral ankle sprains are more common among female patients, medial and high ankle sprains generally show lesser or no gender differences [5]. Among youth, children are at higher risk than adolescents, and adolescents at higher risk than adults, reporting 2.85, 1.94, and 0.72 ankle sprain injuries per 1000 exposures, respectively [3].

Sports activities are widely recognized as the environment where participants are most prone to ankle injuries. This is particularly true of sports that involve jumping and changes in direction.

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Among youth sports, injury to the lateral ligaments of the ankle joint accounts for approximately one-fourth of all sports-related injuries [2]. Half (49.3%) of ankle sprains occur during athletic activity, 41.1% of which are associated specifically with basketball [4]. Analysis of injury risk by gender and sport has shown that female basketball athletes are considered most prone to first-time inversion ankle ligament injury [6]. Male basketball athletes and female lacrosse athletes are also considered high risk to injury [6]. Football and soccer also have high associations with ankle sprains [4].

Collegiate sports are a more unique domain of interest as it includes large athlete populations commonly recognized for the higher stakes of competition, levels of training, greater athlete size, strength and speed, and demand to return to play. It is estimated that among the 25 most common NCAA sports in the United States, there are over 16,000 lateral ligament complex (LLC) ankle sprains each year—accounting for approximately 7.3% of all collegiate sport injuries [7]. LLC sprains are regarded as the most common injury in college sports in the United States, occurring at a frequent rate of 1/2020 (4.95/10,000) athlete exposures—more specifically, the most frequent LLC sprain rates are in men's and women's basketball, which report at 1/836 (11.96/10,000) and 1/1052 (9.5/10,000), respectively [7]. Recurrence of LLC sprains is well recognized as an area of importance when monitoring and treating athletes. Studies have shown that among collegiate athletes 11.9% of LLC sprains are attributed to recurrence. Recurrent injuries are most frequent in women's sports—specifically basketball (21.1%), outdoor track (21.1%), field hockey (20.0%), and tennis (18.2%) [7]. The sports with the most frequent recurrence rate among males include basketball (19.1%), tennis (14.3%), outdoor track (14.3%), and soccer (14.0%) [7]. Rapid identification and treatment of the competitive athlete is paramount. Reassuringly, 44.4% of athletes who suffer an LLC sprain are able to return to play within 24 hours [7]. Alternatively, 3.6% of athletes have higher grade injuries and require more than 21 days before returning to play, with some unable to return [7]. Thus, it is very important to reduce the incidence, severity, and recurrence of LLC sprains [7].

1.3 Anatomy

The ankle joint complex is multiplanar and is made up of the subtalar (talocalcaneal) joint, the tibiotalar joint, and the transverse-tarsal joint [8]. Each of these joints has a particular plane of motion and a specific function associated with it. The subtalar joint allows for ankle inversion and eversion, and the joint is primarily linked via the interosseous talocalcaneal ligament which connects the inferior articular facet of the talus to the articulating facet on the superior surface of the calcaneus [8]. The tibiotalar joint primarily functions is a hinge joint, in the plantarflexion and dorsiflexion movements of the foot [8]. The motion of this joint is limited by three groups of ligaments—the tibiofibular syndesmosis, the medial collateral ligaments, and the lateral collateral ligaments [8]. The transverse-tarsal joint is a combination of articulations between the talus, the calcaneus, and the navicular, and shares an inversion-eversion axis of motion in the foot [8].

Ligaments are an essential structural feature in the ankle joints, providing stability and controlled range of motion across each specific joint. The lateral ligament complex of the ankle is made up of the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL) [1]. The medial (deltoid) ligament complex of the ankle is made up of the deep components—the anterior tibiotalar ligament (ATTL) and the posterior tibiotalar ligament (PTTL)—and the superficial components—the tibionavicular ligament (TNL), the tibiospring ligament (TSL), and the tibiocalcaneal ligament (TCL) [9] (Fig. 1.1).

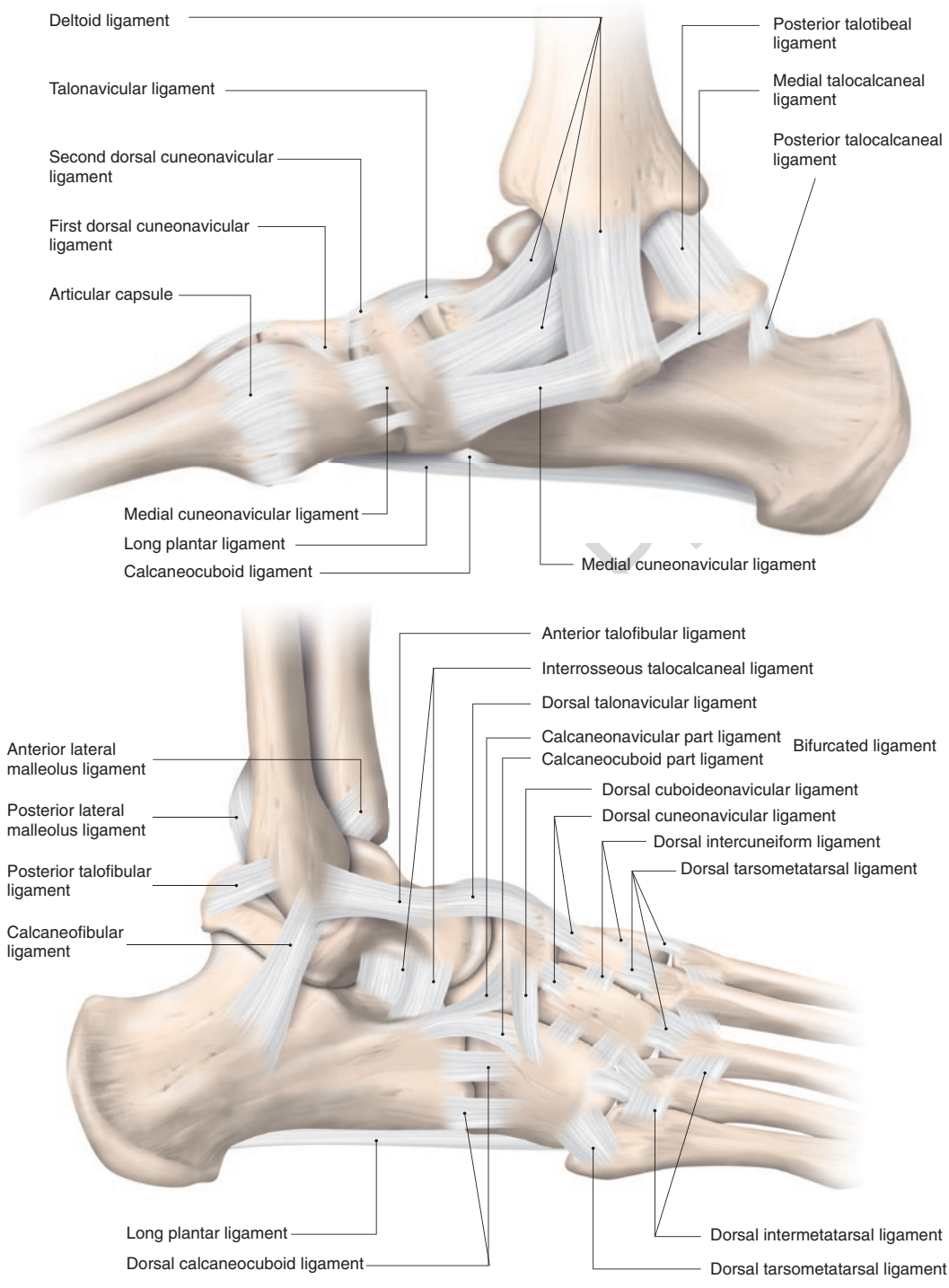

Fig. 1.1 Drawing depicting the ligaments on the medial (**a**) and lateral (**b**) side of the ankle joint

The stability of the ankle joint is multifactorial—many intrinsic elements (articular geometry) and extrinsic elements (ligaments) contribute to supporting the ankle joint [10]. These primary contributing elements also depend upon other factors such as ground condition, loading level, and the direction and magnitude of applied forces when loading and unloading [10]. When considering the articular geometry of the ankle, it is important to recognize that the talus has the bone morphology of a truncated cone, where the medial radius of curvature is lesser to the lateral radius of curvature, but there is variance in the medial-lateral distribution [11]. These structural variances explain the occurrence of high-risk ankles via their alteration in joint mechanics within the ankle [11]. A relatively high bone congruency across the tibiotalar joint distributes the applied loads across the large load-bearing surface area to mitigate impact stress on the ankle—some theorize even more effectively than the hip or knee [8]. Regarding stability of the ankle joint, the value of the high bone congruency has shown that when loaded (1 BW), articular geometry contributes 100% to translational stability and 60% to rotational stability [10]. Ligamentous stability is recognized as the other primary element contributing to ankle joint stability. The anterior stability of the ankle is approximately 70–80% dependent upon the lateral ligaments, when unloaded [10]. The posterior stability of the ankle is approximately 50–80% dependent upon the deltoid ligaments, when unloaded [10]. The rotational stability is 50–80% dependent upon both the lateral and deltoid ligaments, but medial-lateral stability is not primarily dependent upon these ligaments [10]. Due to the unique geometry of the tibiotalar joint, it is recognized that the ankle is more stable in dorsiflexion and less stable in plantarflexion [10, 12].

1.4 Ankle Joint Complex Biomechanics

Direction and ranges of motion of the ankle joint are complex. The ankle joint primarily moves in plantarflexion-dorsiflexion, with the addition of variable amounts of inversion-eversion (and abduction-adduction), allowing for more complex motions like supination and pronation [8]. The degree of multi-axial motion throughout the tibiotalar, subtalar, and transverse-tarsal joints varies depending primarily on the variance in talar anatomy and tissue stiffness [8]. However, typical range of motion for these joints from a neutral stance has been shown to be as much as 20° of dorsiflexion, 55° of plantarflexion, 23° of inversion, and 12° of eversion [8].

When assessing athletes with acute or chronic ligament injuries, it is important to understand the fundamentals of a gait mechanics to appreciate the impact, distribution of force, and flexion of muscles throughout the gait phases. A normal gait is comprised of a stance phase—which is further subdivided into heel rocker, ankle rocker, and forefoot rocker subphases—and a swing phase [8]. The heel rocker phase begins when the heel strikes the ground and ends when the foot is flat—during which the ankle is in a slight plantarflexed position, and the dorsiflexor muscles exhibit eccentric contraction [8]. The ankle rocker phase is transitional phase from plantarflexion to dorsiflexion about the tibiotalar joint [8]. The forefoot rocker phase begins when the heel of the calcaneus lifts off of the ground and ends when there is toe-off from the ground—this is marked at 50% of the gait cycle during which active plantarflexion generates the maximal joint power that propels the walker forward [8]. The swing phase activates slight dorsiflexion to better ensure foot clearance of the ground, before returning to plantarflexion in the heel rocker phase [8]. Inversion complements the plantarflexion at heel strike, and eversion compliments the plantarflexion throughout the forefoot rocker phase, as both biplanar motions are enabled by the subtalar joint [8].

The load and applied forces are skillfully distributed about the ankle joint throughout ones walking gait. The amplitude of the vertical component of the ground reaction force peaks at approximately 1.0–1.5 body weight, with slight proportional increase depending on walking speed [13]. On the superior surface of the talus, the tibiotalar joint bears 83% of the load and the

fibulotalar joint bears 17% of the load [14]. Seventy-seven to ninety percent of the load on the tibiotalar joint is applied on the surface of the talar dome with an appreciable loss across the medial and lateral gutter surfaces [15]. The relatively high bone congruency across the tibiotalar joint is credited for guiding the distribution of loading forces primarily through the tibiotalar joint to mitigate irregular location and magnitude of impact stress on the ankle [8].

1.5 Mechanism of Injury

The most common mechanism of injury to the lateral ligament complex is inversion of the ankle with the foot in plantarflexion [1, 11]. Of the lateral ligaments, a tear of the anterior tibiofibular ligament (ATFL) is most common, particularly in athletes, followed by the calcaneofibular Ligament (CFL) [16]. Other common ligaments injured include PTFL, the cervical ligament and the talocalcaneal ligament—which is more commonly injured when in dorsal-varus-flexion [11]. Common symptoms associated with the acute ankle sprains include pain, range of motion deficit, postural control deficit, and muscle weakness [17].

Ankle sprains are graded, and treated, based on their severity, and the treatment protocol is guided by grading. Severity of ankle sprains is graded I—mild, II—moderate, III—severe [16]. Grade I and II injuries are typically successfully treated by nonoperative management and functional treatments—this includes the use of RICE (rest, ice, compression, elevation), brief immobilization and protection, early range of motion, neuromuscular training, proprioceptive training, balance, and weight-bearing exercise [16]. Treatment of grade III injuries can be more complicated [16]. Grade III "sprains" involve complete tearing of the ATFL and CFL ligaments and much or all of the PTFL. Since the ligamentous complex is completely ruptured, these injuries must necessarily be managed differently. Immobilization, swelling reduction, and functional rehabilitation are initiated to help the ankle recover more quickly while avoiding risks of other complications and sequelae [16]. However the use of surgical repair techniques for primary treatment is growing in popularity given the effectiveness of modern rehabilitation techniques, and the lost time and recurrent injury rates associated with high-grade ligament tears [16, 18].

1.6 Concomitant Injury Considerations

Further complications stemming from injury to the lateral ligaments of the ankle joint often include acute pain local to the site of injury, residual complications such as joint instability, stiffness, swelling, peroneal tendon injury, avulsion fractures, cartilage damage, and recurrent injury that increases the risk of long-term joint degeneration [2]. Common sequelae that occur in 10–30% of patients with chronic lateral ligament injuries include synovitis, tendinitis, ankle stiffness, swelling, pain, nerve stretch injury, and muscle weakness [16]. Pain in the limb, sprain of the foot, and abrasion of the hip/leg are complications that have been found to be more common in lateral ankle sprain events than medial joint injury [5].

1.7 Chronic Ankle Instability

Chronic ankle instability (CAI) is classified by the persistence of lateral ankle sprain symptoms—including pain, range of motion deficit, postural control deficit, and muscle weakness—however the true cause remains controversial [17]. Chronic mechanical instability is characterized by general laxity which is associated with ligament lesions and other complications including impingement, osteochondral lesions, and fibular tendon pathology [11]. Postural factors and proprioceptive deficiencies also favor functional instability and should be evaluated and considered during treatment of chronic ankle instability [11].

There remains debate and uncertainty regarding the factors and mechanisms that contribute to

chronic ankle instability. Some challenge the theory that kinematic variations are a significant mechanism contributing to CAI—as a study showed lower limb kinematics during forward and side jump landing tasks were not different when comparing CAI to healthy subjects [19]. Other studies suggest that while proprioceptive deficits, neuromuscular changes, muscle strength, postural changes, and central adaptations have been shown to contribute towards CAI, the direct mechanism by which these factors lead to CAI remains poorly understood [19–21].

1.8 Risk Factors

Given the ubiquitous nature of ankle ligament injury, and differences in study populations, there are an array of risk factors for recurrent injury and CAI. These include, but are not likely limited to, sex, weight, height, limb dominance, ankle joint laxity, anatomical alignment, strength, reaction time, and postural sway [22]. Factors that have been shown to correlate with an increased risk of lateral ankle sprain include increased body mass index, muscle strength (slow eccentric inversion strength, and fast concentric plantarflexion), proprioception (passive inversion joint position sense), and muscle reaction time (earlier reaction time of the peroneus brevis) [23]. There is inconclusive evidence regarding the associations between decreased ankle eversion strength and delayed ankle evertor reaction time, and lateral ankle ligamentous sprains [23].

Generalized ligamentous laxity is considered a risk factor for instability recurrence following modified Broström procedure for chronic ankle instability [24]. Other metrics that have been shown to be associated with clinical failure following use of the modified Broström procedure for chronic ankle instability include syndesmosis widening, osteochondral lesion of the talus, high preoperative talar tilt angle (>15°), and high preoperative anterior displacement of the talus (>10 mm) [24]. Further research suggests determining additional predictive factors and grading chronic ankle instability to improve patient outcomes, and to better evaluate better treatment options to prevent early failure, including anatomic ligament reconstructions, nonanatomic ligament reconstructions, additional augmentations, tendon grafts, and suture tape [24].

1.9 Evaluations and Diagnosis

Prompt and thorough examination of the ankle is of great importance when assessing ankle sprain injuries. Physical examination within 4–5 days of traumatic injury provides the highest quality diagnosis [1]. Diagnostic features often include swelling, hematoma, local pain on palpation, and a positive anterior door test [1]. When assessing a patient with an ankle sprain, it is important to test for ligamentous disruption and ligament function [16]. There are two main clinical stability tests used—these include the anterior drawer test, which tests ATFL function, and the inversion tilt test, which tests ATFL and CFL function [16]. Further assessment may include radiographic imaging to assess ligament injuries [16]. It is important to be cognizant of the situational needs of your patient. While the Ottawa rules may be applied, weight-bearing ankle radiographs are very helpful to obtain in athletes with higher grade injuries since assessing alignment and identifying fracture, articular or other bony injury can be very useful for treatment. While less common in the lay person, ultrasound and MRI are more commonly used to diagnose associated injury and are routine evaluations in athletes [1]. As always, it is important to consider and balance both the timeliness and accuracy of these evaluations as patients' risks, benefits, costs, and desires vary by injury and by individual [1].

1.10 Treatment

Beneficial treatment methods for acute lateral ligament injuries in the ankle joint include functional treatment, immobilization, NSAIDs, and sometimes surgery [2, 25]. The majority of acute lateral ankle ligament injuries can be managed without surgery, most commonly protected by a semi-rigid ankle brace [26]. Braces have been shown to reduce risk of reinjury following an ankle sprain [22].

Initially, nonsurgical treatment is used to treat mild, moderate, and severe ankle sprains. RICE (rest, ice, compression, and elevation) therapy is commonly used as it is beneficial in reducing pain and swelling in the first 4–5 days following injury [1]. Beyond immediate treatment, immobilization (below knee cast or removable boot) provides treatment of pain for 5–10 days [1]. It is important to note that while immobilization is a common and effective treatment in reducing pain in swelling in the first 7–10 days, it can worsen symptoms if used for more than 4 weeks [2, 25]. RICE, ankle braces, and immobilization remain the most common and effective nonsurgical treatments; however questions still remain concerning which nonsurgical treatments are associated with the lowest re-sprain rates [26].

Surgical treatment is recommended for severe ankle sprain injuries that do not resolve with the initially conservative nonsurgical treatment methods, chronic ankle instability, and injureis with certain associated injuries or pathology. The details of these procedures are explored in later chapters. The goal of ankle ligament repair or reconstruction is to restore soft tissues to the anatomic condition prior to their instability, trauma, or arthritis [10]. Modifications of the Broström procedure are the primary technique used for surgical treatment of lateral ankle instability, specifically ATFL repair; however surgical techniques continue to warrant need for improvement [27]. Surgery may provide increased joint stability, but it is important to consider potential risks of each surgical approach [2, 25]. Surgical repair should be considered on an individual basis, particularly for patients with chronic instability and grade III injuries [26].

Beyond surgical reconstruction and traditional nonsurgical treatment, a few alternative treatment methods are used but effectiveness in improving symptoms remains poorly understood—these treatments include cold treatment, diathermy, homeopathic ointment, physical therapy, and ultrasound [2, 25]. Additionally, neuromuscular balance training has shown to be an effective preventative treatment for patients with previous sprains [26].

When treating athletes, there is a trend toward more aggressive treatments such as surgery for professional athletes with acute grade II or III injuries, as this may provide better long-term stability and mitigate risk of recurrent injury and asociated injur, or prolonged missed time from sports participation [1].

1.11 Prognosis

The vast majority of patients do well following lateral ligament injury and following lateral ligament repair. Barring major concomitant injury (e.g., osteochondral injury), most are able to return to their previous level of function. Prognostic factors for acute lateral ankle sprains remain somewhat elusive in aggregate [28]. Age has demonstrated prognostic value in some studies, but not all [28]. Independent predictors of poor recovery may include but are not limited to female gender, swelling, pain, limited range of motion and ability, injury severity rating, and MRI determined sprained ligaments [28]. Recent work suggests that generalized ligamentous laxity may be an independent predictor of clinical failures and poor radiological outcomes following modified Broström procedure for chronic ankle instability [24].

1.12 Ankle Arthritis and Salvage Strategies

Post-traumatic osteoarthritis, and other degenerative processes, can negatively impact the biomechanical functions of the foot and ankle [29, 30]. Furthermore, a decrease in muscular strength associated with increasing age demonstrates a reduction in the range of motion in the ankle joint across both genders [12]. However, while younger age females (20–39 years) have a higher range of motion than males, elderly women (70–79 years) demonstrate less dorsiflexion and greater plantarflexion comparatively to elder men [12]. These changes in bone strength, muscle strength, and range of motion are important considerations to take particularly when treating more elderly patients.

More complex surgical treatment methods arise for patients whose lateral ankle sprain or chronic ankle instability may be complicated by other factors such as age and arthritis. Total ankle joint replacement is a common surgical intervention considered for end-stage ankle osteoarthritis, as total ankle replacements have shown improvements in walking speed, spatio-temporal function, and range of motion, in exchange for reductions in ankle joint moments and power [29, 30]. Ankle arthrodesis via fusion of the tibiotalar joint into a fixed position is another surgical consideration—this treatment option has been shown to improve walking speed and spatio-temporal function, but a reduction in the range of motion of the joint may result in adjacent joint osteoarthritis and other complications including malalignment, non-union, dysfunction, and pain [31, 32].

1.13 Economics

Ankle sprain emergency department admissions can be costly for both the patient and the hospital [1]. A very high recurrence rate of lateral ankle sprains contributes to significant medical expenses—mainly attributed to care, prevention, and secondary disability [17]. There are nuances that differentiate the costs and related care between various ankle sprain injuries and their treatments. Emergency room treatment of lateral ankle sprains (US $1025) are relatively more costly than medial ankle sprains (US $912), but are comparable in costs for high ankle sprains (US $1034) [5]. These numbers do not include subsequent visits to a specialist, physiotherapy, and related treatments, let alone the costs of those that become chronic and/or require surgical repair. Among sources of expenses, medial ankle sprains are more likely to include diagnostic radiology, lateral ankle sprains are more likely to include medications, and high ankle sprains are more likely to include hospitalizations [5].

When treating patients with an ankle sprain, it is important to consider cost-effective treatment options. One study suggests using the Ottawa ankle rules diagnostic decision aid to exclude fractures of the ankle and mid-foot, rather than using radiographs, as a means of reducing radiograph expenses [33]. Furthermore, semi-rigid ankle braces worn during sports activities have shown to be a more cost-effective secondary intervention for preventing recurrence of ankle sprains than neuromuscular exercise training [34]. Additionally, proprioceptive balance board training programs targeted at players with previous ankle sprains that are prone to recurrence may prove to be a cost-effective long-term intervention [35]. It has been suggested that preventative intervention via use of proprioceptive balance training programs targeted at athletes with previous ankle sprains may reduce costs per player up to $56 USD [7, 35]. More general estimates suggest that the cost of preventing one ankle sprain has been estimated at $483 USD [7]. Overall, preventative and cost-effective treatments for ankle sprain injuries particularly among patients at risk for recurrence can prove to be effective in reducing the financial burden of ankle sprain injuries.

1.14 Summary

Lateral ankle sprains are a very common and often troublesome injuries in athletes and nonathletes alike. There is substantial existing evidence

of anatomic, biomechanical, and ligamentous tissue qualities that provide an explanation for lateral ankle sprain injuries; however predictive and prognostic factors remain incompletely understood. Conservative treatment, such as RICE and semi-rigid ankle braces, are common and effective initial treatments for ankle sprain injuries. Surgical treatment considerations are reserved for more severe injuries that do not resolve and athletes that demand more stable treatment but should be used cautiously among elderly patients that present risks of other ankle complications. Risk for recurrence is important to consider as recurrent injuries can be damaging and costly for the patient and can be indicative of greater chronic instability issues at hand. Ultimately, it is important to treat these patients, but also to identify patients at risk for injury recurrence to mitigate the patient's potential losses and to ultimately improve their outcome, performance, and quality of life.

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Lateral Endoscopy of the Ankle

2

Stéphane Guillo

2.1 Introduction

Endoscopy of the hindfoot has long been limited to arthroscopy of the anterior part of the ankle. More recently, the posterior route described 15 years ago [1] has resulted in a great step forward by making it possible to reach the posterior intra-articular as well as the extra-articular structures. Tendoscopy of the peroneal tendons has by now been described more than 10 years ago [2] for the treatment of tendinopathies. This technique has, however, been used very little to date. Nonetheless, it offers an exceptionally good view of the lateral part of the hindfoot. By following the peroneal tendons, and using accessory portals, it can be used to find, explore, and reach the lateral ligaments of the ankle, the rear side of the lateral malleolus, the entire lateral side of the anterior and posterior subtalar joints, the sinus tarsi, as well as the upper side of the calcaneus to its apophyseal.

Building on tendoscopy, by considering the container but not the content, this new concept of lateral ankle endoscopy hence emerged that nowadays constitutes one of the foremost tools for investigation when treating a greater number of pathologies of the hindfoot. Just like endoscopy of the shoulder, it allows a bona fide endoscopic dissection of the extra-articular structures of the lateral side and it makes endoscopic treatment of chronic lateral instability of the ankle a potential option.

2.2 Indications

Tendoscopy has first of all been described to treat tendinopathies. Adhesions linked with inflammatory phenomena are readily treated by simple passage of the trocar of the optical device (candlelight effect). Other than this candlelight effect, tendinopathy of the peroneal tendon can be treated by straightforward debridement using a shaver. By means of a supplemental mini-open, one can perform a repair of a possible fissuration.

Tendoscopy of the peroneal tendons is also a way to reach the lateral side of the calcaneus, as well as the lateral side of the subtalar joint. In addition to the treatment of ligament pathologies, it therefore allows treatment of possible lateral impingement by the bony spur as well as rectification of certain fragmented fractures of this region (lateral tubercle of the talus, calcaneal apophyseal edge,…).

Tendoscopy also allows peroneal tendon instability to be treated [3, 4]. Lastly, it can constitute the first part of exploration or a procedure at the level of the sinus tarsi. It then allows systemization of the dissection.

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2.3 Surgical Anatomy

The peroneus longus tendon inserts on the proximal two-thirds of the lateral side of the fibula, while the peroneus brevis tendon emerges at the level of the distal third and on the adjacent interosseous membrane. The peroneus longus tendon extends the fleshy body of the muscle 3–4 cm above the malleolus while the muscle fibres of the peroneus brevis tendon very often descend up to fibula tip. This feature can be the basis for genuine impingements between the two peroneal tendons [5].

They are generally described as having three different areas (A, B, and C) [6] to which Sammarco [7] has added a fourth (D) (Fig. 2.1).

Area A corresponds with the posterior side of the malleolus, featuring a gutter in 8 out of 10. The absence of a gutter at this level is recognized as being a risk of dislocation of the peroneal tendons [2]. In this part, the tendons are held back by their sheath, which provides a reinforcement that provides a great deal of stability: the superior peroneal retinaculum, distinct and wide along its entire retromalleolar trajectory (Fig. 2.2). The peroneus brevis tendon is anterior and flattened distally, while the peroneus longus tendon behind has a more round cross-section.

Area B corresponds with the part comprised between the malleolus at the level of the lateral side of the calcaneus and the cuboid bone. At this level, the two tendons are at first free and their

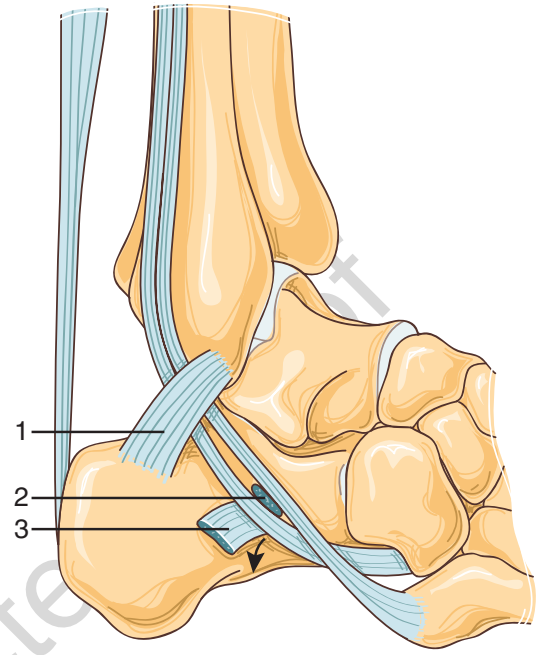

Fig. 2.2 Retinaculum of the peroneal tendon (lateral view): (**a**) Superior retinaculum. (**b**) Inferior retinaculum. (**c**) Tubercle of the peroneal tendons

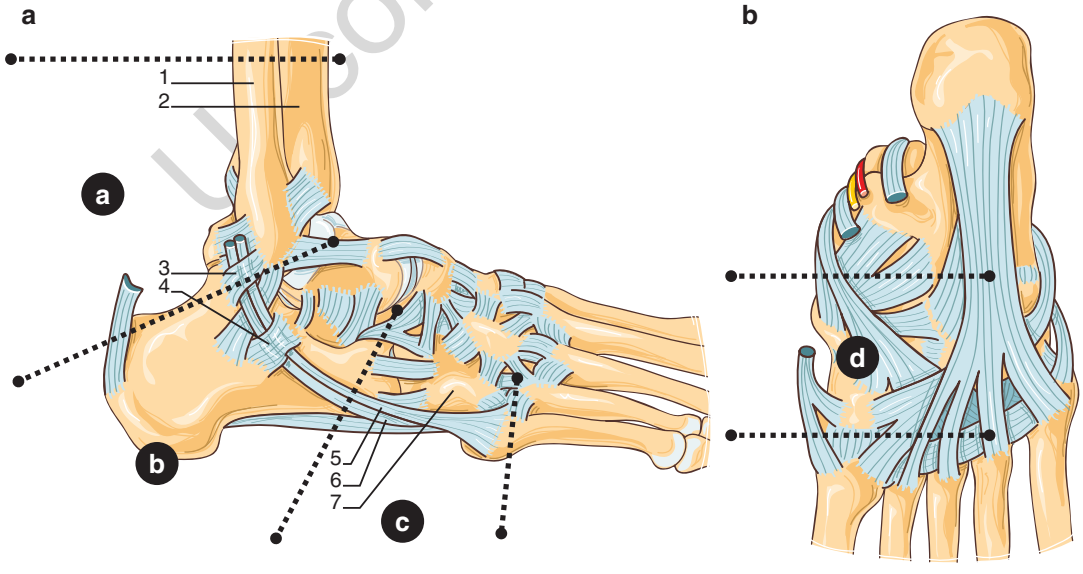

Fig. 2.1 The four areas of the peroneal tendons. (**a**) Lateral view. (**b**) Plantar view

trajectory crosses the calcaneofibular ligament (which stands out in tendoscopy) while following the edge of the posterior subtalar joint. In this part, the peroneus brevis tendon is on top and the peroneus longus tendon underneath. More distally, the two tendons each enter into their own tunnel. This very special area is situated at the level of the peroneal tubercle (PT). The tunnels are separated by a septum that arises from the PT. In this trajectory, each tendon marks a furrow at the lateral side of the calcaneus. The inferior retinacular ligament marks the end of these osteofibrous gutters (Fig. 2.3).

Area C: Situated facing the cuboid bone, this area is that of the plantar crossing of the peroneus longus tendon, while the peroneus brevis tendon remains on the lateral side. In 20% of cases, there is an accessory fibular bone in this area.

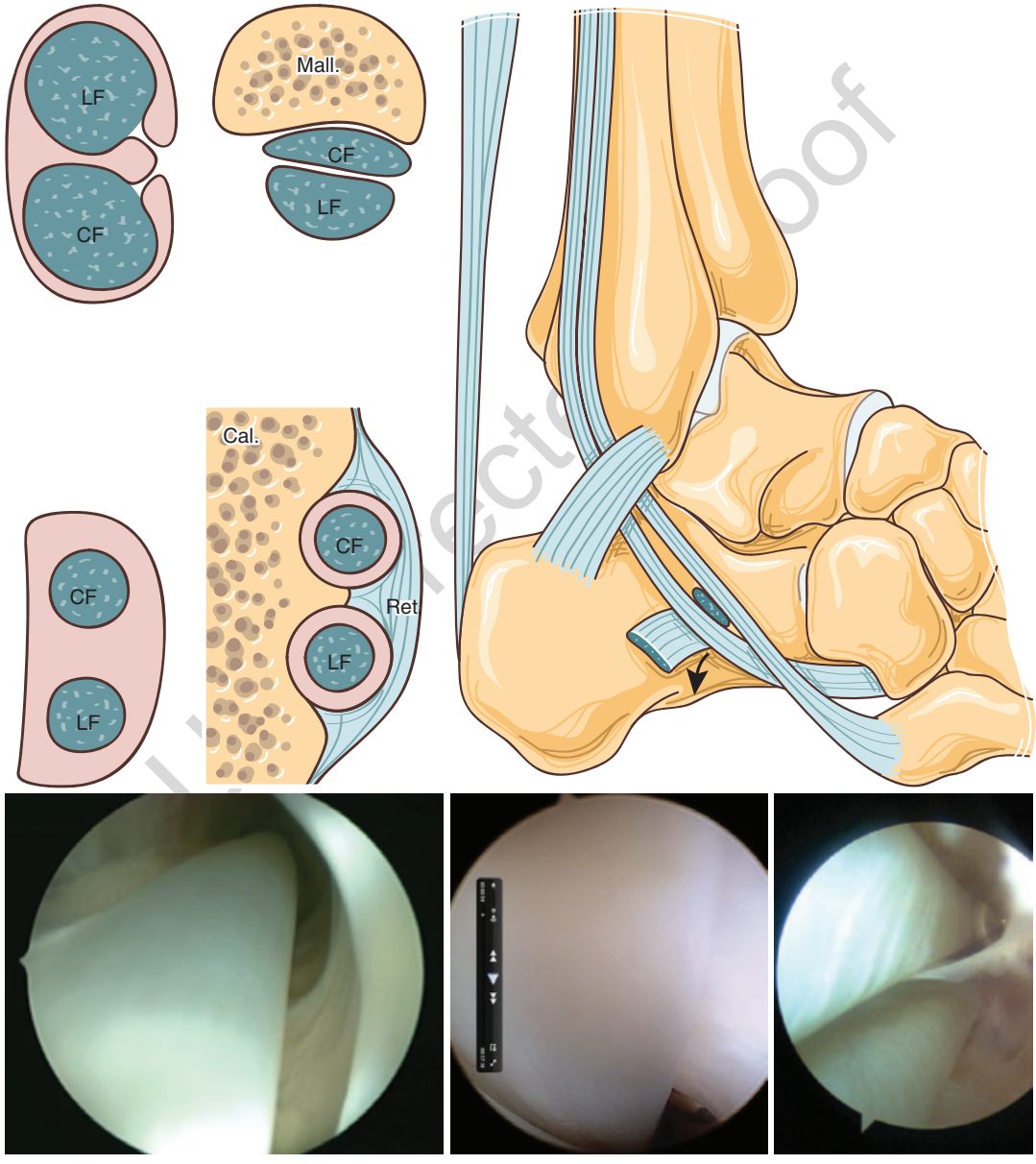

Fig. 2.3 Arthroscopic anatomy. Layered sections of the different areas

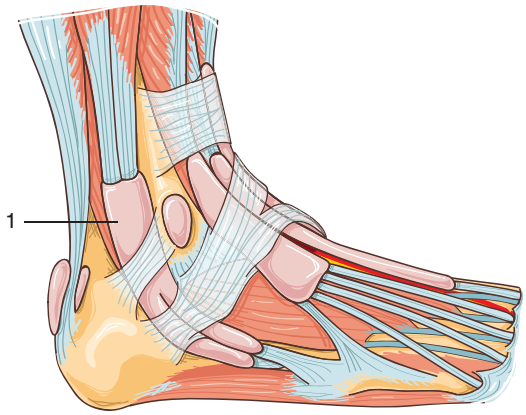

Fig. 2.4 Synovial sheath of the peroneal tendons

Area D corresponds to the trajectory of the peroneus longus tendon.

Tendoscopy is made possible thanks to the presence of a synovial sheath. The sheath is a single entity from the proximal part up to the peroneal tubercle (Fig. 2.4). While this does not have genuine therapeutic implications at present, it should be noted that on this entire trajectory, the two tendons remain connected first to the posterior side of the malleolus, then to the lateral side of the calcaneus, each by their own vincula. It lies in alignment with the muscle fibres and represents the vinculum of the tendons.

The main neurological risk is in regard to the sural nerve, which after having crossed the superficial aponeurosis, typically in the upper third of the leg, rejoins the lesser saphenous vein in the lateral third of the leg, between the fibula and the calcaneal tendon. It crosses the trajectory of the peroneal tendons in area B to then innervate the dorsolateral skin of the foot and the toes. At the level of the malleolus, it gives rise to a cutaneous branch that is important for innervation of the heel (the calcaneal branch). The superficial fibular nerve does not constitute a risk. It runs in the lateral side of the leg, in front of the peroneal tendons, but typically pierces the superficial fascia 7–8 cm above the malleolus. Its superficial trajectory is then more forward, in front of the malleolus, constituting a risk primarily with the anterolateral route for arthroscopy of the ankle.

2.4 Technique

2.4.1 Setup

A tourniquet is placed above the knee, so as to take the path of the tendons into account. Rather than the supine position with a cushion under the prone buttock that is used by some, we preferentially use a sideways recumbent position with the foot raised. Nonetheless, it is sometimes useful to have an intermediate setup in the case where arthroscopy of the ankle is to be undertaken, so as to allow a sideways and an anterior position [7, 8]. The patient is placed lying on their side with their pelvis tilted slightly backward by approximately 30°. The hip and the knee are free. The ankle is held in line with the hip by support placed 10–20 cm more proximal. It is important to carefully verify the setup of the patient that by means of three different positions needs to allow anterior arthroscopy of the ankle (position 1), a lateral endoscopy of the ankle (position 2), and possibly removal of the gracilis (position 3) to be performed.

Position 2 is obtained by performing an external rotation of the hip to place the anterior side of the ankle as the highest point. Position 3 is obtained by resting the ankle on the support. Position 1 is obtained by performing a flexion and an external rotation of the hip (Fig. 2.5).

2.4.2 The Instruments

The instrumentation is conventional with an arthroscope of 4 mm and an arthroscopy shaver of 3.5–4 mm. It is not essential to use an arthropump or even electrocoagulation as the intervention is carried out using a tourniquet.

A basket forceps is very useful to start debriding a fissure tendinopathy. Among the small instruments, we prefer a N°15 scalpel blade, safer and less traumatizing than a blade of 11, and we recommend generating the first portal by employing two small Gillies hooks. It is furthermore indispensable to have a small curved Halstead forceps. This allows trauma to the subcutaneous nerves to be avoided after incision of the skin.

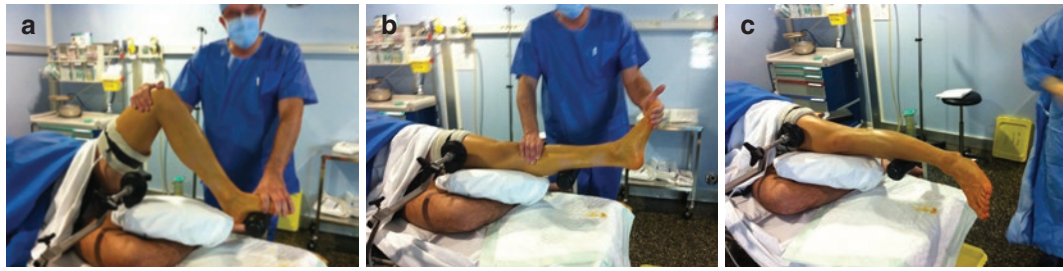

Fig. 2.5 The three positions for the setup. (**a**) Position 1; (**b**) position 2; (**c**) position 3

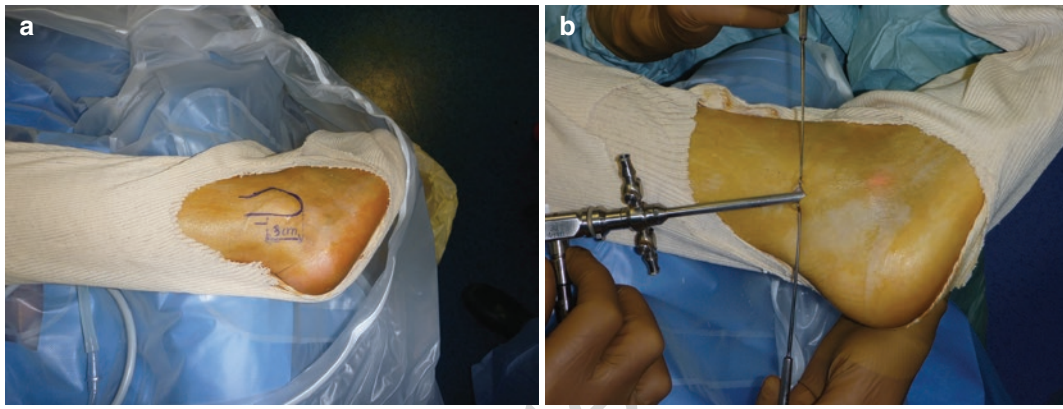

Fig. 2.6 Initial performance of the proximal portal between 2.5 and 3 cm above the malleolar tip. (**a**) Landmarks. (**b**) Opening of the sheath under visual control, equipped with Gillies hooks

2.4.3 The Actual Tendoscopy Technique for the Peroneal Tendons

The intervention can generally be performed under general or locoregional anaesthesia. Performing the procedure under local anaesthesia is also an option, with the major advantage of being able to carry out a dynamic test, which is useful in the diagnosis of certain forms of peroneal instability [2].

2.4.3.1 The Approach Routes

It is possible to generate the portals along the full length of the tendon behind the fibula but also distally on the lateral side of the hindfoot. In the vast majority of cases, however, two portals, one 3 cm above and the other 3 cm below the malleolus, are sufficient. The proximal portal is performed first. It offers the advantage of allowing easier identification of the sheath of the peroneal tendons, which is thicker at this level. The risk of nerve injury is much less, it is not necessary to dilate the peritendinous space, and the descent of the arthroscope in the sheath of the peroneal tendons is easier than when going up as the wall becomes thinner distally and there is more room.

A subcentimeter longitudinal incision using a blade of 15 therefore only opens the skin 2.5–3 cm above the malleolar tip for the sheath of the peroneal tendons. We recommend going down 1 cm when the intervention is in regard to the sinus tarsi (Fig. 2.6).

Using Gillies hook-type spreaders, and under visual control, the sheath of the peroneal tendons is then exposed for the longitudinal incision. It is then very easy to control and then to introduce the soft arthroscopy trocar into the sheath. The arthroscope is then pushed distally, beyond the tip of the malleolus. It is then possible to position the second portal using a needle. Transillumination allows the sural nerve to be avoided (Fig. 2.7).

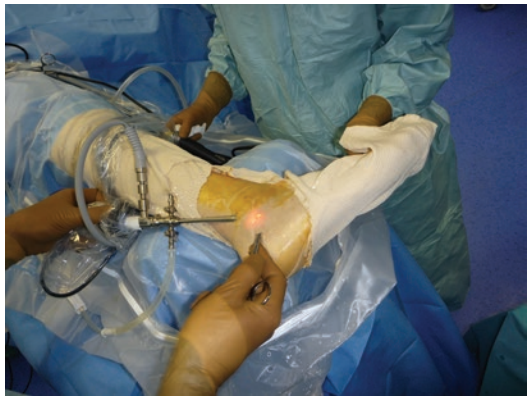

Fig. 2.7 Performance of the distal portal by transillumination

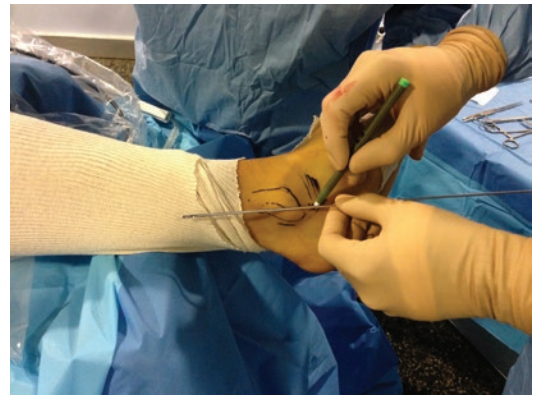

Fig. 2.8 Performance of the sinus tarsi portal

An initial inspection can then start from the distal emergence of the tendons, each from their own groove, up to the posterior side of the malleolus. It allows nearly all of the area to be visualized.

The vast majority of fissure tendinopathies are situated in the tendon reflection areas, under the malleolar tip.

By distally continuing the exploration after the malleolar groove, the base of the calcaneofibular ligament can be visualized. Its debridement with a shaver allows the posterior subtalar articulation to be visualized on its lateral and anterior side. It is then possible to perfectly control the resection of small fragments or exostoses of this region by this arthroscopic portal. As was shown recently, arthroscopic treatment of the lateral impingement, proposed by Lui [9], particularly after fracture of the calcaneus, has proven to be an interesting conservative alternative both as a result of its efficacy and of its absence of morbidity [10].

This same route moreover allows access to the sinus tarsi to be fully secured: it suffices to perforate the adipose tissue right after the base of the calcaneofibular ligament. It amounts to a bona fide conversion of a tendoscopy into subtalar arthroscopy since one can thereby reach the anterior part of this joint, as well as the calcaneal apophyseal edge and even the calcaneocuboid joint.

2.4.4 The Actual Lateral Endoscopy Technique

The intervention takes place under general anaesthesia only because locoregional anaesthesia does not allow for easing of the external rotation of the hip necessary for performing the anterior arthroscopy.

2.4.4.1 Placement of the Portals

Three portals are required to perform this surgery. The conventional anteromedial portal is called portal N° 1. The second portal (route N° 2) is not drawn on the skin; it is performed using transillumination after having placed the arthroscope. The third portal (route N° 3) is that of the sinus tarsi. It is necessary to draw two lines on the skin: The upper edge of the peroneus brevis is a line passing through the malleolar insertion point of the anterior talofibular ligaments (ATFL) and of the calcaneofibular ligament (CFL) and oriented at 10° relative to the axis of the malleolus. Portal N° 3 is situated at the intersection of these two lines (Fig. 2.8).

2.4.4.2 Stage N° 1

The arthroscope is placed in the anteromedial portal (N° 1). In order to obtain a good view of the lateral talofibular gutter, it is very important to position portal N° 1 correctly, that is to say, in dorsal hyperflexion and as close as possible to the anterior tendon. The positioning of the view

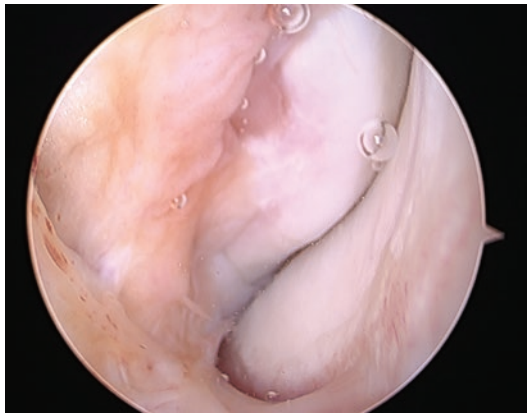

Fig. 2.9 View of the lateral gutter with the talus to the right and the malleolus to the left

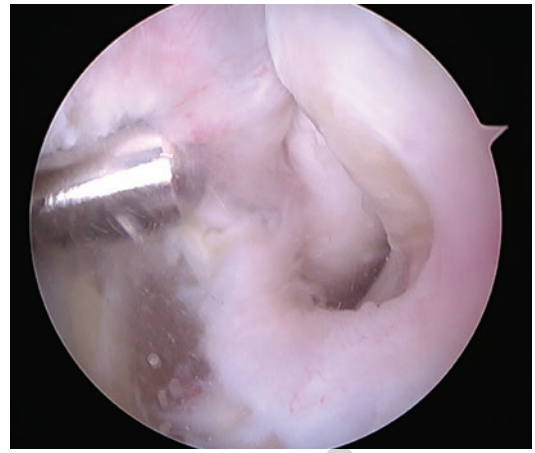

Fig. 2.10 View of the lateral gutter after preparation

spot needs to allow the anterolateral gutter to be seen (Fig. 2.9). The luminous spot generated by the arthroscope on the skin then allows the anterolateral approach to be performed (portal N° 2). Using a shaver placed in this portal, debridement of all of the lateral gutter is performed. This preparation needs to allow all of the scar tissue between the anterior tibiofibular ligament and the anterior talofibular ligament (ATFL) to be withdrawn. The preparation continues with the release of the ATFL on its malleolar insertion. It is then possible to fully expose the ATFL by preparing it in the same way as a tendon of the cuff on its upper side but also on its lateral edge (Fig. 2.10).

2.4.4.3 Stage N° 2

The arthroscope is placed in portal N° 2. An instrumental portal (portal N° 3) is performed at the level of the sinus tarsi using previously drawn cutaneous marks. A shaver is then introduced through this portal to complete the preparation at the level of the malleolar insertion of the ATFL and of all of its lateral side and its lower edge. The dissection is then pursued by following the lateral articular surface of the talus until encountering the subtalar joint. The lateral edge of the calcaneus is identified below the joint. By staying in contact with the calcaneus with the shaver, the calcaneal insert of the calcaneofibular ligament (CFL) is sought behind and within the fibular tendons while taking good care to remain in contact with the lateral cortex of the calcaneus and by moving from the front to the back. This stage needs to be done carefully in order to identify the CFL at its insertion.

2.4.4.4 Stage N° 3

The arthroscope is introduced in N° 3. Using a shaver placed in portal N° 2 it is possible to pursue the dissection and full visualization of the talar insertion of the ATFL (Fig. 2.11).

2.5 Conclusion

In addition to a lateral approach of the bone and joint structures of the hindfoot, lateral endoscopy allows for full exposure of the lateral ligamentous apparatus and of the tendons. It hence constitutes a minimally invasive way to treat a considerable number of pathologies of this region. It allows a targeted treatment by à la carte endoscopic dissection. The indications are broader nowadays with the treatment of lateral impingement, fragment fractures (resection), subtalar arthrodesis, instability of the peroneal tendons, and above all treatment of instability of the ankle.

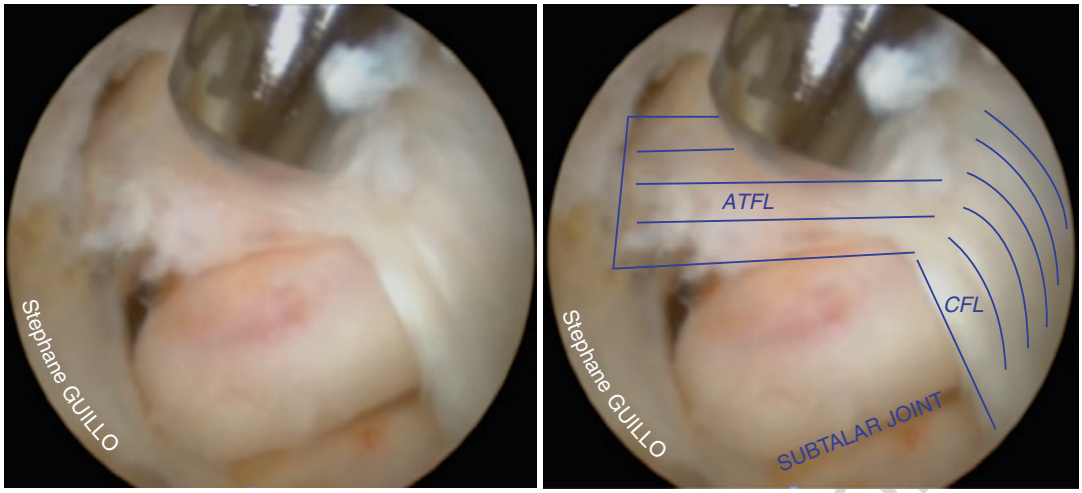

Fig. 2.11 Visualization after arthroscopic dissection of the anterior talofibular ligament and the calcaneofibular ligament

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3 All-Inside Endoscopic Broström-Gould Procedure for Chronic Ankle Instability

Haruki Odagiri, Stéphane Guillo, and Thomas Bauer

3.1 Introduction

Ankle sprains are the most common sports-related injury. The main complication is the development of chronic ankle instability (CAI), which occurs in about 20% of patients [1, 2]. Surgery to stabilize the ankle is indicated when nonoperative treatment fails. The goal of surgery is not only to restore stability but also to prevent the development of lesions due to chronic instability such as osteo-chondral lesions at the talar dome and, most importantly, tibio-talar osteoarthritis [3–6].

There are basically two main groups of surgical procedures for CAI, with many variants and modifications: repair techniques (retensioning and direct suturing of the anterior talofibular ligament [ATFL] and calcaneo-fibular ligament [CFL]) and reconstruction techniques (in which a tendon graft is used to rebuild the ATFL and CFL). The most popular repair technique was described by Broström in 1966 [7] with retensioning and direct suturing of the ATFL. Augmentation by advancing the extensor retinaculum as described by Gould et al. [8] can be added. A Broström-Gould procedure seems to remain the gold standard for CAI [9].

In recent years, several studies reported good short-term outcomes of arthroscopic repair techniques [10–18]. The arthroscopic technique of the Broström-Gould repair technique for CAI is described. Although the role for arthroscopy in the management of CAI remains controversial, these arthroscopic procedures may improve the detection of ligament lesions, as well as of concomitant lesions amenable to same-stage treatment [19–21]. Theoretical advantages of arthroscopic surgery for CAI include lower rates of cutaneous and infectious complications and a shorter time to recovery. However, these techniques were introduced only recently, and further studies are needed to assess their reliability, reproducibility, and potential for iatrogenic injury [22–24].

3.2 Tools (Fig. 3.1)

The technique is performed with the 4 mm 30° angle arthroscope because of a better view, and the laxity usually allows a complete exploration of the joint. Arthroscopic dissection is performed using a 4.5 mm bone/soft tissue shaver blade. Suture passers and push knot are helpful. This

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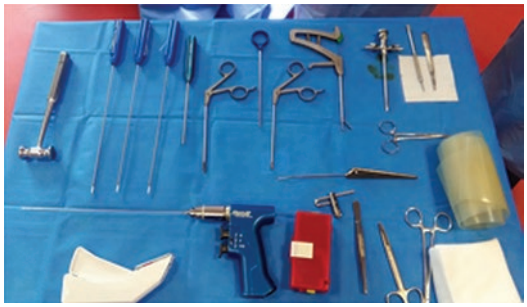

Fig. 3.1 Standard tools

technique can be performed with different types of anchors: with knot and knotless [10–17] (Fig. 3.1).

3.2.1 Patient Positioning

Two installations are possible: in a prone position or in lateral decubitus. If the patient is placed in a prone position, a bag must be positioned under the buttock to have the foot in a vertical position and avoid automatic external rotation and having access to the lateral aspect of the ankle. In case of lateral decubitus position the patient is placed with the pelvis slightly rotated 30° posterior. Position 1 is used for anterior arthroscopy. The hip is externally rotated. Position 2 is used for the lateral hindfoot endoscopy. The hip is internally rotated (Fig. 3.2).

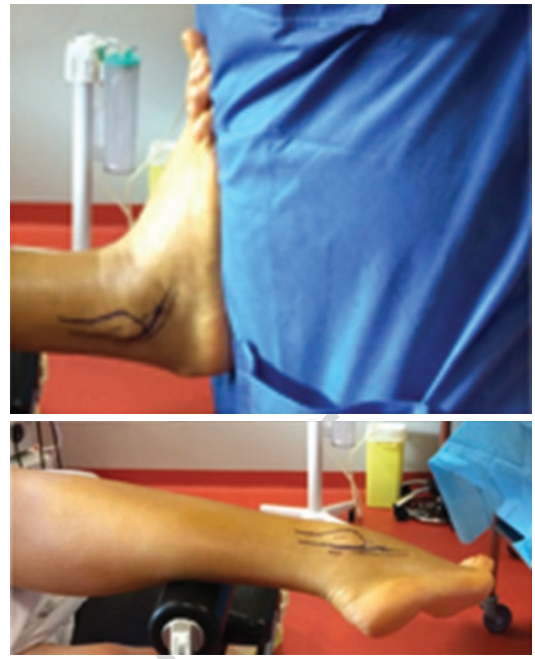

Fig. 3.2 Patient setting in lateral position

3.2.2 Landmarks: Identification and Marking of Portals

Three portals are usually created to perform the procedure. The anteromedial portal is the first portal (portal 1). It has to be made medial to the tibialis anterior tendon, in hyperdorsal flexion of the ankle in order to have the portal as much lateral as possible. In this way, the anterior working area is bigger, the cartilage is protected because of the dorsiflexion, and the tibialis anterior tendon is at the most lateral position.

After ankle joint exploration, the second portal is the accessory anterolateral portal (portal 2) which is not marked on the skin as it is made under transillumination guidance when the arthroscope is positioned in portal 1 and viewing the lateral gutter. The placement of this portal is between the spotlight and malleolus (Fig. 3.3). The third portal is the sinus tarsi portal (portal 3). Through the sinus tarsi portal, it is possible to have a full access to the lateral aspect of the ankle and to have a complete vision of the inferior extensor retinaculum (IER). Portal 3 is made 1 cm anteriorly to the mid-distance point between the tip of the fibula and the proximal tip of the fifth metatarsal (Fig. 3.4).

3.2.3 Step 1: Anterior Arthroscopy, Making the Broström Repair

The arthroscope is introduced in portal 1. Once the arthroscope is perfectly well centered on the external gutter, portal 2 is positioned between the spotlight and the lateral malleolus. For the realization of this portal, we can use a needle. The position should be anteriorly to the malleolus in the external gutter above the ATFL (Fig. 3.5). A mosquito clamp is introduced using the nick and spread technique. A debridement is then begun with the shaver. The resection starts with the scar

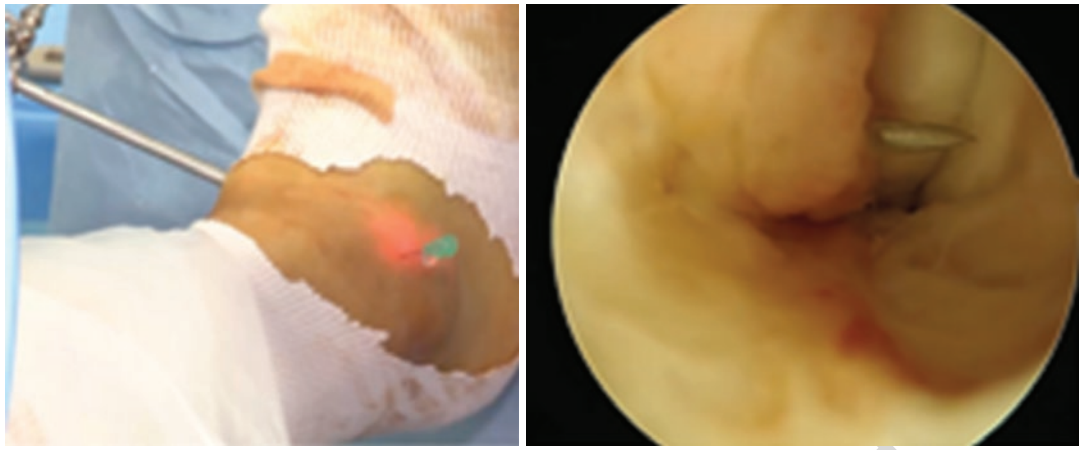

Fig. 3.3 Portal 2 by transillumination

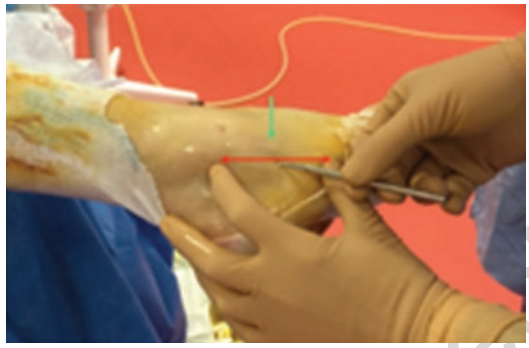

Fig. 3.4 Portal 3

tissue in the lateral gutter. The first anatomical landmark is the distal fascicle of the anterior tibiofibular ligament (Basset ligament) that always appears as an oblique structure between the anterolateral edge of the distal tibia and the lateral malleolus (Fig. 3.5a). Following this ligament from medial to lateral and from proximal to distal, it is easy to reach the malleolar insertion of the anterior talo-fibular ligament (ATFL) [25]. It is important then to move backward the scope in order to visualize the talar neck and have a general vision. The other important landmark is the anterolateral corner of the talar dome without cartilage. This landmark is constant and is just above the talar insertion of the ATFL. Then a capsulotomy is performed with a beaver blade between the ATFL and the capsule, at the lateral aspect of the ATFL, from proximal to distal, to get a complete vision of the ATFL from its malleolar insertion to its talar insertion (Fig. 3.5b, c). The ATFL is then peeled off from its malleolar origin (as usually the avulsion is from the malleolar side with scar tissue at this location). The anterior facet of the distal malleolus, at the ATFL footprint, is then prepared with a burr, to enable a good healing of the ATFL reinsertion on the distal malleolus. This preparation of the malleolus is extended from the most distal to the distal insertion of the anterior tibiofibular ligament. The inferior part of the final malleolar preparation is going to receive the ATFL reinsertion and the superior part will receive the retinaculum augmentation (Fig. 3.6). The first anchor is positioned in the footprint of the ATFL, always with the arthroscope in portal 1, instruments and the anchor by portal 2. The second and/or third anchor will be placed for the Gould augmentation with IER.

The first suture is passed through the ATFL. The stand from the ligament is passed into the loop to obtain a lasso around the portion of ligament (Fig. 3.7) [11–17]. This technical pearl is made to reinforce the suture. The ATFL is then reinserted on the malleolus, with the anchor, with the ankle in a neutral position.

3.2.4 Step 2: Lateral Hindfoot Endoscopy, Making the Gould Augmentation

From the sinus tarsi portal (portal 3) the smooth trocar of the arthroscope is introduced and passed between the IER and the skin to create a working

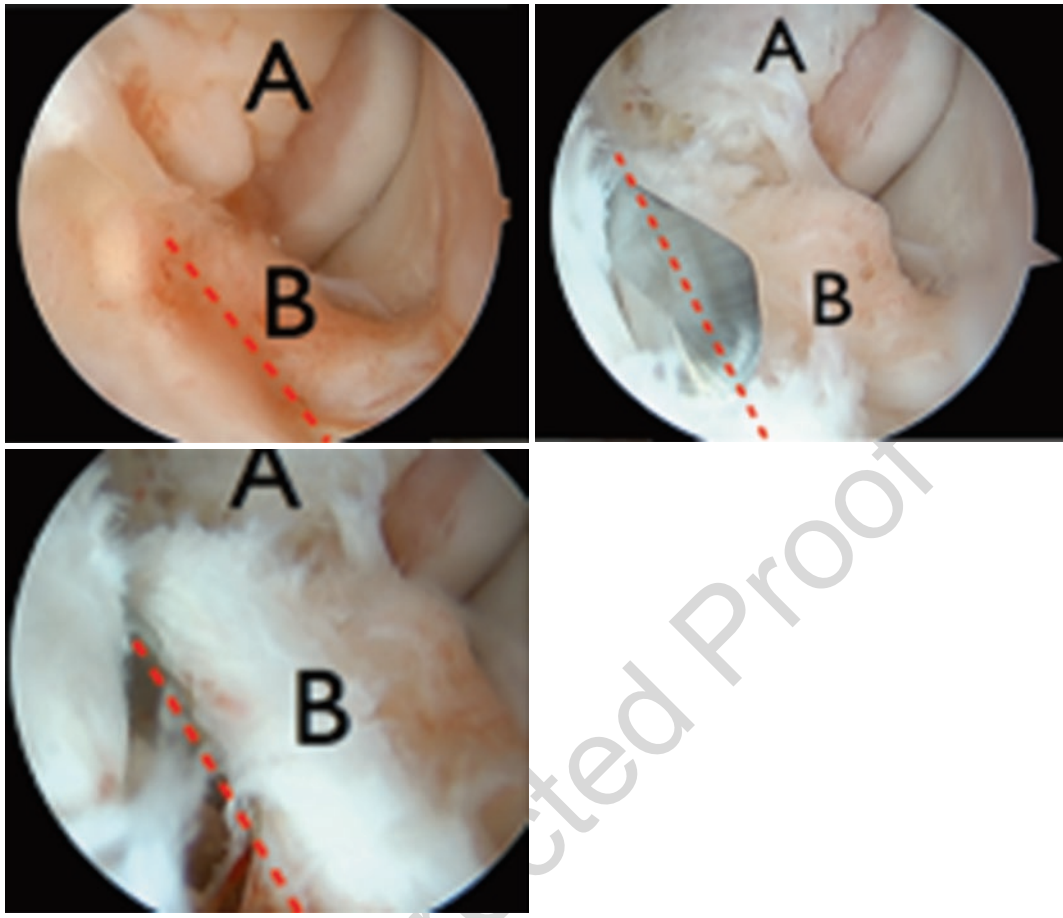

Fig. 3.5 Lateral gutter dissection: visualization of the distal part of the Basset ligament (**a**) and superior bundle of the ATFL (**b**)

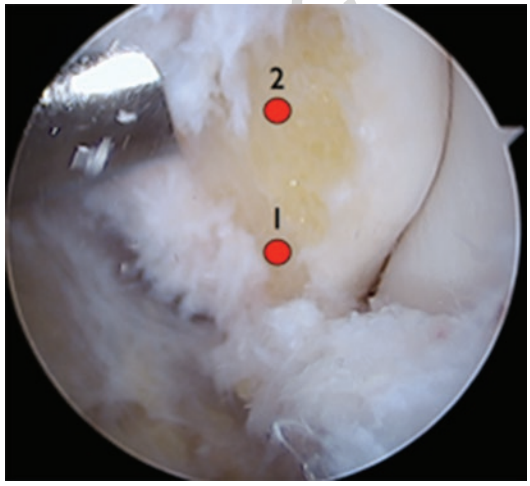

Fig. 3.6 ATFL footprint preparation: positioning for the anchor for ATFL repair (1) and for IER augmentation (2)

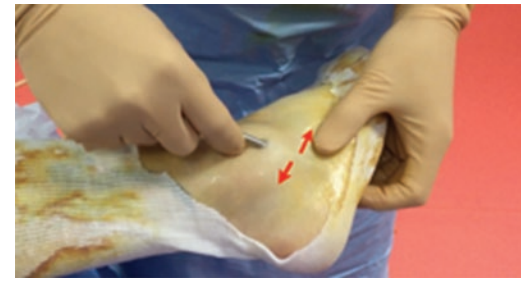

Fig. 3.7 Lasso loop on the ATFL

area around the IER. In this way, the cutaneous nerve stays with the fatty subcutaneous tissue and as it is avascular, there is no vascular or neurological danger (Fig. 3.8).

The arthroscope is then positioned in portal 3, looking at portal 2 from inferior to superior. A

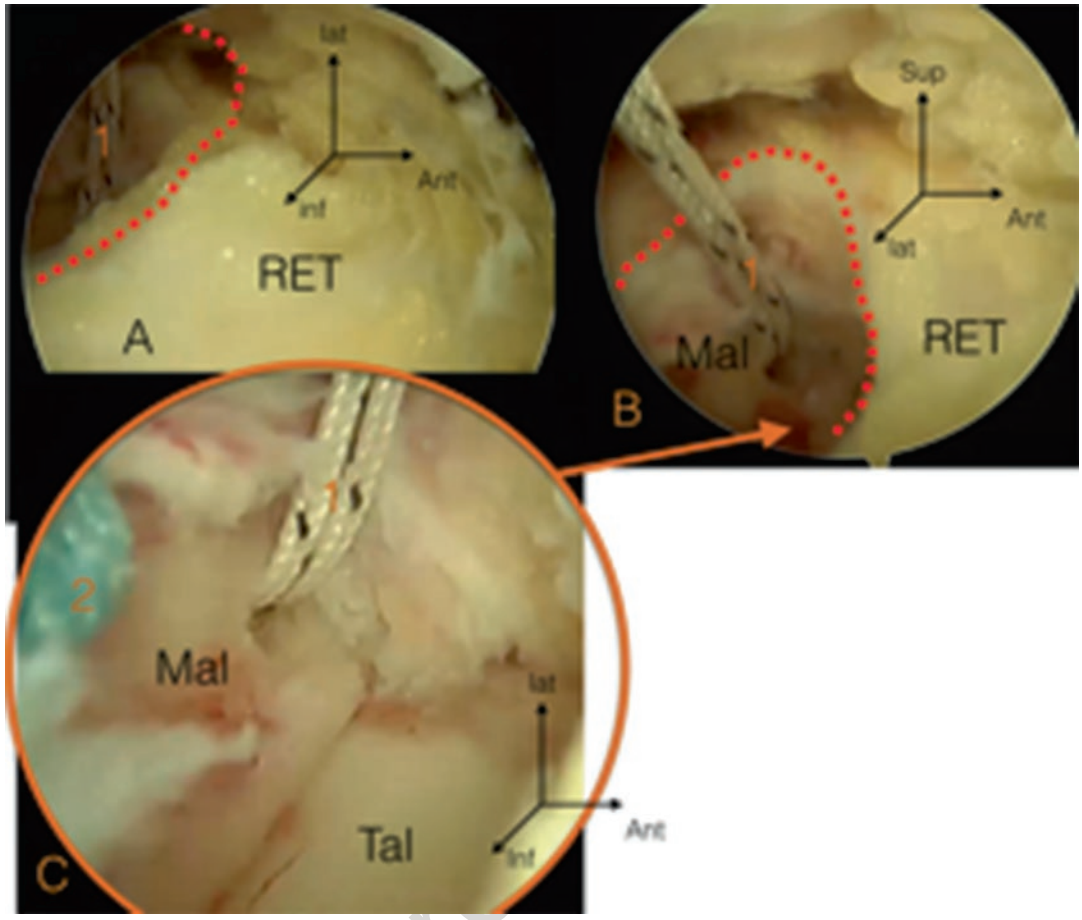

Fig. 3.8 Preparation of the working area for IER dissection

shaver introduced by portal 2 is finishing the preparation and dissection of the IER. The window of the shaver must always be under arthroscopic vision. It is important to obtain a perfect visualization of the IER as well as the hole created in step 1 via the portal 2 to know where the augmentation has to be placed with accuracy and safety. It is important to see on one side the prepared malleolus and on the other side the IER, ready to be sutured on the malleolus above the ATFL repair. More deeply, it is possible to have a vision of the Broström repair and more superiorly the lateral side of the talus (Fig. 3.9).

The second anchor is then introduced by portal 2 and placed on the anterior part of the malleolus at 1 cm superior to the previous anchor in the prepared zone. Once the anchor is inserted, the suture is passed into the IER. By passing the 2 strands, it is possible to realize a mattress suture. It is possible to add a second anchor more inferiorly to have two fixations in the IER. In this case, it is important to put the anchor before doing the knot of the first one. The suture is tight on the malleolus to create the augmentation on the ATFL repair (Fig. 3.10).

3.2.5 Postoperative Care

ATFL repair is performed in outpatients. The patient is immobilized in a normal brace with immediate full weight bearing as tolerated. Foot elevation and ice are required for the first 2 weeks

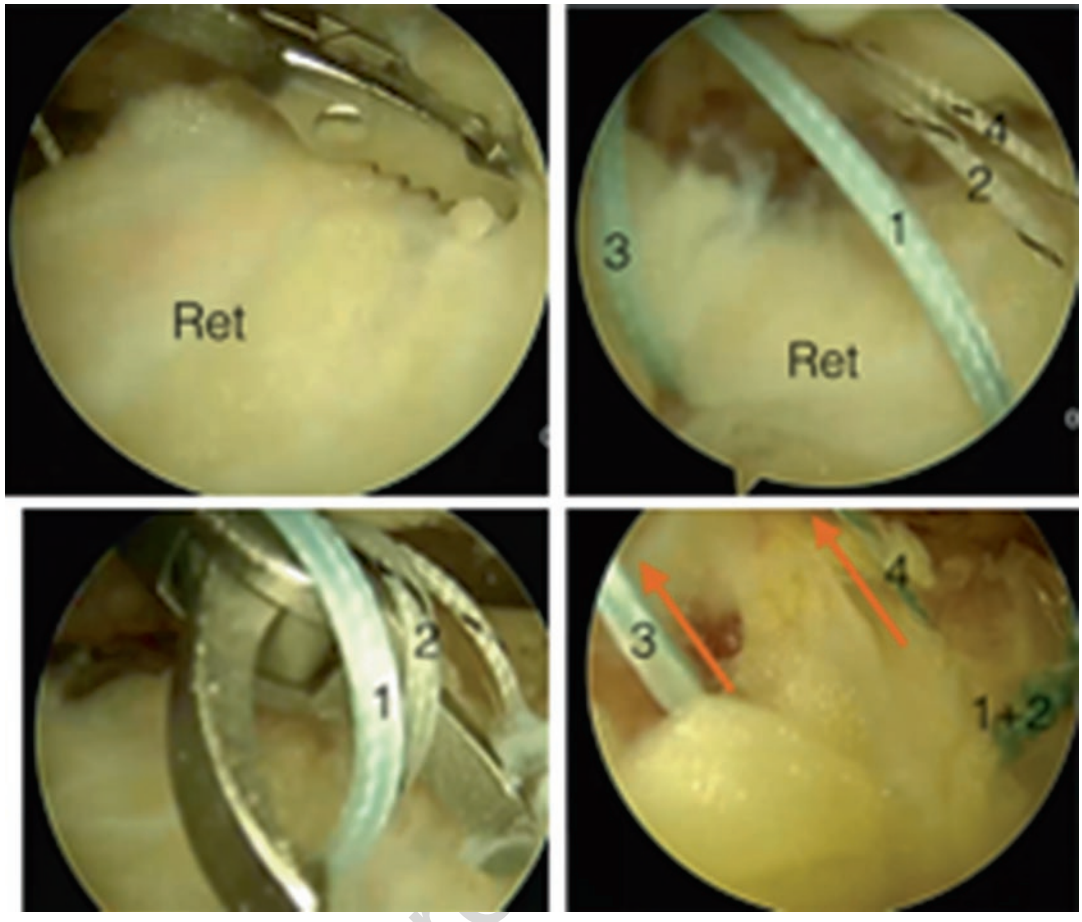

Fig. 3.9 Suture in the IER

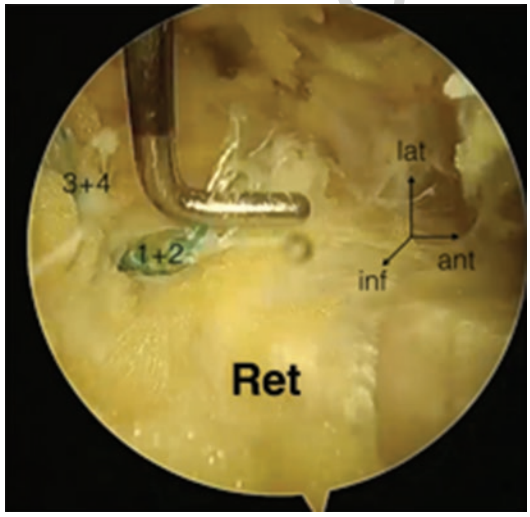

Fig. 3.10 Gould augmentation: Anchor in the malleolus (mal). Suture in the IER (ret)

to avoid swelling and pain. Rehabilitation is begun after 3–4 weeks for mobilization and proprioception. Return to sports activities is allowed after 6 weeks depending on the pain.

3.3 Discussion

Arthroscopy is gradually moving to a central position in the management of CAI, as it allows the diagnosis and treatment of concomitant lesions and, most importantly, provides a more accurate assessment of ATFL lesions, thereby guiding the treatment decision. Although arthroscopic techniques have not been proven superior over conventional open ligament repair and reconstruction, arthroscopy deserves to be viewed as a technique of choice for the treatment

of CAI, as it provides a comprehensive assessment of the ligament lesions and helps to choose the optimal surgical technique [19].

Arthroscopy improves the evaluation of lesions to the lateral ligament complex. Arthroscopic findings have modified the concept of anterolateral impingement by showing that the cause is micro-instability or rotational instability, which cannot be detected on imaging studies [26–28]. Arthroscopic exploration of the talo-fibular gutter is simple to perform and is conducted as the first step of the procedure to allow an evaluation of the ligament lesions [25]. When the ATFL is present and of good quality, or is distended or avulsed but exhibits good mechanical resistance, ATFL repair with or without advancement of the extensor retinaculum can be performed. In contrast, if the ATFL is thin, fragile, or absent, with a bald malleolar tip and abnormally good visibility of the talo-fibular gutter and fibular tendons, anatomic reconstruction with tendon grafting is in order. Thus, simple arthroscopic exploration provides definitive objective criteria for choosing the surgical technique best suited to the ligament lesions.

These arthroscopic techniques are simple and reproducible, as they are performed by anterior arthroscopy without distraction [9]. The learning curve of arthroscopic ATFL repair is quite short and the different steps must be carefully respected.

Arthroscopic ATFL repair, with or without extensor retinaculum advancement, is indicated if the ATFL is present and of good quality [10–17, 24]. These arthroscopic ATFL repair techniques carry a lower risk of cutaneous and infectious complications compared to open surgery [22–24]. The main complication of arthroscopic ATFL repair is injury to the superficial fibular nerve, which occurred in 4.3% of a recent prospective study of 286 cases, about half the rate reported with open surgery [24, 29–31]. Superficial fibular nerve injury usually manifests chiefly as transient dysesthesia, whose frequency is similar to that seen after any anterior ankle arthroscopy procedure [32]. No increase in the risk of nerve injury was seen in patients managed with versus without extensor retinaculum advancement or with versus without knots [24, 33–36].

The main difficulty is the patients selection in order to know if ATFL repair remains the best option for each case. Further assessment with longer follow-up is in progress to have better indications and results of this arthroscopic technique.

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4 Arthroscopic Ligament Repair and Reconstruction

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Surgical treatment is sometimes required to treat the chronic lateral instability of the ankle to prevent the development of articular cartilage damage [1–3] and to prevent performance deterioration especially in toe-off phase. Recently, arthroscopic repair/reconstruction surgery for lateral instability of the ankle has been rapidly developing.

In this chapter, I describe about how to decide whether repair or reconstruction before surgery, the technique of all inside arthroscopic Broström repair with a reinforcement by inferior extensor retinaculum (arthroscopic Broström-Gould repair) [4–6], and the technique of anatomical reconstruction of the lateral ligament of the ankle (AntiRoLL).

4.1 How to Decide Repair or Reconstruction

Choice of a surgical procedure is done by evaluating the quality of the residual ligament with stress ultrasonography before surgery (Fig. 4.1a, b), and determined with arthroscopic evaluation during surgery (Fig. 4.2). Arthroscopic Broström-Gould repair is selected if the ligament fibers remain, and anatomical reconstruction of the lateral ligament of the ankle (AntiRoLL) is selected if there is no ligament fiber.

4.2 Arthroscopic Broström-Gould Repair

4.2.1 Position

The position is supine, and the lower leg is held with a leg holder (Fig. 4.3a). If it is needed to treat the intra-articular concomitant lesions including osteochondral lesions of the ankle and/or free body, distraction device is used according to the condition of the lesions (Fig. 4.3b). The tourniquet is not normally used, but it should be worn on the thigh for use when the field of vision is hindered by bleeding. The ankle position should be kept slightly dorsiflexion by surgeon's belly to widen the lateral pouch for pleasant view and enough working space (Fig. 4.4a). If the surgeon doesn't have adequate round belly, applying towel and corset is effective (Fig. 4.4b, c).

4.2.2 Surgical Procedure

4.2.2.1 Step 1: Making Portals

Medial midline (MML) portal as viewing portal and accessory anterolateral (AAL) portal as working portal are used (Fig. 4.5). MML is put

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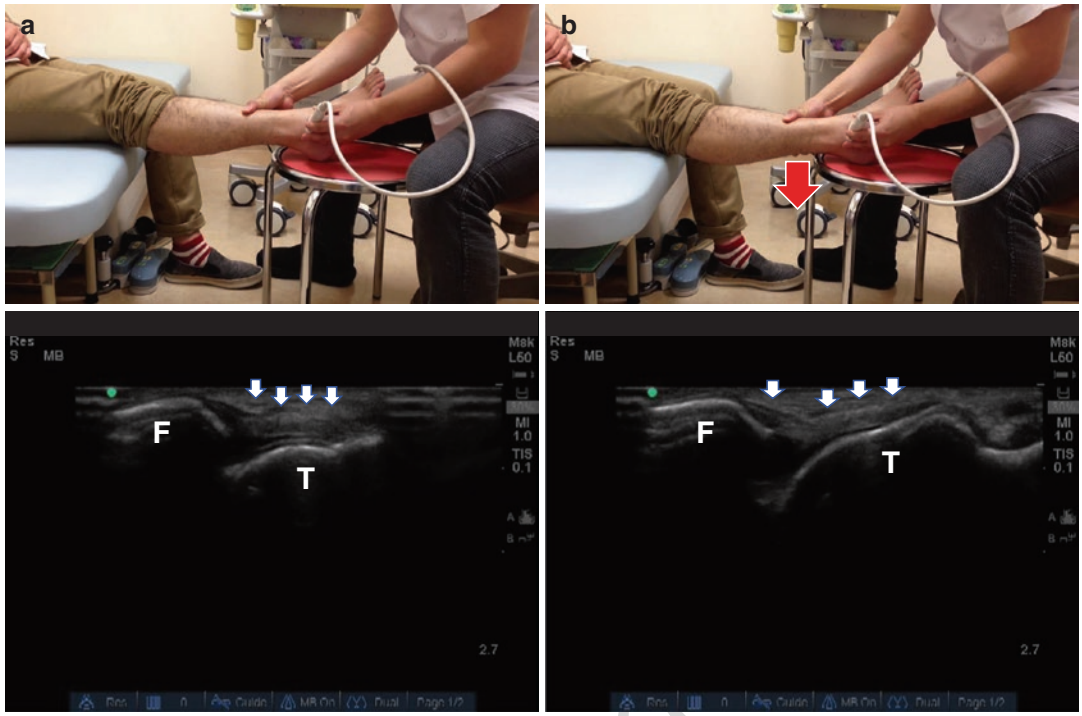

Fig. 4.1 Stress ultrasonography. (**a**) Before applying stress. (**b**) After applying stress. *F* fibula, *T* talus, red arrow: direction of stress force, white arrows: anterior talofibular ligament. ATFL is ruptured at its fibular attachment. After applying stress, fibular and talus separate from each other

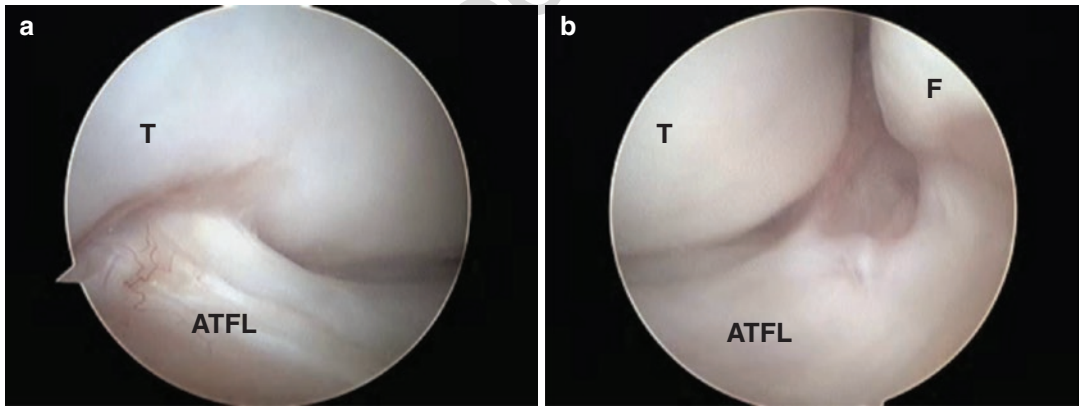

Fig. 4.2 Arthroscopic view of ATFL (same case to Fig. 4.1). (**a**) Talar attachment. (**b**) Fibular attachment. *F* fibula, *T* talus. ATFL is ruptured at its fibular attachment

just lateral of anterior tibial tendon at the level of talocrural joint space. After putting a 5 mm vertical incision through the skin only, capsule is penetrated by a straight mosquito pean. The arthroscope (2.7 mm in diameter, 30° perspective scope) is inserted via the MML portal and viewed in the lateral gutter. In this process, ankle should be in slightly dorsiflexed position for extending

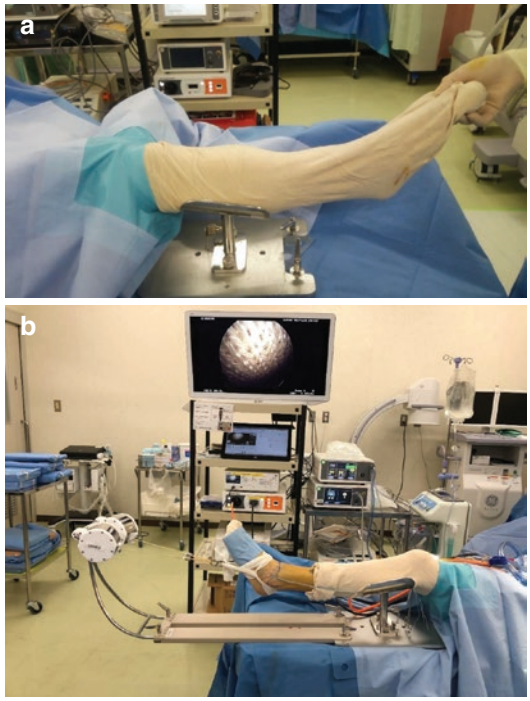

Fig. 4.3 Position. (**a**) In arthroscopic repair. The position is supine, and the lower leg is held with a leg holder. (**b**) In treating the intra-articular concomitant lesions. Distraction device is used according to the condition of the lesions including osteochondral lesions of the ankle and/or free body

the lateral pouch, turning the light cable upside to face the scope for directing the field of view of the arthroscope to the back to obtain a good field of view.

Next an AAL portal is made. A 22G needle is inserted at about 10 mm medial to the fibular obscure tubercle and make sure that the tip of the needle is in the proper position to easily approach the fibular attachment of the ATFL with arthroscopy (Fig. 4.6a) and to put a 5 mm vertical incision through the capsule (Fig. 4.6b).

If the field of vision is hindered by hypertrophic synovium, minimum resection is done using a 3.5 mm motorized shaver so as not to damage the joint capsule and residual ligament.

If it is needed to treat the intra-articular lesions, we add anterolateral (AL) portal.

4.2.2.2 Step 2: Insert a Suture Anchor

After confirming that the ligament fiber of ATFL remains, insert suture anchor for suturing the remaining ligament to fibula attachment. A drill hole is drilled about 5 mm proximally from the distal end of the articular surface of the lateral malleolus and about 5 mm outward from the lateral side of the articular surface (Fig. 4.7). After inserting the anchor suture, it is confirmed that the thread slides.

4.2.2.3 Step 3: Suture Relay Technique

Insert an 18G needle through 2-0 nylon thread via AAL portal and penetrate ATFL remnant fiber from front to back as deeply as possible (Fig. 4.8a). Rotate the needle forward for several times and reverse rotate the same number of times to enlarge the nylon loop (Fig. 4.8b). After that, insert a hook probe from the AAL portal and guide the nylon loop from the AAL portal to the outside (Fig. 4.8c, d).

4.2.2.4 Step 4: Suture the Remnant—Modified Lasso-Loop Stitch

Pass one thread of anchor suture to the nylon loop about 2/3 from the distal end. By pulling both ends of the nylon thread, the thread of anchor suture is looped through the remaining ligament (Fig. 4.9a). Rotate this loop half a turn; first pass the anchor suture thread on the opposite side (Fig. 4.9b). Then turn the loop again, pass the anchor suture thread on the same side through this second loop (Fig. 4.9c), pull the end of the anchor suture thread on the same side as the loop, and lightly tighten the loop (Fig. 4.9d). Finally, make the ankle at 0° neutral position and strongly pull the end of the anchor suture thread on the opposite side. Then the stump of the remaining ligament is crimped onto the fibular attachment, and at the same time the thread is appropriately slipped in the nodule and the knot is tighten strongly (Fig. 4.9e). After three more knot sutures are added, unnecessary threads are removed using a line cutter (Fig. 4.9f).

Fig. 4.4 Position of the ankle. (**a**) The ankle position should be kept slightly dorsiflexed by surgeon's round belly to widen the lateral pouch for pleasant view and enough working space. (**b**, **c**) If the surgeon doesn't have adequate round belly, applying towel and corset is effective

ATFL and CFL are connected with lateral talocalcaneal ligament [7] and attach together to fibula (Fig. 4.10a). And the rupture site in most cases of lateral instability of the ankle is close to fibular attachment [8] (Fig. 4.10b). Accordingly, CFL is automatically moved to its fibular attachment and will recover to work well after ATFL suture alone (Fig. 4.10c).

4.2.2.5 Step 5: Gould Augmentation

Recently we add a Gould augmentation to Broström repair to reduce the stress for repaired ligament in early phase after surgery.

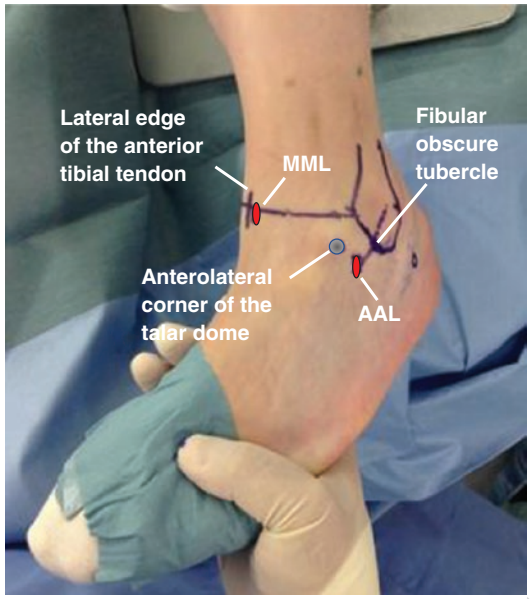

Fig. 4.5 Portals. *MML* medial midline portal, *AAL* accessory anterolateral portal

Insert second suture anchor about 5 mm proximal from the first suture anchor insertion (Fig. 4.11a). Since the upper edge of the inferior extensor retinaculum is close to the AAL portal, blunt dissection is performed on the surface layer and the deep layer of the inferior extensor retinaculum using mosquito pean or a blunt rod. And after touching the upper edge of the inferior extensor retinaculum, it is grasped by mosquito pean (Fig. 4.11b). After attaching the end of one thread of second suture anchor to a semicircular needle (Fig. 4.11c), insert the tip of the needle from the AAL portal and penetrate from the deep side of the inferior extensor retinaculum to the skin (Fig. 4.11d). Pull out the thread on the skin, remove the needle (Fig. 4.11e), then grip this thread with mosquito pean inserted subcutaneously from the AAL portal, and pull it out of the AAL portal (Fig. 4.11f). At this point, one thread of second suture anchor penetrates the inferior extensor retinaculum from the deep layer to the surface layer and is led out from the AAL portal. Then, a sliding knot technique (Navy knot) is performed. It is desirable to use as small a sliding

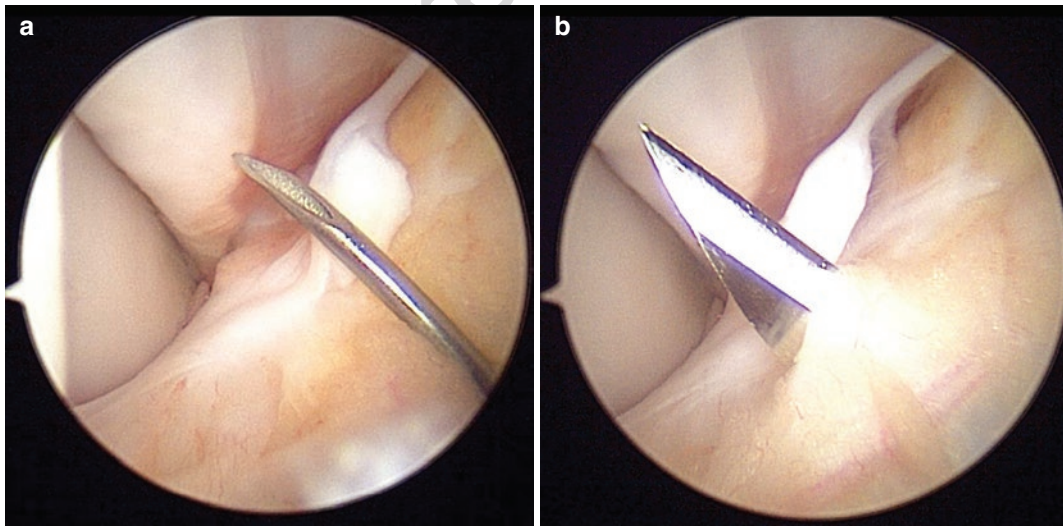

Fig. 4.6 Make an AAL portal. (**a**) Insert a needle. A 22G needle is inserted at about 10 mm medial to the fibular obscure tubercle and make sure that the tip of the needle is in the proper position to easily approach the fibular attachment of the ATFL with arthroscopy. (**b**) Put a 5 mm vertical incision through the capsule

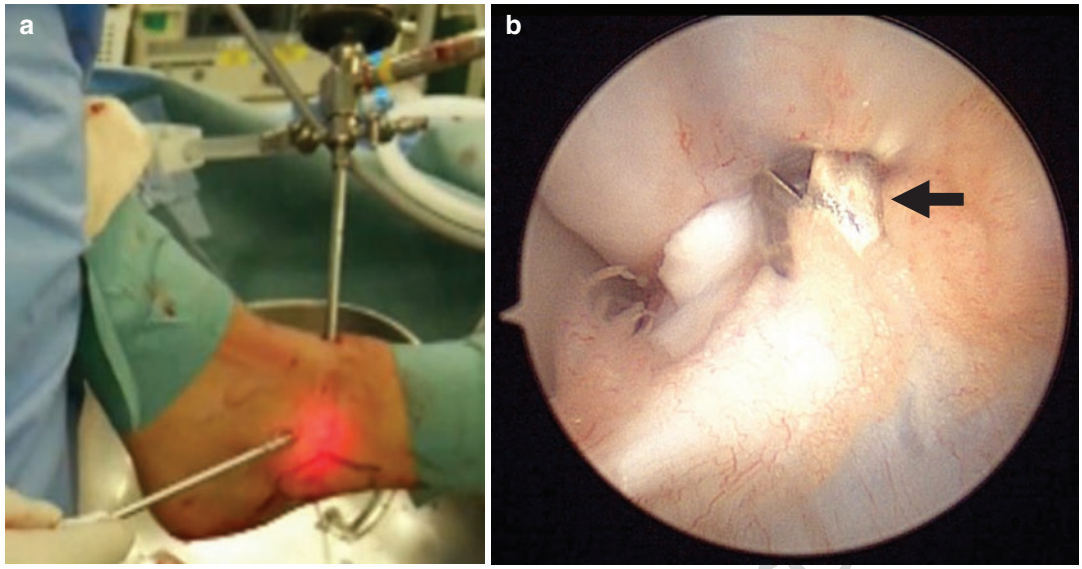

Fig. 4.7 Placement of the suture anchor for ATFL suture. (**a**) Viewing portal is MML and working portal is AAL. (**b**) Arthroscopic view. Arrow shows an inserted suture anchor

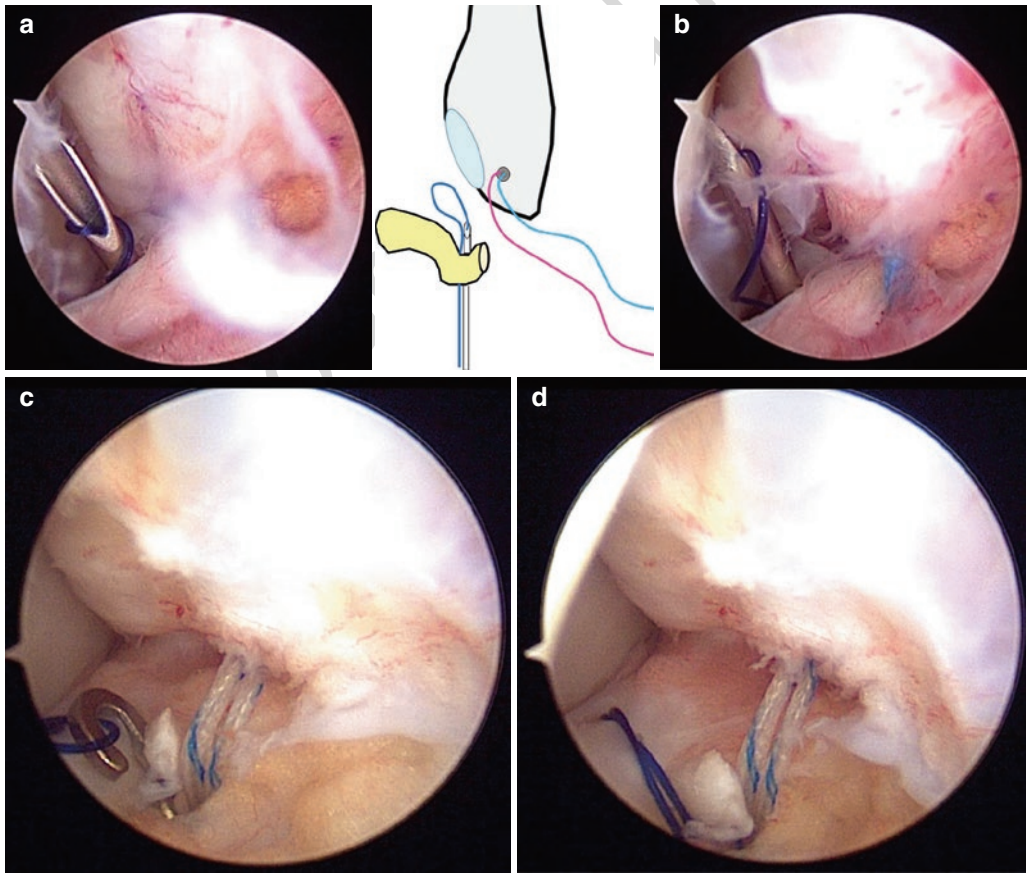

Fig. 4.8 Suture relay technique. (**a**) Insert an 18G needle through 2-0 nylon thread from AAL portal and penetrate ATFL remnant fiber. (**b**) Rotate the needle forward for several times and reverse rotate the same number of times to enlarge the nylon loop. (**c**) Insert a hook probe from the AAL portal. (**d**) Guide the nylon loop from the AAL portal to the outside

knot method as possible to prevent nodules from touching subcutaneously after surgery (Fig. 4.11g). Tighten the sliding knot and cut the thread with a knot cutter (Fig. 4.11h). If the Gould augmentation is completed, the ankle moves about 10° in the dorsiflexion direction when knotting, and after tightening the maximum plantar flexion cannot be achieved passively. But in almost all cases it improves to normal range within 4 weeks after surgery. This is because the inferior extensor retinaculum is loosened within 4 weeks after surgery. So, Gould augmentation should be regarded as a temporary reinforcement after the operation.

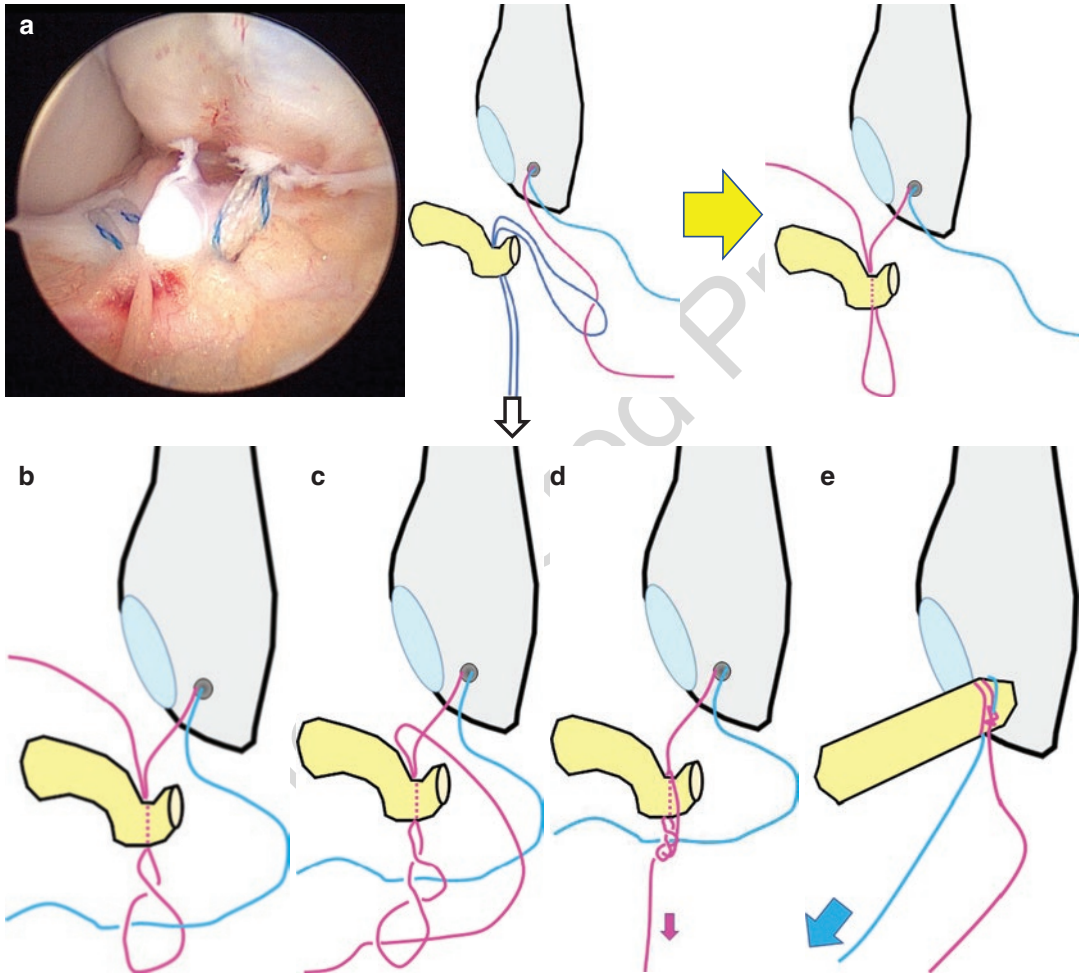

Fig. 4.9 Modified lasso-loop stitch. (**a**) Pass one thread of anchor suture to the nylon loop about 2/3 from the distal end. By pulling both ends of the nylon thread, the thread of anchor suture is looped through the remaining ligament. (**b**) Rotate the loop half a turn, and first pass the anchor suture thread on the opposite side. (**c**) Then turn the loop again, and pass the anchor suture thread on the same side through this second loop. (**d**) Pull the end of the anchor suture thread on the same side and lightly tighten the loop. (**e**) Strongly pull the end of the anchor suture thread on the opposite side. Then the stump of the remaining ligament is crimped onto the fibular attachment and at the same time the thread is appropriately slipped in the nodule and the knot is tightened strongly. (**f**) Cut the suture anchor threads

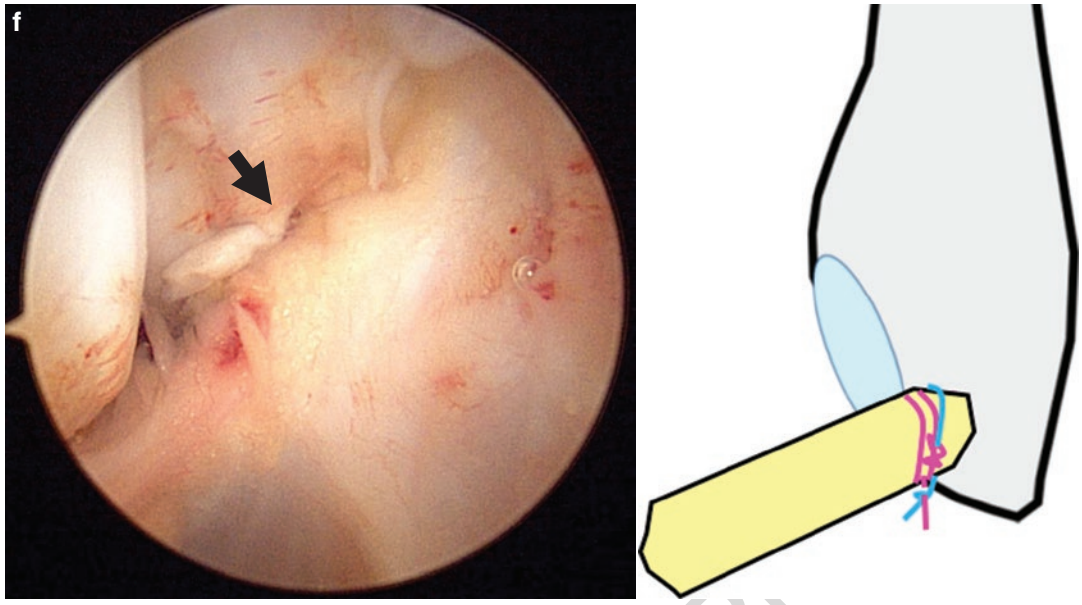

Fig. 9 (continued)

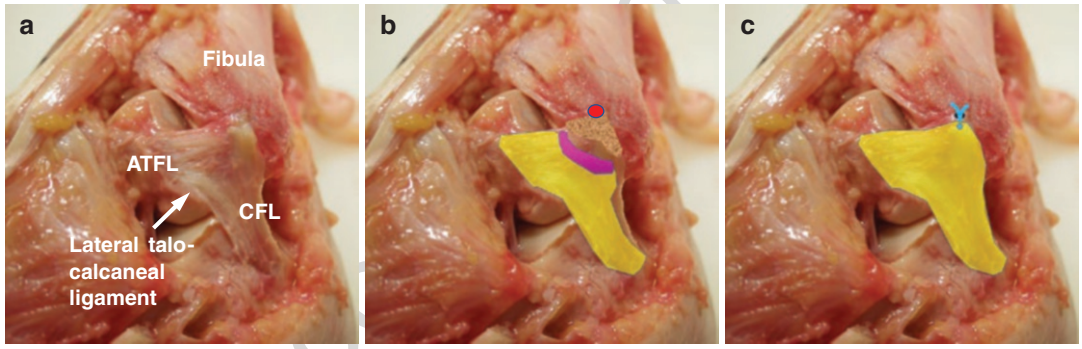

Fig. 4.10 Actual anatomy of the lateral ligament complex. (**a**) ATFL and CFL are connected with lateral talocalcaneal ligament and attached together to fibula. (**b**) The rupture site in most cases of chronic lateral instability of the ankle is close to fibular attachment. (**c**) CFL is automatically moved to its fibular attachment and will recover to work well after ATFL suture alone

If enough stability is obtained with the arthroscopic Broström method, there is no need to add Gould augmentation.

4.2.3 Postoperative Management

After surgery, the elastic bandage is applied for 2 days, and the full weight-bearing walking is allowed according to pain from a day after surgery. Jogging and proprioceptive training will be from 2 weeks postoperatively and return to sports without external fixation shall be after 5 weeks postoperatively.

4.3 Arthroscopic Reconstruction (A-AntiRoLL)

AntiRoLL is the word made by Dr. Glazebrook, aligning underlined parts of the phrase "Anatomical Reconstruction of the Lateral

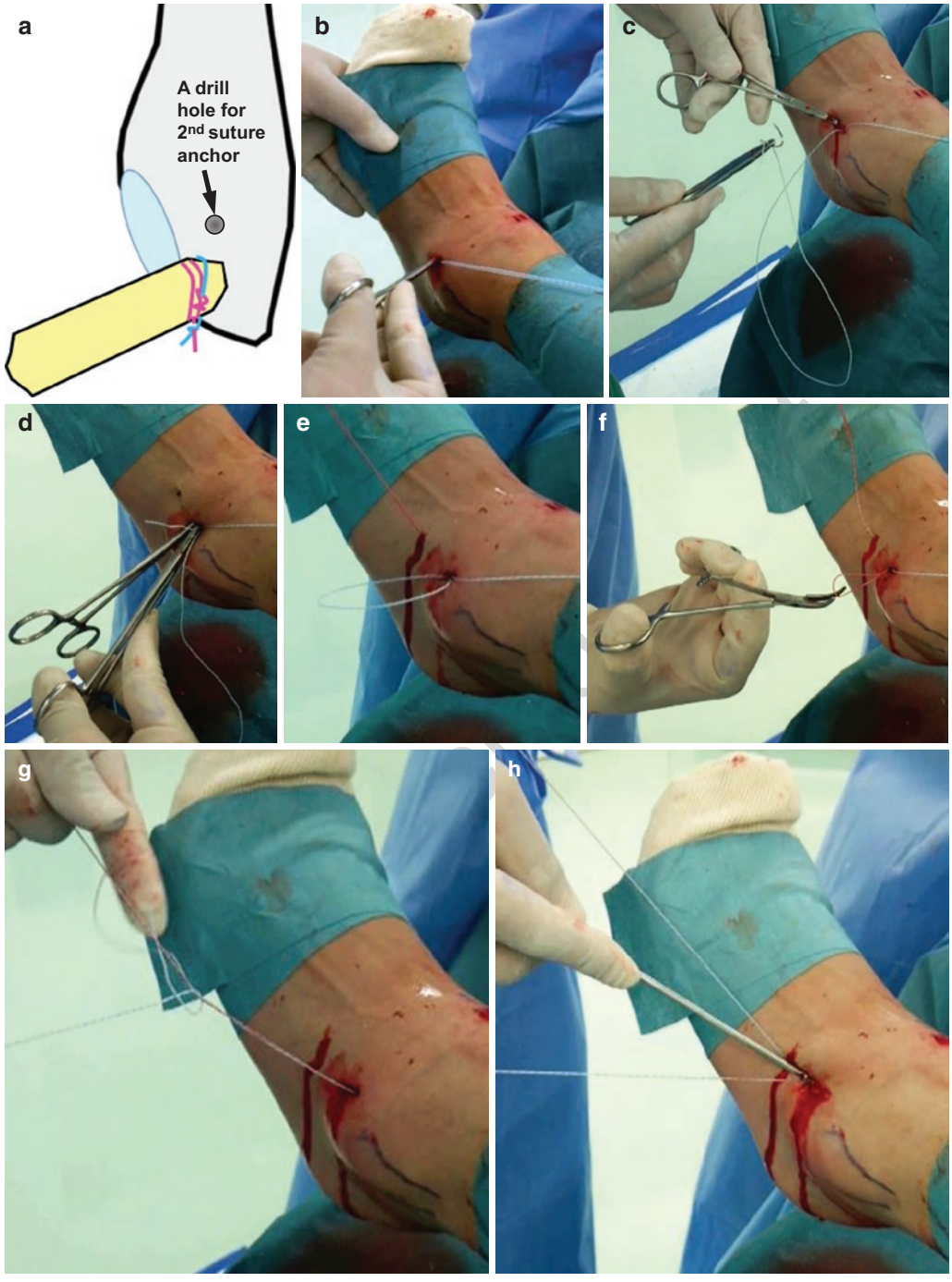

Fig. 4.11 Gould augmentation (reinforcement by inferior extensor retinaculum). (**a**) Insert a second suture anchor at 5 mm proximal to the first suture anchor. (**b**) Blunt dissection is performed on the surface layer and the deep layer of the inferior extensor retinaculum using mosquito pean via AAL portal. (**c**) The upper edge of the inferior extensor retinaculum is grasped by mosquito pean, and attach the end of one thread of second suture anchor to a semicircular needle. (**d**) Insert the tip of the needle from the AAL portal and penetrate from the deep side of the inferior extensor retinaculum to the skin. (**e**) Pull out the thread on the skin and remove the needle. (**f**) Grip the thread with mosquito pean inserted subcutaneously from the AAL portal and pull it out of the AAL portal. (**g**) Sliding knot technique (Navy knot) is performed. (**h**) Tighten the sliding knot and cut the thread with a knot cutter

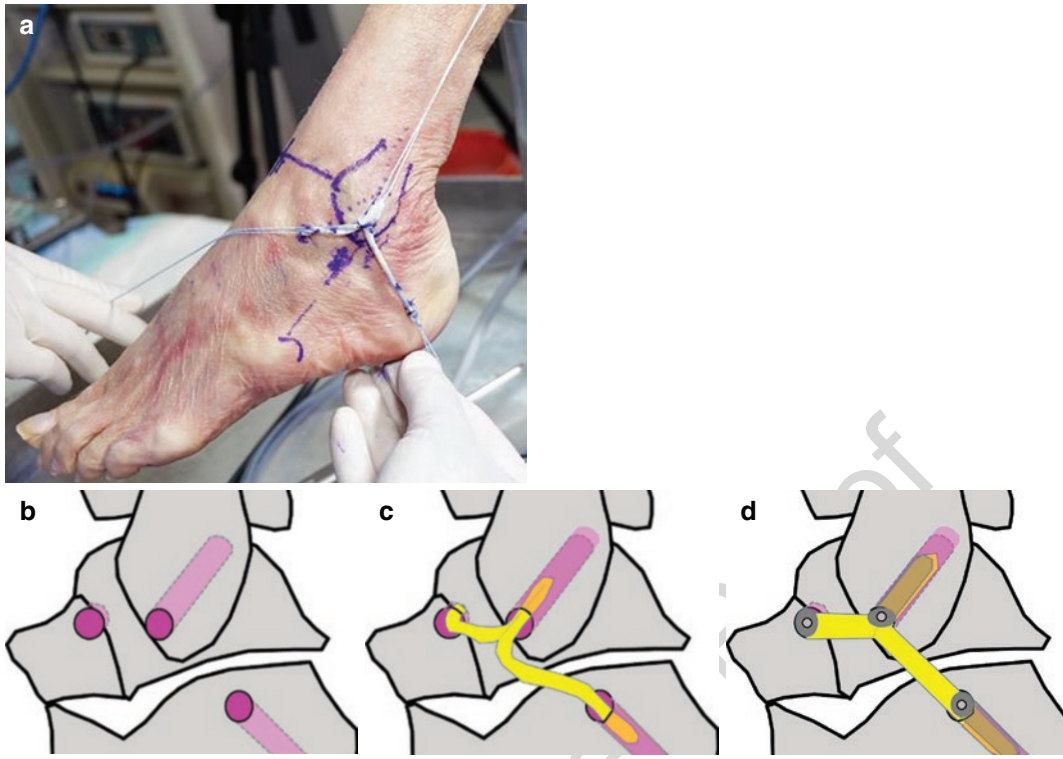

Fig. 4.12 Steps of AntiRoLL. (**a**) Make a Y-shaped graft. (**b**) Make the bone tunnels at each attachment to fibula, talus, and calcaneus. (**c**) Introduce a Y-shaped graft into the bone tunnels. (**d**) Fix with the interference screw

Ligament of the ankle" [9]. There are three types of AntiRoLL, arthroscopic (A-AntiRoLL) [9], percutaneous [10] (P-AntiRoLL), and open AntiRoLL [11].

4.3.1 Surgical Procedure

The position is supine, and the lower leg is held with a leg holder. The tourniquet is not normally used, but it should be worn on the thigh for use when the field of vision is hindered by bleeding.

There are four steps for AntiRoLL; in step 1 make a Y-shaped graft (Fig. 4.12a); in step 2 make the portals; in step 3 make the bone tunnels at each attachment to fibula, talus, and calcaneus (Fig. 4.12b); and in step 4 introduce a Y-shaped graft into the bone tunnels (Fig. 4.12c) and fix with the interference screw (Fig. 4.12d).

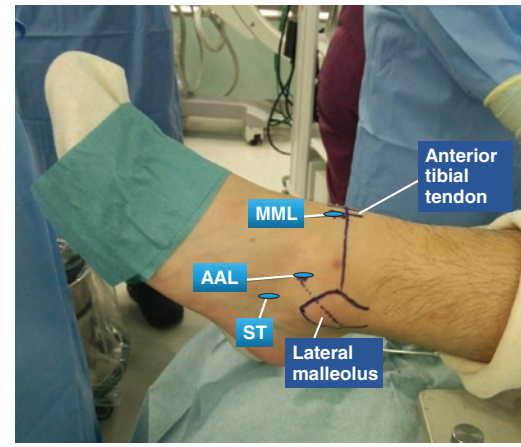

Fig. 4.13 Portals. *MML* medial midline portal, *AAL* accessory anterolateral portal, *ST* subtalar portal

4.3.1.1 Make Portals

Medial midline (MML) portal, accessory anterolateral (AAL) portal, and subtalar portal (ST) are used (Fig. 4.13). If it is needed to treat the intra-

articular lesions, we add additional anterolateral (AL) portal.

4.3.1.2 Make a Y-Shaped Graft

An autologous gracilis tendon is harvested from ipsilateral knee (Fig. 4.14a). Marking is done nine times every 15 mm; the resulting 135 mm length tendon should be needed as a tendon graft (Fig. 4.14b). Next fold back at the site 60 mm from the end, pass the guide thread through this fold, and then suture the tendons with the 3-0 bioabsorbable thread at the position 15 mm from the turning point. Finally fold back at 15 mm from both ends, pass guide thread through folded back, and then suture the tendons with 3-0 bioabsorbable thread (Fig. 4.14c). The short leg of the Y-shaped tendon graft is ATFL and the long leg is CFL.

4.3.1.3 Make the Bone Tunnels at Each Attachment to Fibula, Talus, and Calcaneus

Positioning of each fibular, talar, and calcaneal bone tunnels is made by using the landmarks existing on the bone surface as shown in Fig. 4.15 [12].

In making a fibular bone tunnel, a viewing portal is MML and a working portal is ST (Fig. 4.16a). A landmark for fibular bone tunnel is fibular obscure tubercle (FOT) which exists at the border of the footprints of the ATFL and the CFL (Fig. 4.16b). After to identify a FOT to remove a part of remnant of ATFL using motorized shaver, a guide wire for cannulated drill is inserted via ST portal and it penetrates the center of the fibula at FOT toward the direction of proximal-posterior fibular cortex as 30° to the axis of fibula, and finally penetrates the posterior leg skin (Fig. 4.16a). Next an overdrilling 6 mm in diameter and 20 mm in depth is done using cannulated drill. Finally, a guide wire is replaced to guide thread.

In making a talar bone tunnel, a viewing portal is MML and a working portal is AAL (Fig. 4.17a). A landmark for talar bone tunnel is anterolateral and posterolateral corners of the talar body (Fig. 4.15). On the line to connect the anterolateral and posterolateral corners of the talar body, about 40% inferior point from anterolateral corner of the talar body is the center of the footprint of the ATFL. But in actual cases, there remains a ligament fiber at the attachment of the ATFL to the talus in most cases and it is a good landmark to make a talar bone tunnel. A guide wire for cannulated drill is inserted via AAL portal and it penetrates the center of the footprint at talus (Fig. 4.17b) toward the direction to tip of the medial malleolus, and finally penetrates the skin. Next an overdrilling 6 mm in diameter and 20 mm in depth is done using cannulated drill. Finally, a guide wire is replaced to guide thread.

In making a calcaneal bone tunnel, a viewing portal is ST and a working portal is AAL (Fig. 4.18a). A landmark for calcaneal bone tunnel is the posterior facet of the talocalcaneal joint (Fig. 4.15). On the line perpendicular bisector of the posterior facet, 17 mm inferior point from posterior facet is the center of the footprint of the CFL. But in actual cases, peroneal tendons run just over the insertion of the CFL. To avoid the damage to the peroneal tendons, the authors make a calcaneal bone tunnel proximal to the peroneal tendon sheath, about 10 mm inferior point from posterior facet (Fig. 4.15). A guide wire for cannulated drill is inserted via AAL portal and it penetrates the calcaneus as the direction of center of the posterior corner of the calcaneus, and finally penetrates the posterior heel skin (Fig. 4.18b). Next an overdrilling 6 mm in diameter and 20 mm in depth is done using cannulated drill. Finally, a guide wire is replaced to guide thread.

In this time, a guide thread of the fibular bone tunnel is inserted via ST portal. This thread is grasped by forceps via AAL portal inside the joint and introduced to the AAL portal. Accordingly, all guide threads come out from an AAL portal.

4.3.1.4 Introduce a Y-Shaped Graft into the Bone Tunnels and Fix with the Interference Screw

A Y-shaped graft is introduced and fixed into the bone tunnels, firstly fibula, next talus, and finally calcaneus. It is important to insert a guide wire for interference screw before introducing a graft

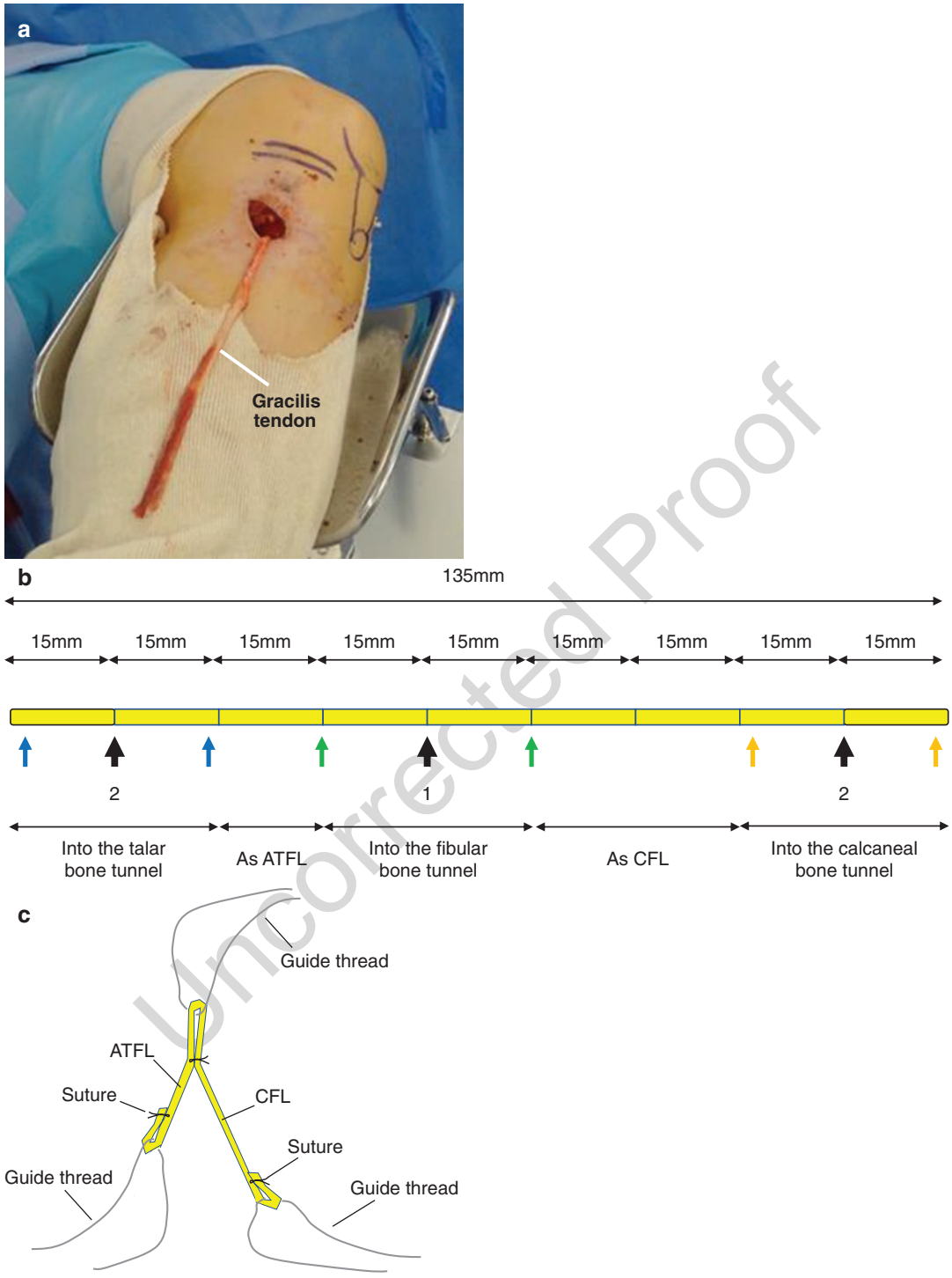

Fig. 4.14 Make a Y-shaped tendon graft. (**a**) An autologous gracilis tendon is harvested from ipsilateral knee. (**b**) Marking on the harvested graft. Marking is done nine times every 15 mm; the resulting 135 mm length tendon should be needed as a tendon graft. Black arrow: point to fold back, green arrow: marking suture for fibular bone tunnel, blue arrow: marking suture for talar bone tunnel, yellow arrow: marking suture for calcaneal bone tunnel. (**c**) The short leg of the Y-shaped tendon graft is ATFL and the long leg is CFL

into the bone tunnels to prevent the graft from being penetrated by a guide wire following graft damage by an interference screw.

Viewing a fibular bone tunnel via MML portal, a fibular end of a Y-shaped graft is introduced into the fibular bone tunnel using guide thread with inside-out technique at the level of the suture which ties the graft at the position 15 mm from the turning point (Fig. 4.19a, b). A graft is fixed into the bone tunnel with an interference screw 6 mm in diameter and 15 or 20 mm in length (Fig. 4.19c, d).

Viewing a talar bone tunnel via MML portal, a talar end of a Y-shaped graft is introduced into the talar bone tunnel using guide thread with inside-out technique at the level of the suture which ties the graft at the position 15 mm from the turning point. Tension the transplantation tendon by manually pulling the guide thread with the ankle at the position of 0 degrees in axial motion, a graft is fixed into the bone tunnel with an interference screw 6 mm in diameter and 15 or 20 mm in length (Fig. 4.19e).

Viewing a calcaneal bone tunnel via ST portal, a calcaneal end of a Y-shaped graft is introduced into the calcaneal bone tunnel using guide thread with inside-out technique at the level of the suture which ties the graft at the position 15 mm from the turning point. As ankle positioned in 0° neutral position and to tensile the tendon graft pulling a guide thread manually, a graft is fixed into the bone tunnel with an interference screw 6 mm in diameter and 15 or 20 mm in length (Fig. 4.19f).

All guide thread can be removed easily to cut the one end near the skin and pull the other end manually.

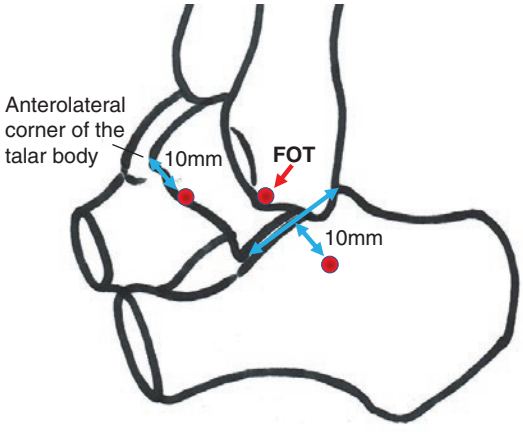

Fig. 4.15 Landmarks for each bone tunnel. A landmark for fibular bone tunnel is fibular obscure tubercle (FOT) which exists at the border of the footprints of the ATFL and the CFL. For talar bone tunnel, on the line to connect the anterolateral and posterolateral corners of the talar body, about 40% inferior point from anterolateral corner of the talar body is the center of the footprint of the ATFL. For calcaneal bone tunnel, on the line perpendicular bisector of the posterior facet, 10 mm inferior point from posterior facet should be a landmark for calcaneal bone tunnel to avoid damage to the peroneal tendons

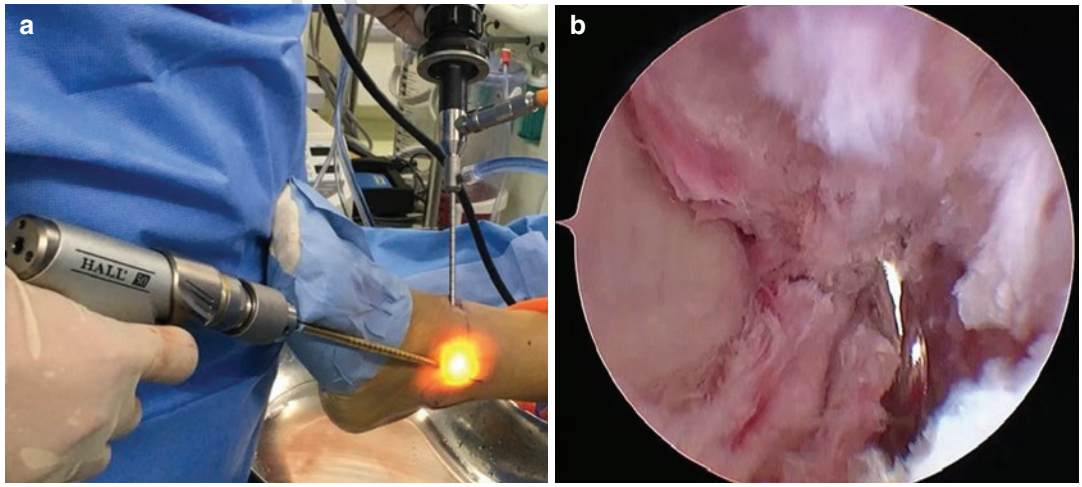

Fig. 4.16 Make a fibular bone tunnel. (**a**) A viewing portal is MML and a working portal is ST. (**b**) Arthroscopic view of the fibular obscure tubercle (FOT)

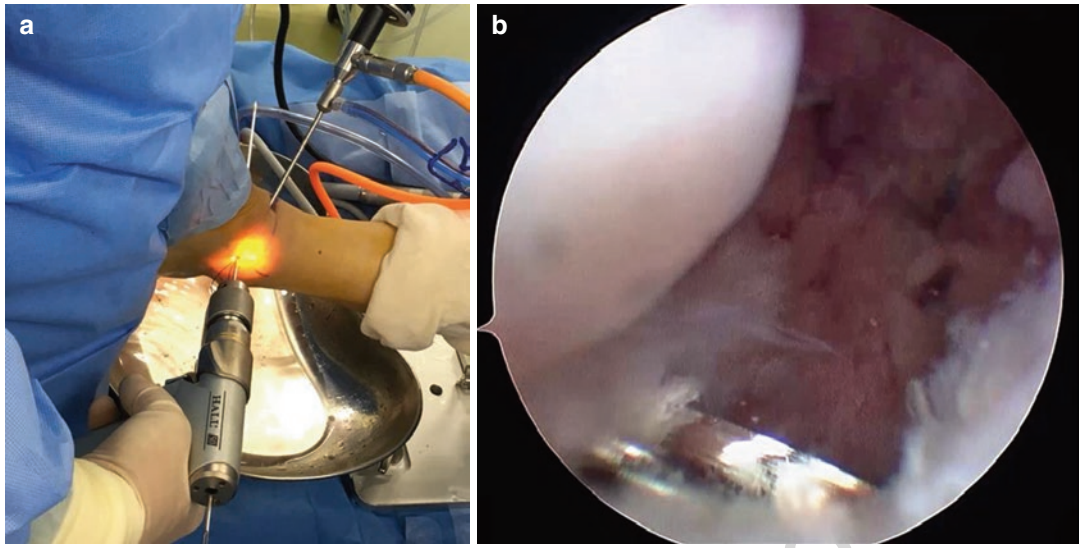

Fig. 4.17 Make a talar bone tunnel. (**a**) A viewing portal is MML and a working portal is AAL. (**b**) Arthroscopic view of the remaining ligament fiber at the attachment of the ATFL to the talus

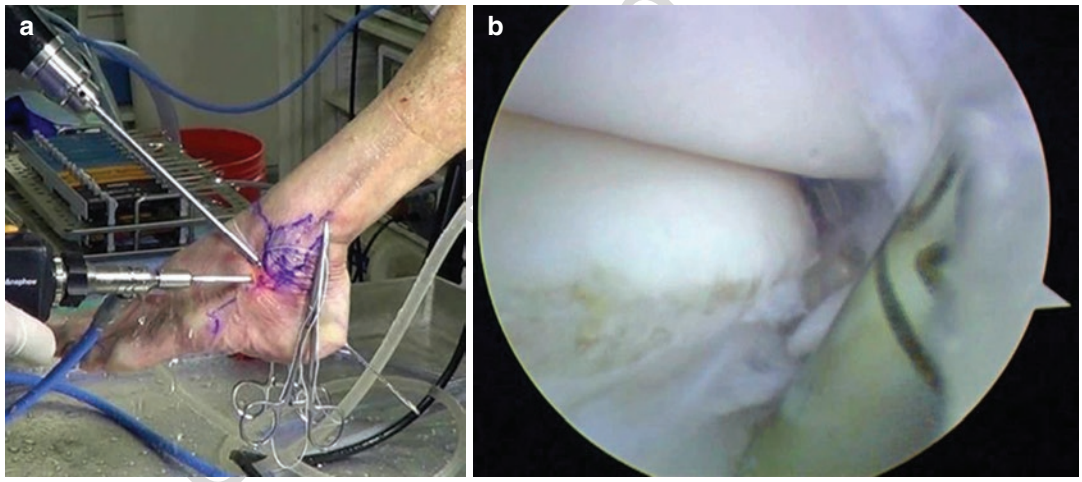

Fig. 4.18 Make a calcaneal bone tunnel. (**a**) A viewing portal is ST and a working portal is AAL. (**b**) Arthroscopic view of the posterior facet and the point to make a bone tunnel

4.3.2 Postoperative Management

After surgery, the elastic bandage is applied for 2 days, and the full weight-bearing walking is allowed according to pain from a day after surgery. The bone tendon attachment gradually increases the strength of biological bonding and becomes nearly normal strength at 4 weeks postoperatively [13]. Accordingly, jogging and proprioceptive training will be from 4 weeks postoperatively and return to sports without external fixation shall be after 6–8 weeks postoperatively.

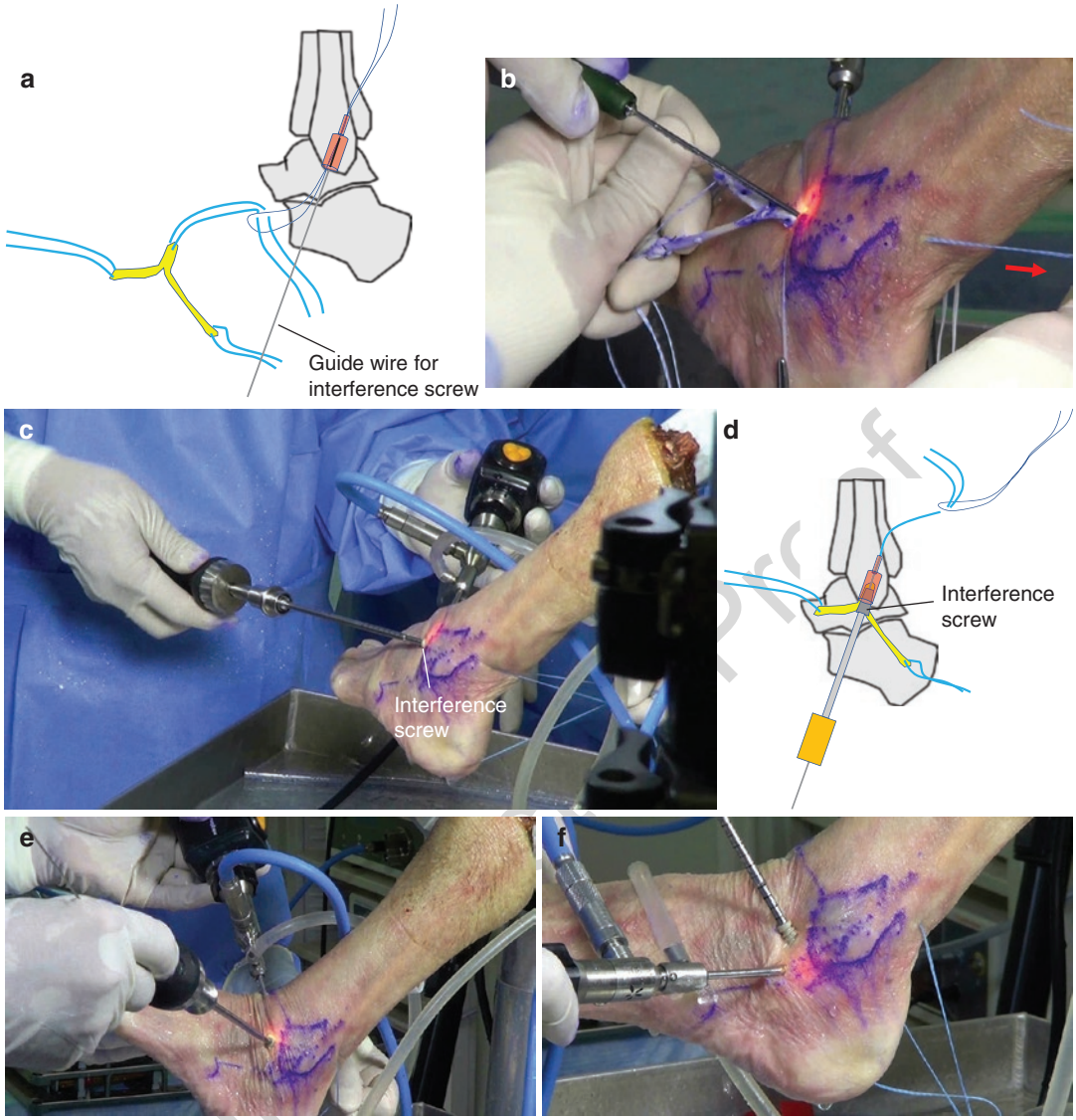

Fig. 4.19 Introduce a Y-shaped graft into the bone tunnels and fix with the interference screw. (**a**, **b**) A fibular end of a Y-shaped graft is introduced into the fibular bone tunnel using guide thread with inside-out technique at the level of the suture which ties the graft at the position 15 mm from the turning point. (**c**, **d**) A graft is fixed into the bone tunnel with an interference screw 6 mm in diameter and 15 or 20 mm in length. (**e**, **f**) Fix a graft into talar bone tunnel. (**f**) Fix a graft into calcaneal bone tunnel

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5 Mini-Incision Technique for Lateral Ankle Ligament Repair in Chronic Instability

Gian Luigi Canata, Valentina Casale, and Luca Pulici

Abbreviations

ATFL	Anterior talofibular ligament
CFL	Calcaneofibular ligament
FAAM	Foot and ankle ability measure
MRI	Magnetic resonance imaging
PTFL	Posterior talofibular ligament
ROM	Range of movement

5.1 Background and Classification

Ankle sprain is one of the most common injuries sustained during sports activities [1]. It is most frequently reported by basketball, soccer, running athletes and ballet/dancers [2] accounting for 40% of all athletic injuries [3, 4]. In the systematic review of Fong et al., the ankle was the most commonly injured area of the body in 24 of 70 sports analyzed [5].

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Three-quarters of the ankle injuries comprise the lateral ligamentous complex [6, 7] (Fig. 5.1).

Among the three lateral ankle ligaments, the anterior talofibular ligament (ATFL) torn occurs in 80% of cases, while the other 20% of ankle lateral sprains involve both the ATFL and the calcaneofibular ligament (CFL) [8]. The posterior talofibular ligament (PTFL) is less commonly injured [8].

Over the years, many classifications of ankle sprains and ligamentous injuries have been proposed [9, 10].

Traditionally, ankle sprains are classified as grade I (mild), II (moderate), or III (severe) [11–13]. This classification incorporates anatomical damage with patient's symptoms [14].

The grade I sprain is characterized by the stretch of the ATFL, minimal swelling, or tenderness, without functional loss or joint instability [14]. The grade II injury is a partial microscopic tear of the ATFL, with or without the involvement of the CFL; a moderate swelling, pain, and tenderness develop, and the range of motion reduces with an initial joint instability. In the grade III sprain, a complete ligamentous rupture is present; both the ATFL and CFL are involved, with or without a capsular tear and a PTFL tear; swelling and tenderness are intense and hemorrhage is usually present. A marked alteration of motion is evident, as well as the loss of function and the severe joint instability [14].

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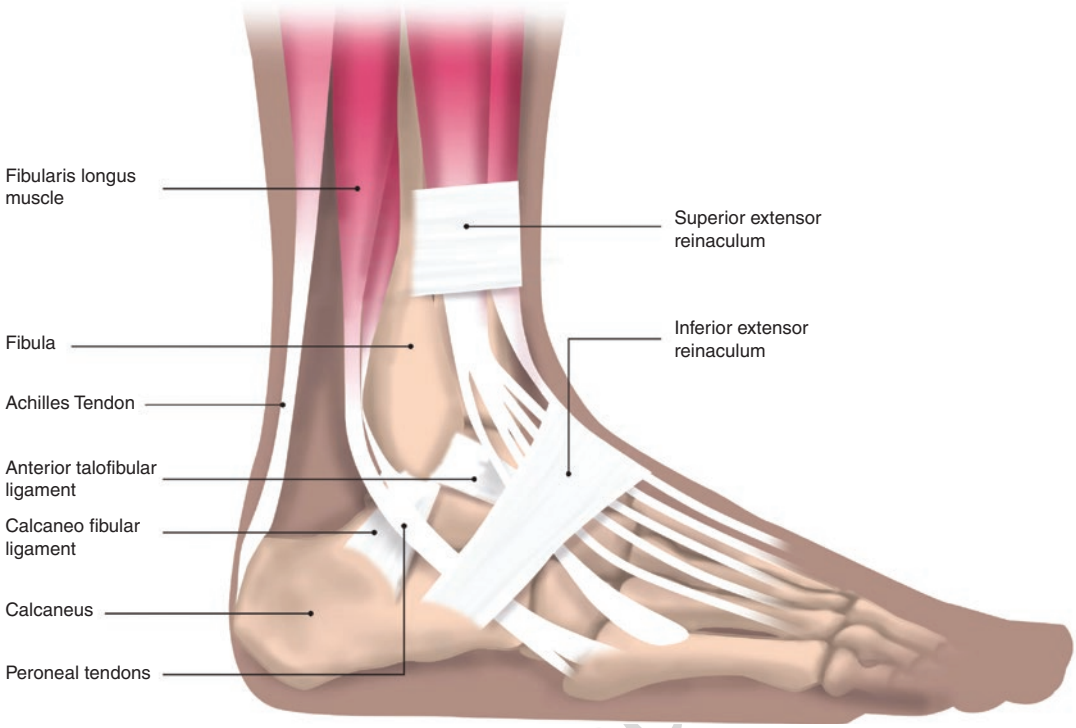

Fig. 5.1 Anatomy of the ankle

Chronic ankle instability persists in 5–20% of acute injuries, and it is defined as an instability lasting more than 6 months [1].

Ankle instability may be classified as caused by lateral, medial, and syndesmotic ligament injuries [15].

5.2 Diagnosis

The "on field" assessment is not accurate enough to diagnose the grade of injury; however it is important to provide initial first care to protect the athlete from further injury or stop him from continuing the match. In the outpatient setting, a careful history and physical examination is fundamental when evaluating a patient both with an acute ankle sprain and chronic ankle instability.

In the most acute stage, the patient usually describes "rolling over" of the ankle often due to an inversion movement of the ankle. When eliciting the history from a patient with chronic ankle instability, the main complaint includes intermittent "giving out of the ankle" with a history of at least two or three severe lateral ankle sprains.

The most important features of physical examination are tenderness, hematoma, pain, swelling, and difficulty in weight-bearing.

The diagnosis requires a thorough history of the triggering event and the recurrent traumatic episodes, as well as an accurate physical examination [16].

The Ottawa ankle rules have been developed to exclude fractures after an acute trauma; however, most athletes are examined using radiographs despite the fact that the incidence of ankle fractures is less than 15% [17].

It is fundamental to observe any foot and ankle malalignment, to palpate the ankle expecting pain and tenderness especially around the lateral gutter.

Two provocative tests are essential to assess stability of the lateral ankle ligaments, and they must be performed in comparison with the uninjured leg. If increased laxity is present, the tests

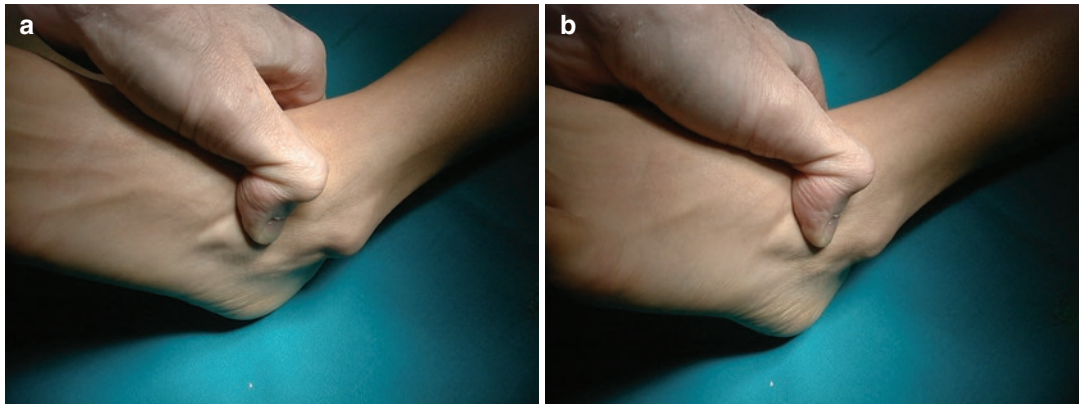

Fig. 5.2 (**a**, **b**) Positive Talar Tilt test

are considered positive. The anterior drawer test evaluates the integrity of ATFL, while the talar tilt test assesses the integrity of CFL [18–21] (Fig. 5.2a, b).

The gold standard in the diagnosis of acute lateral ligament injury is the delayed physical examination (4–5 days post-trauma). This timing provides a diagnostic modality of high quality to perform the tests with a better sensitivity and specificity compared with an earlier examination, though limited due to pain and swelling [19, 21]. A positive anterior drawer test in combination with pain on palpation at the ATFL and hematoma has a sensitivity of 98% and a specificity of 84% for having an ATFL injury [21].

Standard antero-posterior and lateral weight-bearing radiographs are important to evaluate joints' morphology, alignment, and the possible presence of arthritis [16]. The use of stress radiographs is controversial, because they are useful to assess the joint, but concerns remain on their reproducibility and on which values should be considered as normal [18, 22].

Ultrasound and magnetic resonance imaging (MRI) can be useful in diagnosing associated lesions like bone, chondral, or tendon injuries and they are routinely performed in professional athletes. MRI has a very high specificity but a relatively low sensitivity [18, 19].

Peroneal muscle strength and proprioception should be also assessed during physical examination.

5.3 Conservative vs. Surgical Treatment

As for the treatment of grade I and II ankle sprains, conservative management remains the gold standard; the treatment for grade III ankle sprains is instead less standardized.

The Cochrane review of Kerkhoffs et al. [23] regarding the comparison of surgical versus conservative treatment for acute lateral ankle ligament injuries fails to demonstrate a clearly superior treatment approach. The likelihood of operative complications, stiffness of the ankle joint and the higher costs associated with surgical treatment, would suggest functional treatment as the best available option for most patients [23]. A further motivation to not recommend surgery is that delayed surgery is equally effective as primary surgery [24].

Surgery seems to provide better outcomes only in objective ankle stability without functional differences [23].

The most recent guidelines [25] for the conservative treatment of ankle sprains suggest that 3–5 days of rest, ice, compression, and elevation with early weight-bearing, after which active exercise is commenced, lead to a fastest resumption of work and daily life activities compared with any other kind of treatment. For prevention, both tape and brace may be used and the choice of modality should always be based on patient preferences. Supervised exercises are advised

with the focus on proprioception, strength, coordination, and function [25].

Eighty percent of acute ankle sprains make a full recovery with conservative management, but 20% develop mechanical or functional instability resulting in chronic ankle instability [26].

Although for general population conservative treatment is preferred over surgical options, in professional players remains a lack of consensus. Athletes have greater load and demand on their ankle joints, and they could easily develop residual complaints [2].

For high-level sports teams, absence of key players due to injury may result in defeat and economic loss. Since increased objective instability is a predictor for future ankle sprains [27], an acute reconstruction should be taken into consideration in professional athletes.

The time of the season, athlete's expectations, and stage of his career are all features to take into account when considering an operative treatment of lateral ankle injuries in a competitive athlete.

Modern post-surgical treatment protocols report that the time to return to sport will be similar, with or without surgery.

Conservative treatment is usually preferred in case of acute lesions, while surgery is recommended for chronic instability [1, 23, 28] or in presence of osteochondral lesions [29].

Conservative treatment aims to restore mechanical joint stability and consists in neuromuscular reprogramming, foot evertor muscles reinforcement, and muscular pre-activation. If these procedures have been unsuccessful, a surgical stabilization is required [30].

Furthermore, the role of the CFL in the management of chronic ankle instability is an evolving concept [31]. It is the only ligament bridging both the talocrural and the subtalar joints [32], and it has been demonstrated that the CFL can be tensioned while approaching the ATFL, due to the presence of connecting fibers between these two ligaments [33].

Due to the stabilizing function of both the ankle and the subtalar joints, an injury of the CFL may cause increased subtalar laxity in addition to ankle instability [2].

For this reason, in order to restore the normal ankle and subtalar mechanics, it is fundamental to recreate the anatomy and the orientation of the lateral ankle complex, including the repair of the CFL ([2, 31]).

5.4 Surgical Techniques

It has been reported that since 1966 over 60 different procedures for treating ankle instability have been described [34], including the use of autologous or synthetic grafts [35] and the arthroscopic approach [16].

Numerous surgical procedures have been described for lateral ankle instability, which can be divided into three categories: nonanatomic reconstruction, anatomic reconstruction, and anatomic repair. The first surgical procedures described in literature were nonanatomical reconstruction techniques.

- *Nonanatomic reconstruction* was the first reported procedure for the treatment of lateral ankle instability and described by Elmslie in 1934 using fascia lata graft to reconstruct the lateral ankle ligaments [36]. In 1952, Watson-Jones used the peroneus brevis and re-routed the tendon in a posterior to anterior fashion through the fibula and securing it onto the talar neck [37]. In 1953, Evans reported a modified version of the Watson-Jones procedure by routing the peroneal brevis tendon obliquely through the distal fibula in an anterior-distal to posterior-proximal fashion [38]. In the Chrisman-Snook reconstruction, the peroneus brevis tendon is split and transferred through the fibula and into the calcaneus [39].
- *Anatomic reconstruction:* Colville described a reconstruction using a split peroneus brevis tendon to augment a repaired ATFL and CFL. The peroneus brevis is placed into the anatomic origins and insertions of the ligaments. The indications for this technique require poor tissue quality or revision surgery [40].
- *Anatomic repair* include the original Broström technique [8]. Broström first described a

mid-substance repair of the ATFL in 1966. In 30% of cases, the CFL repair was also performed. Subsequently, Gould modified the Broström procedure adding the mobilization and the reattachment of the lateral portion of the extensor retinaculum to the fibula in order to obtain an additional talocrural and a secondary subtalar joint stability [41]. Furthermore, in 1988 Karlsson proposed the reattachment of the ATFL to its anatomical peroneal insertion through drill holes [42].

Nonanatomic procedures show excellent results in the initial period, but in the long run they develop persistent instability, abnormal kinematics, stiffness, loss of movement, and weak eversion [39, 42–46]. Also the extensive incision required for the procedure heightens the risk of wound infection and sural nerve damage [47, 48]. However, these invasive procedures required long immobilization time and were responsible for joint stiffness and secondary muscle imbalance because of the sacrifice of the peroneal tendons [16, 49].

Anatomic procedures show fewer complications and less restriction in mobility compared to the nonanatomic ones as reported by Sammarco in his study [50].

In 1996, Hennrikus [51] compared the Chrisman-Snook with the Broström-Gould procedure. Both groups showed same good results but in the nonanatomic operation were found more complications. Wainright et al. [52] recently reported improved ankle joint kinematics in unstable ankles after modified Broström-Gould repair, with a significant decrease in anterior translation and internal rotation of the talus. Cadaveric studies have shown greater mechanical stability obtained with this anatomic technique as opposed to Watson-Jones and Chrisman-Snook reconstructions [53, 54].

The reported advantages of the modified Broström-Gould procedure mainly concern the preservation of ankle kinematics and the native ATFL fibers, fundamental for proprioceptive function as they ensure a stronger fixation and the absence of peroneal weakness observed in nonanatomic reconstruction.

These data allow Petrera et al. [55] to demonstrate that immediate, protected, full weightbearing after a modified Broström surgery with an accelerated rehabilitation could allow athletes an early return to sport. Another study [56] published in 2016 reports excellent results in athletes affected by chronic ankle instability treated with this type of repair also in long-term follow-up (10–15 years).

It may be summarized that the anatomic procedures consist in either a direct repair of the injured structures or an anatomic reconstruction with auto- or allografts [57].

The first option is usually preferred in presence of adequate ligamentous remnants, while reconstruction is suggested in cases of constitutional ligamentous laxity, obesity, failed prior stabilization, and poor or insufficient ligamentous remnants [58].

To date, the Broström-Gould reconstruction technique is still considered the “gold standard” for the treatment of chronic ankle instability [49, 59, 60].

Anatomic repair is largely supported in the literature [61–64].

After an initial skepticism due to the high complexity, more complications, a longer surgical time, and controversial results compared to the open techniques, the arthroscopic stabilization first introduced by Hawkins [65] and Ferkel [66] has been recently revalued [16, 67].

The reasons are less invasive, a faster return to sports activities, and the opportunity to treat other intra-articular associated problems simultaneously, such as osteochondral lesions or synovitis [16]. In fact, it has been proved that the poor accuracy of MRI in showing the intra-articular lesions is often associated with chronic instability [68].

Nevertheless, it has been highlighted how the arthroscopic fixation devices increase the risk of nerve or tendon entrapment [69], thus requiring the identification of an inter-nervous and inter-tendon safe zone before surgery [70].

With reference to the several techniques described, the treatment of CFL should not be undervalued. It is a primary stabilizer of the tibiotalar and subtalar joints [57], especially in

dorsiflexion [71]. CFL is four times stronger than ATFL; thus its reinforcement should be mandatory during the stabilization of chronic ankle instability where talar tilt test is constantly positive while anterior drawer test is frequently negative [71], though some authors do not agree [72, 73].

5.5 The Lateral Reefing Procedure

It is not unfrequently that patients undergoing surgery for lateral ankle instability have associated intra-articular pathology; therefore possible associated lesions are arthroscopically treated before proceeding with the stabilization if detected preoperatively by an MRI or in presence of intra-articular symptoms.

This procedure addresses chronic instabilities. It may be performed in local or peripheral anesthesia, with or without the use of a tourniquet.

The patient is positioned supine with the heel at the very bottom of the table and knee extended, a tourniquet placed on the proximal lower thigh, and a sandbag under the ipsilateral buttock to improve the access to both lateral and medial side of ankle.

The surgeon performs a longitudinal incision that passes over the distal fibula for 3 cm towards the talar neck. The capsule and the lateral ligaments are exposed by retracting the peroneal tendons. During the tissue dissection, it is important to avoid damages to the lateral cutaneous branch of the superficial peroneal nerve and the branches of the short saphenous vein.

The ATFL and CFL are then plicated and sutured to the periosteum of the inner distal fibula (Figs. 5.3, 5.4, 5.5, 5.6, 5.7, 5.8).

Two or three nonabsorbable sutures are passed from inside to outside the capsulo-ligamentous complex. Ankle stability is intraoperatively checked, and further sutures are eventually added. Before tying the suture knots, the ankle is held by the assistant in dorsiflexion and eversion.

The Gould modification is added in specific cases, if the previous sutures do not restore full

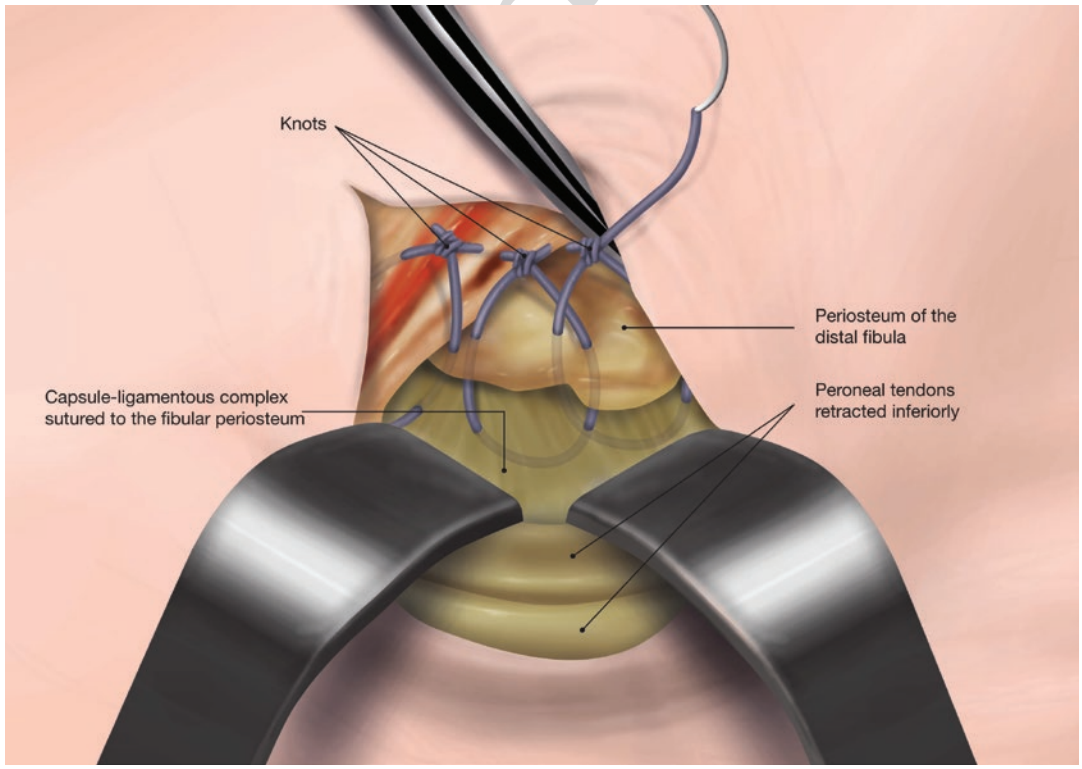

Fig. 5.3 Surgical technique: reefing of the lateral capsulo-ligamentous complex to the peroneal periosteum with multiple stitches

Fig. 5.4 Surgical access: retinaculum is incised and peroneal tendons are exposed

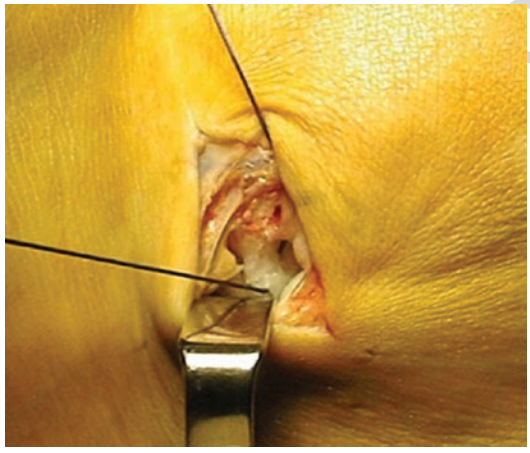

Fig. 5.5 Peroneal tendons are retracted posteriorly, below the fibula. The lateral capsulo-ligamentous complex is exposed and the reefing is started grasping the lax tissue

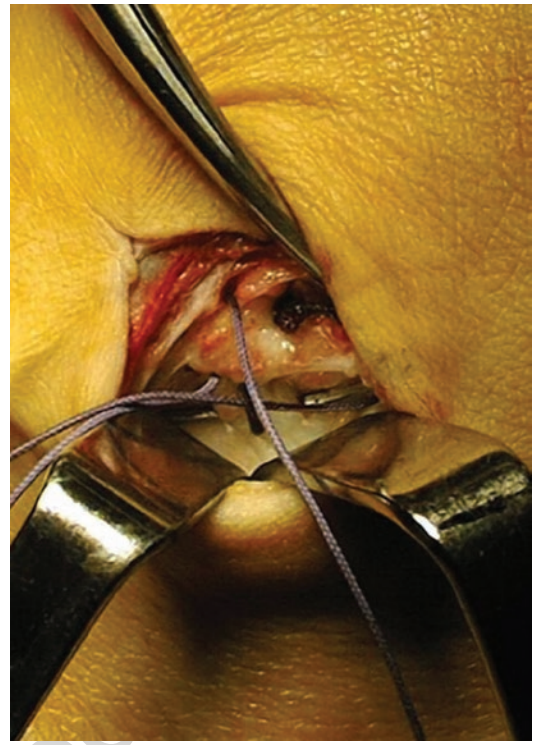

Fig. 5.6 Second suture: the needle is passed through the calcaneofibular ligament

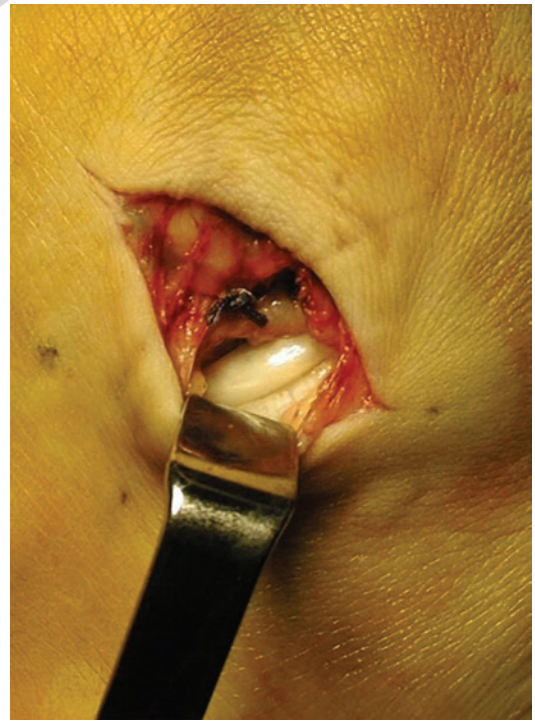

Fig. 5.7 After suturing, the peroneal tendons are repositioned anatomically

ankle stability and consist in inserting sutures from the fibular periosteum into the inferior extensor retinaculum.

If a gentle anterior drawer and talar tilt tests confirm the stability of the ankle, after an irrigation with 0.9% saline, the peroneal retinaculum and the skin incision can be sutured. Steri strips

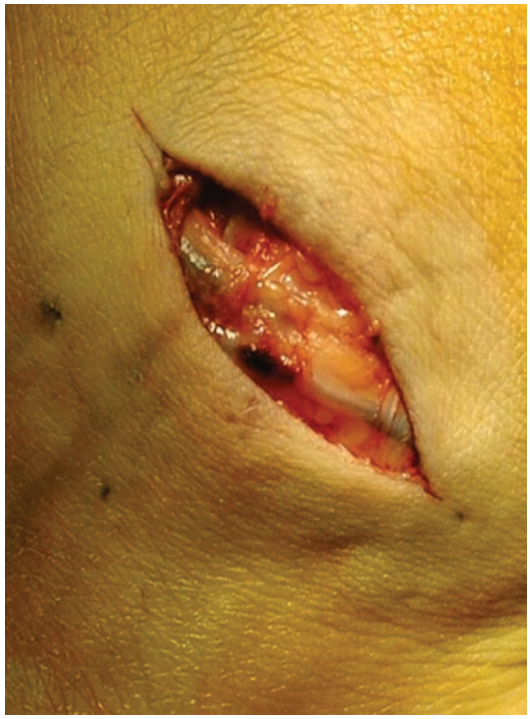

Fig. 5.8 Suture of the peroneal retinaculum

are applied and a brace is added to keep the ankle in slight dorsiflexion and eversion.

5.6 Postoperative Management and Return to Sport

The management after a surgical reconstruction should always be a balanced approach with an initial protection of the repair and an early rehabilitation in order to prevent the complications of a longer immobilization. A recent case series, cited above, showed good results and no increased rates of complication with immediate weight-bearing and early range of motion exercises after this surgery technique [55], but it is still very risky to rehabilitate a patient too quickly. In a study about foot injuries conducted in professional rugby players [74], it was found that rehabilitating a relapse can be three and a half times longer than rehabilitating the original injury. When performing an anatomic repair, as in the modified Broström procedure, we need to remember that we are trying to re-establish the bone-ligament interface and an excessive load could damage the repairing insertion site, although there is much basic scientific evidence that an insufficient load could lead to a catabolic environment [75, 76].

For this reason, an approach based on a short period of immobilization followed by gradually increased movement and load bearing seems to be reasonable. In their paper, the members of the ESSKA-AFAS Ankle Instability Group recommend the patient fully weight-bearing with the boot on after 10–14 days of immobilization [77]. At this time, mobilization of the ankle under safe conditions with a limited ROM is also possible; however walking without protection until week 6 should not be admitted.

The goals of the next phase (after week 6) include the increase of strength, range of motion, and the possibility to achieve daily activities pain-free. Ankle and foot strengthening should include exercises to address tibialis anterior, tibialis posterior, gastrocnemius, and foot intrinsics but the emphasis must be put on the peroneal muscles, progressing from isometric to isotonic to resistive exercises. Ankle strengthening can also progress from non-weight-bearing to weight-bearing positions and in this phase of recovery proprioception and balance exercises should also be initiated.

The late rehabilitation phase typically occurs between weeks 8 and 12 post-surgery, and to enter this phase, patient should demonstrate symmetrical gait patterns, ankle strength at least 90% of the contralateral side, and the ability to perform the functional tests described.

Running should begin at slow speed and progress to higher speed and longer distances. Agility drills are also included in this period. During the late rehabilitation stage, functional tests can be particularly useful in identifying when to advocate the return to play phase. These tests include the single leg hop test, the triple hop test, the vertical jump, the drop jump, the 6-meter timed hop test, the Star Excursion Balance Test, and the Foot and Ankle Ability Measure (FAAM) questionnaire [77].

The return to sport phase typically falls between 12 weeks and 4 months following

surgery. The use of taping or bracing is recommended in the early phases of return to full sports activities in order to avoid re-injury. It is important to underline that these recommendations are a general guide for management, but it is always necessary to consider individual response to treatment [31].

5.7 Conclusions

Minimally invasive surgery for chronic lateral ankle instability is rapidly evolving.

This technique is simple, effective, and addresses calcaneofibular ligament insufficiency with the additional advantage of a shorter surgical time than other open or arthroscopic approaches.

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Syndesmosis Injuries

6

Pieter D'Hooghe

6.1 Introduction

Syndesmotic injuries, or high ankle sprains, comprise 10% of all ankle sprains [1]. These injuries are frequently sustained during athletic competition, particularly soccer [1, 2]. However, as imaging studies suggest that up to 20% of acute ankle sprains involve the syndesmosis, the prevalence of syndesmotic injuries may be underestimated [3, 4]. Syndesmotic injuries often require twice as long to return to sport as compared to isolated lateral ligament sprains and can lead to prolonged pain and disability [5–8]. Further, the most common cause of chronic ankle dysfunction 6 months from an ankle trauma is related to syndesmotic injuries [7]. Recurrent and undiagnosed ankle instability is known to ensue and eventually lead to premature ankle arthritis [9]. Therefore, a timely diagnosis of unstable syndesmotic injuries is essential. A rapid pivoting and forced ankle dorsiflexion of the ankle with a forceful external rotation and pronation of the foot is the most common mechanism of a high ankle sprain [10]. Planovalgus foot alignment, high competitive sports level, and male gender are potential risk factors [9, 11, 12]. As the talus rotates in the mortise, the fibula rotates externally and moves posteriorly and laterally. This mechanism then separates the distal tibia and fibula and sequentially tears the AITFL, deep deltoid ligament (or causes a malleolar fracture), the inferior oblique ligament (IOL), and finally the posterior inferior talo-fibular ligament (PITFL) [10, 13]. When there is a combined syndesmotic injury with a deltoid ligament disruption, talar instability occurs [14].

Less commonly, the injury may occur in forced dorsiflexion without rotation since the anterior part of the talus is wider than the posterior part. The magnitude and duration of force application appear to be predictive factors of lesion severity [9]. Syndesmotic injuries are classified in three grades, ranging from a partially torn AITFL to a complete disruption of all ligaments with mortise widening [15].

Stress radiographs and magnetic resonance imaging (MRI) can be helpful in the diagnosis of these injuries, but currently there is no best evidence-based test available that can identify syndesmotic instability (especially in grade II lesions). This is particularly relevant in the athletic population, where appropriate management is crucial for the player to return to the team [3]. There is a consensus to use arthroscopy in the evaluation of syndesmotic stability in doubtful cases, but there is no validated surgical protocol available (except expert opinion) to identify syndesmotic stability under direct visualization with arthroscopy [16].

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6.2 Anatomy

A syndesmosis is defined as a fibrous joint in which two adjacent bones are linked by a strong membrane or ligaments [17, 18]. The distal tibiofibular joint is a syndesmotic joint between the tibia and fibula, linked by four ligaments: the anterior inferior tibiofibular ligament (AITFL), the interosseous ligament (IOL), the posterior inferior tibiofibular ligament (PITFL), and the inferior transverse ligament (ITL). The distal tibiofibular joint employs both its bony and ligamentous structure for stability (Fig. 6.1).

The architecture of the bony components of the syndesmosis provides significant stability to this joint. The fibula sits in a groove created by bifurcation of the lateral ridge of the tibia into the anterior and posterior margins of the tibia, approximately 6–8 cm above the level of the talocrural joint [17, 19]. The anterior margin ends in the anterolateral aspect of the tibial plafond called the anterior tubercle, or Chaput's tubercle.

The posterior margin ends in the posterolateral aspect of the tibial plafond called the posterior tubercle, or Volkmann's tubercle. The apex of this fibular notch is the incisura tibialis, which has a depth that varies from concave (60–75%) to shallow (25–40%) [17, 20, 21]. Its depth varies from 1.0 to 7.5 mm [17, 22, 23] and is a little less in women than in men [17, 24].

A shallow notch may predispose to recurrent ankle sprains or syndesmotic injury with fracture dislocation [18]. The bony architecture of the fibula mirrors that of the fibular notch.

The medial aspect of the fibula forms a convex structure that complements that of the tibia, with an anterior and posterior margin, as well as a ridge that bifurcates that margins and aligns itself with the incisura tibialis.

The AITFL originates from the anterior tibial tubercle and runs distally and laterally in an oblique fashion to insert onto the anteromedial distal fibula (Figs. 6.1 and 6.2). This ligament has a width of approximately 18 mm, length between 20 and 30 mm, and a thickness of 2–4 mm. It is the most commonly sprained ligament in syndesmotic injuries and is always disrupted with joint space widening or frank diastasis [17, 18].

It is often multifascicular, and its most inferior fascicle has been described as a discrete structure called the accessory AITF ligament.

The fibers can be seen during ankle arthroscopy and have been reported to be a source of impingement [17, 25]. The PITFL originates on the posterior aspect of the fibula and runs horizontally to Volkmann's tubercle. This ligament has an approximate width of 18 mm and a thickness of 6 mm and is the strongest component of the syndesmosis.

Because of its extensive breadth of attachment coupled with elasticity, the PITFL is able to withstand greater forces without failure than the AITFL and reaches maximal tension during dorsiflexion [17, 19, 26].

The inferior transverse ligament is deep and inferior to the PITFL, extending over to the posterior aspect of the medial malleolus. The inferior transverse ligament is often difficult to distinguish from the PITFL as it runs just distally in the same plane.

It forms the most distal aspect of the articulation (Fig. 6.2). A portion of this ligament lies below the posterior tibial margin preventing posterior translation of the talus and deepening the ankle mortise to increase joint stability by functioning as a labrum.

The interosseous ligament spans the space between the lateral tibia and medial fibula and is confluent with the proximal interosseous membrane. It is the main restraint to proximal migration of the talus between the tibia and the fibula [9, 17] (Fig. 6.2).

6.3 Epidemiology

Syndesmosis or "high ankle" sprains are reported to occur in 1–18% of patients with an ankle sprain [27, 28]. However, this is probably an underestimate, as 20% of athletes with an acute ankle sprain have evidence of syndesmotic injury on MRI [28].

This variation can be explained by the fact that some sports have extrinsic risk factors asso-

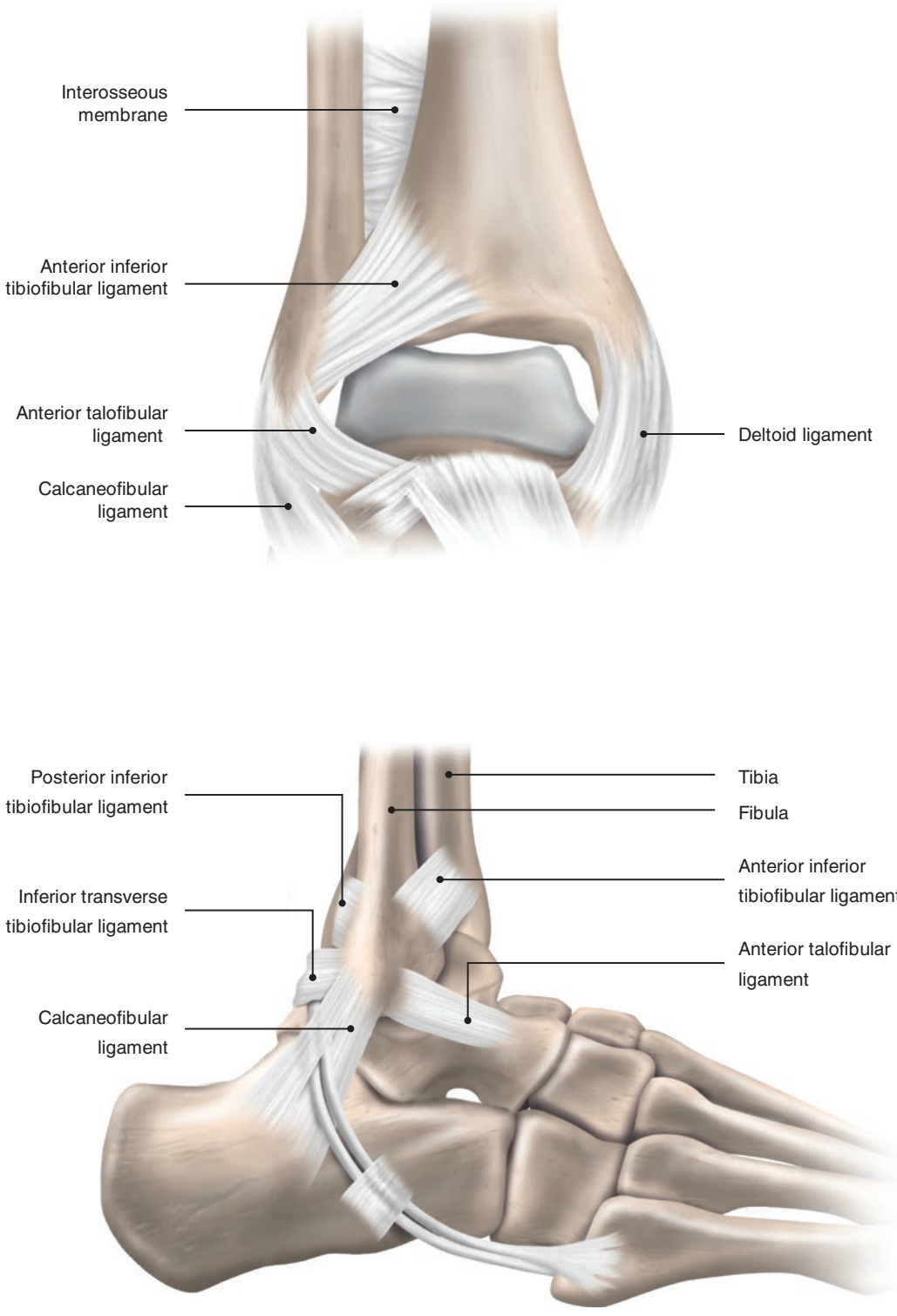

Fig. 6.1 Antero-posterior and lateral view of the ankle ligamentous complex

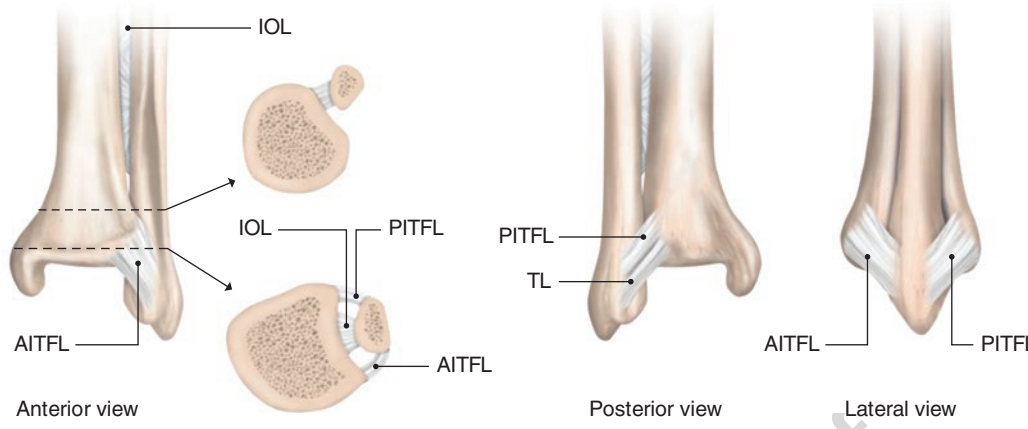

Fig. 6.2 Detailed antero-posterior and lateral view of the ankle syndesmosis ligaments

ciated with syndesmosis injury. Skiers and ice hockey players wear boots causing rigid immobilization of the ankle leading to high-torque external rotation of the foot [28–31] and American football is often played on artificial turf instead of natural surfaces [28, 32–35]. Another plausible explanation is that an isolated syndesmotic injury can be frequently misdiagnosed as an ankle sprain [28].

A recent epidemiological overview on isolated syndesmosis injuries in elite football indicated a significant increase in the incidence of these injuries with an average return to play time following injury that exceeded 5 weeks. Also, no change in injury burden was found over 15 consecutive football seasons. This was primarily linked to the more aggressive playing style during matchplay [28].

Male gender, elite performance, and a planovalgus alignment are risk factors for syndesmosis injury in athletes [36, 37]. Syndesmosis injuries can occur with an ankle sprain only, with fractures or as a combination of both. In fact, 23% of ankle fractures are reported to have combined syndesmosis injuries [36, 37].

The associated fractures are commonly either of the fibula or of the posterior and medial malleoli. Syndesmosis injury should be increasingly suspected if there is an associated fracture of the proximal fibula (Maisonneuve fracture, Fig. 6.3) and they are associated with prolonged pain, disability, and an unpredictable time away from sports [27, 37].

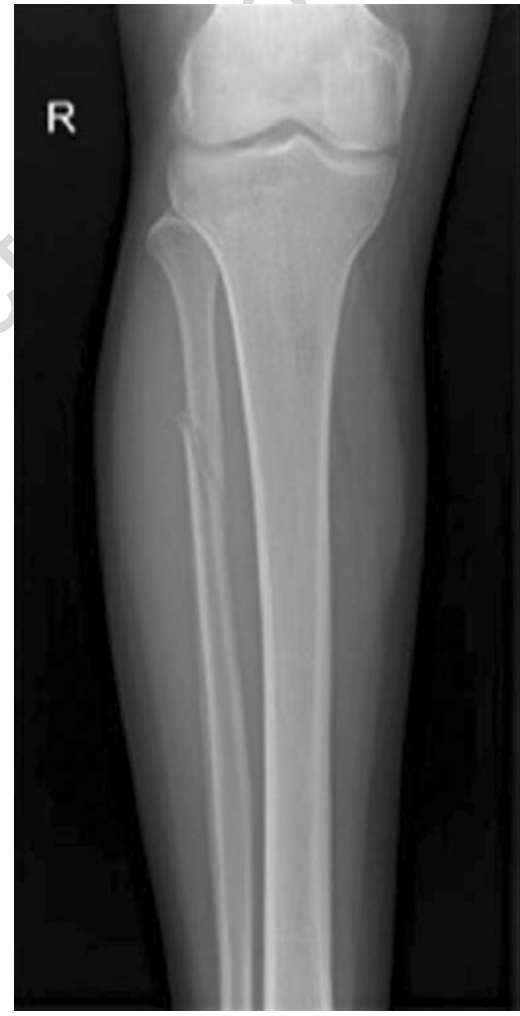

Fig. 6.3 Antero-posterior X-ray image of a Maisonneuve fracture

6.4 Mechanism of Injury

The general mechanism of injury for syndesmosis ankle sprains is a forceful external rotation of the foot and ankle with the ankle in dorsiflexion and the foot pronated [27, 38]. While the talus rotates in the mortise, the fibula rotates externally and moves posteriorly and laterally, separating the distal tibia and fibula.

This will sequentially cause tears of the anterior inferior tibiofibular ligament (AITFL), the deep deltoid ligament, or might alternatively cause a malleolar fracture. This shall be in turn followed by a tear of the interosseous ligament (IOL) and finally the posterior inferior tibiofibular ligament (PITFL) [27, 38, 39].

Severity of syndesmosis injury varies, ranging from a partially torn AITFL to a complete disruption of all ligaments with mortise widening. It has been shown that combined deltoid and syndesmosis injury will critically compromise talar stability [14, 27]. The magnitude of force and its duration will determine the extension of syndesmosis and interosseous injury proximally [9] and this may eventually lead to a Maisonneuve fracture (Fig. 6.3). Another injury mechanism for syndesmosis ankle sprains is hyper-dorsiflexion.

Forced dorsiflexion of the ankle causes the wider anterior talus to act as a wedge that can cause injury to the syndesmosis ligaments [27].

6.5 Clinical Evaluation

Athletes frequently present with an inability to bear weight, anterolateral pain between the distal tibia and fibula, medial ankle pain, ankle effusion, and pain during gait push off [40, 41]. However, anterolateral pain is not specific, as up to 40% of patients with an ATFL tear describe pain over the AITFL. Clinically it has been suggested that the more proximal the patient's pain, the more significant the injury [40, 41].

Several clinical tests can be used in the evaluation of a syndesmotic injury. The external rotation test and the squeeze test are the most commonly described tests, but the Cotton test, the fibular translation test, the heel thump test, the dorsiflexion compression test, and the cross-legged test can also be used [15, 27]. The combination of tenderness on palpation over the ATFL, a positive fibular translation test, and positive Cotton test is considered highly clinically suspicious [16, 27].

Although the squeeze test has been shown to be highly sensitive, there is no one "gold standard" for the clinical diagnosis of syndesmotic instability [27, 42]. In case of clinical suspicion, advanced imaging, such as MRI, is warranted.

It has been shown that there is a significant correlation between how far this tenderness radiates proximally in the leg and the severity of the injury and, consequently, the time to return to sports [27, 37].

Patients with high ankle sprains may complain of the inability to bear weight, swelling, pain during the push-off phase of gait, and pain anteriorly between distal tibia and fibula, as well as posteromedially at the level of the ankle joint [15, 27]. Ankle ROM will often be limited, with pain felt more at terminal dorsiflexion [27, 42]. Numerous special tests are used to detect syndesmosis injuries. However, a recent systematic review on eight different tests reported a low diagnostic accuracy of these tests [43]. The squeeze test was the only test with a clinical significance [43].

Diagnosing an athlete with a syndesmotic injury can however still be difficult.

The pain is often diffuse and difficult to differentiate from a lateral ankle sprain. Additionally, as previously noted, there can be overlap in injury patterns. This can further cloud the diagnosis and potentially lead to missed syndesmotic injuries.

However, a thorough history might uncover a mechanism that would increase the treating physician's suspicion. A thorough physical examination includes visual inspection for swelling, palpation for tenderness, and evaluation of the proximal extent of the tenderness.

The latter physical examination finding, known as "syndesmosis tenderness length" (the most proximal site of tenderness measured from the distal tip of the fibula), has been shown to correlate with the time to return to sports [40, 44].

The typical location of tenderness in a syndesmotic injury is at the anterolateral and/or posteromedial joint line.

All current clinical syndesmosis tests have been shown to be difficult to interpret with a low

predictive value in the presence of a painful or swollen ankle [45]. Although the squeeze test has most clinical significance in recent literature [15, 27, 43], the external rotation test has been shown to be most sensitive with the lowest false positive rate [40, 46]. This is performed with the ankle in neutral or slight dorsiflexion and the heel in neutral or varus position, with subsequent external rotation of the foot relative to the tibia to the point of resistance and pain.

Additionally, a stress radiograph can be obtained to evaluate for medial clear space (MCS) or tibiotalar widening [40, 47].

6.6 Imaging

Plain radiographs should still always be obtained when there is concern for syndesmotic injury. The tibiofibular clear space, defined as the distance between the medial border of the fibula and the lateral border of the posterior tibia, is one of the most reliable indicators of syndesmotic disruption [41]. This distance is measured at 1 cm proximal to the tibial plafond and should not exceed 6 mm in both the AP and mortise views [41].

In the case of a suspected syndesmotic injury, radiographs must be carefully scrutinized. Signs of syndesmotic injury include avulsion fractures of the anterior tubercle of the tibia (Tillaux-Chaput fragment, Fig. 6.4a–d), anterior fibula (Wagstaffe le Fort fragment), and posterior malleolus (Volkmann fragment).

Radiographs should be evaluated for the tibiofibular clear space (TFCS) (normal = mean 4.4 ± 0.8 mm on antero-posterior view and 3.9 ± 0.9 mm on mortise view, respectively), the tibiofibular overlap (normal = mean 8.8 ± 2.4 mm on antero-posterior view and 4.6 ± 2.1 mm on mortise view, respectively), and for any increased MCS (normal <5 mm) [48]. However, it has been shown that tibiofibular overlap and TFCS do not correlate with syndesmotic injury seen on magnetic resonance imaging (MRI) [49]. Additionally, MCS

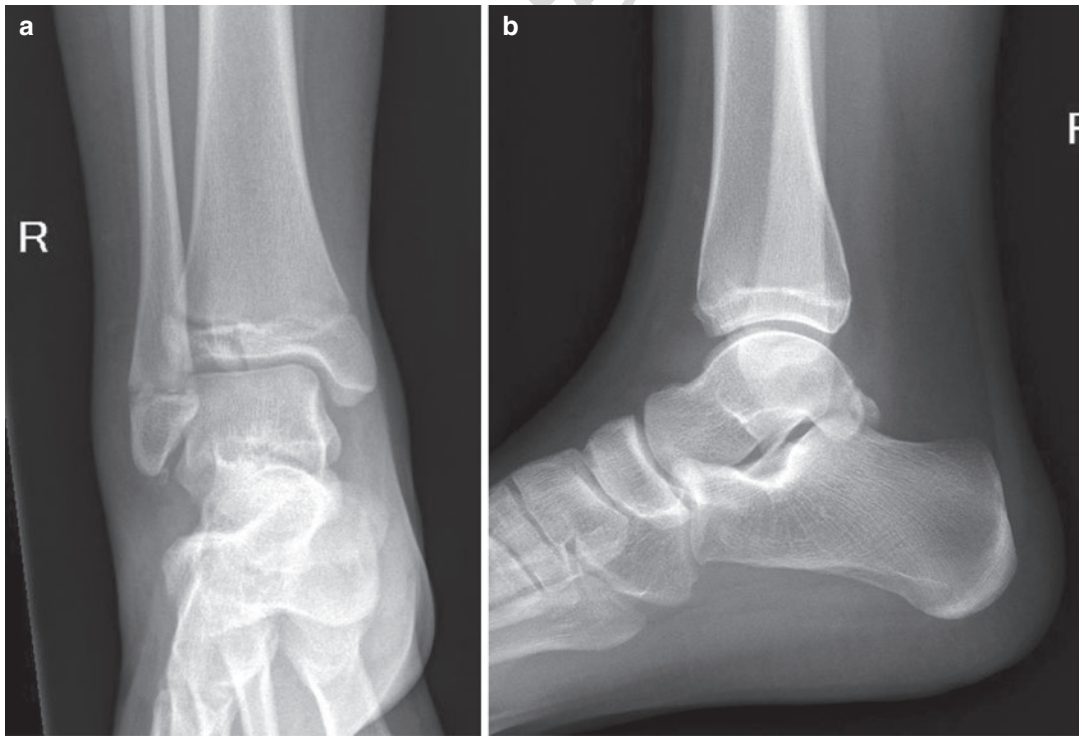

Fig. 6.4 (**a**–**d**) Avulsion fracture of the antero-lateral tubercle (**a**, **b**) of the tibia (Tillaux-Chaput) and after mini-open fixation fracture treatment (**c**, **d**)

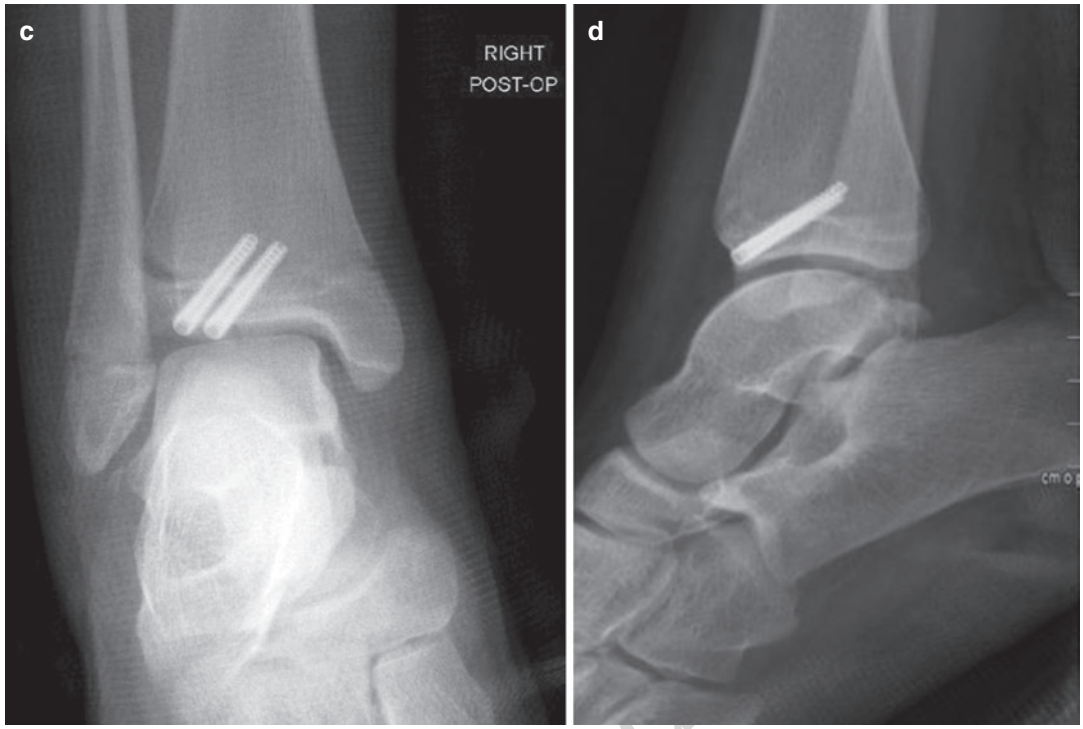

Fig. 6.4 (continued)

measurements have been shown to have poor accuracy and precision even among experienced providers. In a recent cadaver study, three specimens were evaluated with a known amount of displacement (6, 4, and 1.7 mm). Measurement errors ranged from 16% at 5° of internal rotation to 36% at 15° of external rotation for the specimen with 6 mm of known MCS widening but were even greater ranging from 3% at neutral to 100% at 5° external rotation for the intact specimen with 1.7 mm of MCS [40]. Although the sensitivity and specificity of detecting a syndesmotic injury on MRI has been shown to be up to 100%, determining the severity of that injury and the need for surgery is not straightforward and often only when frank diastasis is seen on radiography is the final determination for operative intervention made [41, 50–52]

Stress radiographs are no longer routinely recommended in the routine evaluation of syndesmotic instability since biomechanical studies have not shown significant advantage over plain radiographs [53, 54].

If an injury could potentially be managed nonoperatively, then stress radiographs can however be helpful in assessing the integrity of the syndesmosis and of the deltoid ligament. Still, there is no standardized technique or amount of force applied and the quality of the test can be significantly limited by the patient's pain [40, 41]. One recent study found that gravity stress radiographs (with the foot suspended via a bump under the calf allowing gravity to pull the foot in external rotation) resulted in equivalent MCS widening to manual stress radiographs [41]. Conversely, if there is an operative fracture, then stress radiographs can be postponed until surgery.

Computed tomography (CT) scanning can be helpful in identifying minor diastasis and small avulsion fractures [55]. Although its value still needs further evaluation, promising new diagnostic types of bilateral standing CT scan stress view are useful [56]. Magnetic resonance imaging (MRI) can identify most ligamentous syndesmotic injuries and combined injuries [53]. MRI shows a sensitivity of 100% and a specificity of 93% for AITFL injuries (positive likelihood ratio of 14, Fig. 6.5) and a sensitivity and specificity of 100% for PITFL injuries (infinite positive likelihood

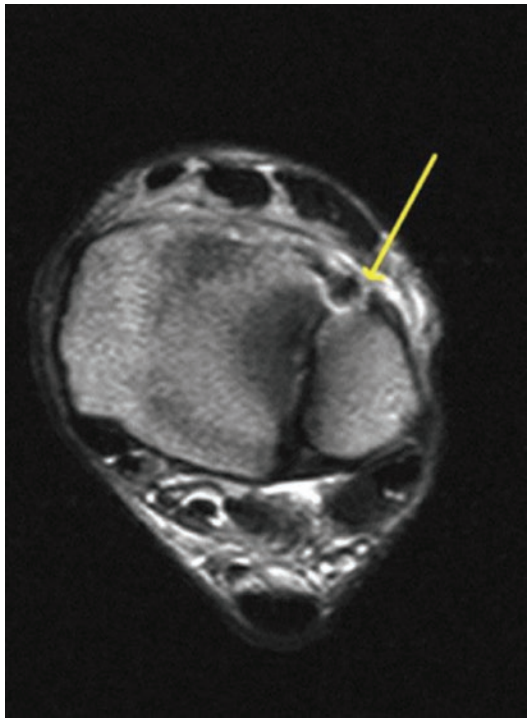

Fig. 6.5 Axial MRI image of an AITFL rupture in an elite football player

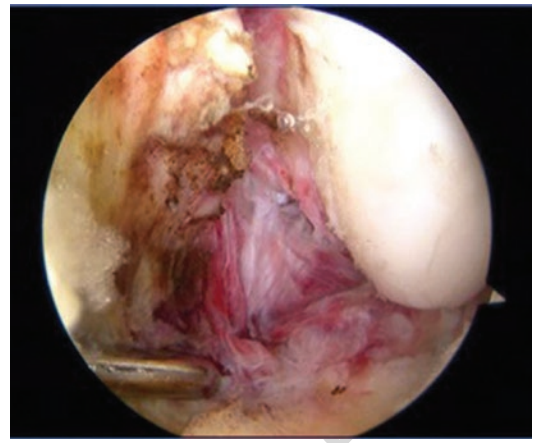

Fig. 6.6 Arthroscopic view of a grade III syndesmosis injury

ratio) [57] and has a high degree of interobserver reliability [49]. Ultrasonography is a fast and inexpensive tool to evaluate distal tibiofibular stability and does not expose the athlete to radiation. Further, it enables a dynamic assessment of the ligamentous injury, which is useful in cases of subtle instability. Patients with an acute AITFL rupture (confirmed on MRI) show a 100% sensitivity and specificity on dynamic ultrasound evaluation [58]. The disadvantages are that ultrasonography cannot detect associated injuries and is proven to be investigator dependent [41, 53].

6.7 Classification and Treatment

6.7.1 Classification of Syndesmotic Injuries

Syndesmotic injuries are divided into three grades. Grade I represents an AITFL sprain without instability. Grade II represents an AITFL tear and a partial IOL tear with mild instability. Grade III represents a complete rupture of all three syndesmotic ligaments with evident instability [15, 54].

The severity of the syndesmotic instability guides the choice of treatment. Grade I injuries are treated nonsurgically [59] while the treatment of grade II injuries depends on the presented syndesmotic (in)stability testing [16]. Stable syndesmotic injuries (type I and IIa) should be treated conservatively, whereas unstable injuries (type IIb and III) warrant surgical fixation. A recent study found that a positive squeeze test and combined injury to the ATFL and deep deltoid ligament are key factors in differentiating stable (type IIa) from unstable grade II injuries (type IIb).

Nowadays, there is a consensus to perform an examination under anesthesia and arthroscopic evaluation of the syndesmosis in case of a grade II injury with clinical and/or radiological suspicion of dynamic instability (type IIb) [16, 60, 61]. In case of 2 mm or more dynamic distal tibiofibular diastasis, arthroscopic-assisted surgical fixation is warranted [59] (Fig. 6.6).

Grade III injuries often present with associated injuries and are inherently unstable. Surgical fixation by means of screws or suture buttons can be used to reduce the mortise and stabilize the syndesmosis [16, 62]. The Hook or Cotton test is regarded as reliable intraoperative stress tests to evaluate syndesmotic (in)stability [63] (Fig. 6.7b).

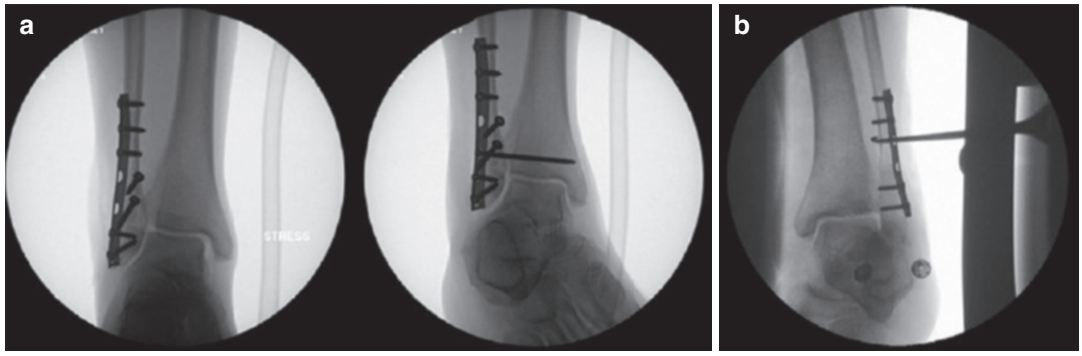

Fig. 6.7 (**a**) Intraoperative fluoroscopy of ankle fixation. Left: Stress radiograph following fixation of a Weber B fibula fracture with medial clear space widening [40]. Right: Radiograph following syndesmotic screw fixation. (**b**) Hook test performed in which the fibula is pulled lateral to assess for medial clear space widening [40]

Cadaveric studies have shown that the syndesmosis becomes unstable (opens more than 5 mm in tibiofibular clear space) when a force above 87–100 N is applied [63]. Arthroscopy is considered 'the golden standard' in the diagnostic assessment of syndesmotic (in)stability [64] and in case of doubt, fixation is advised because of the problems caused by chronic syndesmotic instability [63].

6.7.2 Management of Syndesmotic Injuries

6.7.2.1 Purely Ligamentous Injuries

In the case of sprains without diastasis, nonoperative management has been shown to result in good functional outcomes [65]. However, there is currently no consensus on the nonoperative regimen, with treatments ranging from taping to fracture boots to non-weight-bearing cast immobilization. Other interventions such as injections, physical therapy, ultrasonography, and nonsteroidal anti-inflammatory drugs are discussed throughout the literature without consensus. Reported lengths of immobilization vary from 1 to 6 weeks [46, 66].

Athletes should be informed that return to full sport takes longer compared to lateral ankle sprains.

The syndesmosis tenderness length can be used to estimate the time loss from sports using the equation [67]: Days lost from competition = 5 ± (0.93 × [tenderness length in centimeters]) ± 3.72 days.

Rehabilitation is implemented in three phases. Phase I is the acute phase. Goals include joint protection, minimization of inflammation, and pain control. Phase II is the subacute phase in which restoration of mobility, strength, and gait is emphasized. Finally, in phase III, emphasis is placed on strengthening, neuromuscular control, and sports-specific tasks [68].

A recent cohort-controlled study by Samra et al. suggested that ten rugby players with MRI-confirmed syndesmosis injury (involvement of the AITFL, IOL, and PITFL) treated without surgery who received a single autologous PRP injection into the AITFL had significantly shorter time to return to play than a historical cohort (20.7 days less for the intervention group vs. historical control). Following return, these patients had higher agility, increased vertical jump, and lower level of fear avoidance [69]. However, although they reported similar baseline characteristics between groups, the intervention was not blinded and there was no placebo control, both of which could have resulted in bias.

In contrast, all injuries with frank diastasis require syndesmotic fixation [70]. Taylor et al. reported on six intercollegiate athlete patients with grade III syndesmosis injuries treated with a 4.5-mm stainless steel cortical screw and reported good to excellent clinical outcomes in all patients with a mean return to sports at 40.7 days [71]. In their series, all

hardware was removed at an average of 74 days (range 52–97) [40].

6.7.2.2 Fractures with Syndesmotic Instability

Carr et al. recently performed a large database analysis of ankle fracture and syndesmotic fixation between 2007 and 2011 and found no significant increase in procedures for all ankle fracture types (lateral malleolus, bimalleolar, and trimalleolar) during that time [72]. However, the number of procedures to treat isolated syndesmotic injuries increased by 18% during that time period. In addition, the rate of syndesmotic fixation that accompanied fixation of ankle fractures significantly increased with a nearly twofold increase among bimalleolar fractures. The authors also reported that the rate of implant removal after syndesmotic fixation significantly decreased. This suggests an overall increase in recognition and operative treatment of isolated syndesmotic injuries and those associated with ankle fractures. Although factors associated with higher energy ankle fractures (e.g., bimalleolar involvement or the need for initial external fixation) are associated with delayed union, the need for syndesmotic screw fixation has not been shown to be associated with delayed union of ankle fractures that undergo fixation.

Nevertheless, although bony union can be followed via routine radiographs, the healing of the syndesmosis is significantly slower, requiring prolonged periods of non-weight-bearing up to 12 weeks [73]. Following fixation of medial and/or lateral malleolus fractures, an intraoperative stress radiograph can assess the integrity of the syndesmosis and guide the decision of whether or not syndesmotic fixation is of benefit (Fig. 6.7a).

Special consideration should be given to cases of bimalleolar ankle fractures in which there is an anterior colliculus avulsion of the medial malleolus. Tornetta reported on 27 patients with bimalleolar fractures who underwent external rotation stress radiographs intraoperatively after medial malleolar fixation and found that 7 (26%) had MCS widening even after medial fixation. He explained that this represents an injury to the deltoid ligament in which the stronger deep component has been ruptured and the weaker superficial component, which attaches to the anterior colliculus, remains intact. If this occurs in conjunction with a syndesmotic injury, it has the potential to present as late syndesmotic widening and significant instability [74].

6.7.2.3 Syndesmotic Fixation

Syndesmotic Screws

Syndesmotic screws have long been considered the gold standard for fixation of syndesmotic injuries (Fig. 6.7a). Most authors prefer 3.5 or 4.5 cortical screws which have equivalent biomechanical characteristics [75].

While some cadaveric studies have shown increased resistance to an applied load, specifically in shear stress, with a larger diameter screw [55] this has not been reproduced in clinical studies [66, 75]. In Europe, most surgeons utilize a single 3.5-mm tricortical screw, 2.1–4 cm above the joint line for stabilization of Weber B or C fractures [46]. However, a cadaveric study suggested that two screws provide a superior biomechanical construct compared to one [76].

Location of screw placement is often debated. McBryde et al. reported less syndesmotic widening when the screw was placed at 2 cm above the joint compared to 3 cm [77]. However, other studies have reported that screw placement at 2, 3, or 5 cm above the joint line shows no difference in functional outcome [77].

Tricortical screws (3.5 mm) were compared to quadricortical lag screws (both 3.5 and 4.5 mm) in terms of compression force in a 2012 cadaveric study. The lag screws maintained a significantly greater compression force after forceps removal compared to the tricortical screw.

Additionally, after each 100 cycles of loading, the lag screws significantly exceeded the amount of compression force maintained by the tricortical screw. No differences were seen between the 3.5- and 4.5-mm lag screws [78].

Ultimately, although cadaveric studies have suggested that four cortices provide more rigid fixation, screws with purchase in three cortices have been shown to more closely replicate tibio-

talar biomechanics [66] (Fig. 6.7a). Additionally, tricortical screws have decreased risk of screw breakage albeit at the cost of an increased rate of screw loosening [57, 75, 79]. There is no current evidence to suggest a clinically appreciable difference between these two methods of screw fixation [76].

In terms of screw removal, there has been a longstanding debate in the literature. Although some recommend removal of quadricortical screws to prevent screw breakage [79, 80], there is no consensus on when this should be performed and there have been reports of diastasis at screw removal [46].

Additionally, studies have suggested similar or better outcomes when the screw is retained [81] and therefore, there is growing consensus that screw removal should be reserved for screws that are symptomatic (i.e., painful prominence) [66, 82–84].

A recent systematic review by Dingemans et al. concluded that although there is insufficient evidence overall to draw definitive conclusions regarding routine removal, the lack of evidence to justify removal along with the additional cost and increased risk to the patient would suggest that routine removal should be avoided [85].

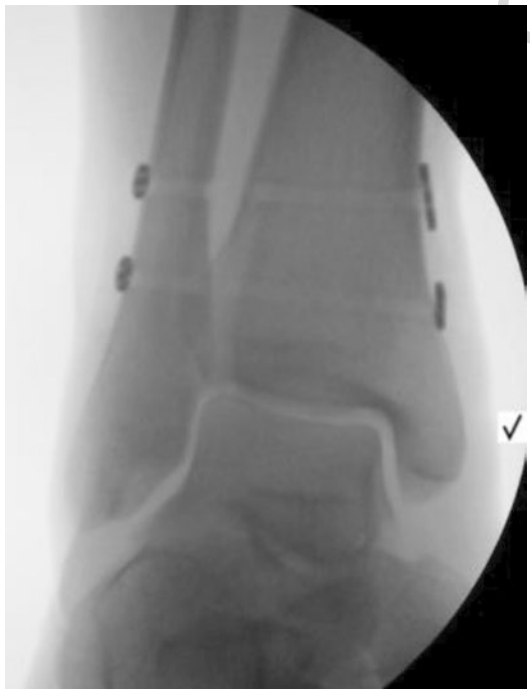

Fig. 6.8 Intraoperative fluoroscopic antero-posterior view of a double suture-button fixation

Suture-Button Constructs

While screw fixation is still considered the gold standard, there are a number of theoretical advantages of suture-button fixation (Fig. 6.8).

These have been theorized to allow physiologic motion at the syndesmosis while maintaining reduction. Further, there is less risk of symptomatic hardware and need for implant removal.

Finally, these constructs have been suggested to safely allow earlier ankle range of motion as the reduction can be held with progression of motion without the concern for implant failure (i.e., screw breakage) and recurrent diastasis [46].

The argument that these constructs might be superior because they do not require routine removal is weakened by the growing evidence against routine screw removal. However, it has been suggested that these constructs might allow earlier weight-bearing. This is due to concern that early stress on a syndesmotic screw might lead to breakage prior to ligamentous healing.

Conversely, less rigid constructs such as the TightRope (Arthrex, Naples, FL) are purported to be sturdy enough to withstand physiologic loading that occurs with weight-bearing and normal ankle motion [86].

Teramoto et al. performed a cadaveric study on six ankles comparing single suture-button fixation, double suture-button fixation, anatomic suture-button fixation (from posterior fibula to anterolateral distal tibia), and screw fixation. The authors evaluated the amount of diastasis with various stresses on the ankle, including anterior traction, medial traction, and external rotation. With single suture-button fixation the diastasis increased significantly with all forces, whereas with double fixation the diastasis increased significantly with medially directed force and with external rotation but not with anterior traction. They found that with anatomic suture-button placement, there were no significant differences compared to ankles tested prior to syndesmotic

disruption. The screw fixation proved to be the most rigid fixation, with significantly decreased diastasis compared to suture-button results [87].

However, the clinical implications of that amount of motion are not currently known. Naqvi et al. reported retrospectively on 49 patients with suture-button syndesmotic fixation. Patients with syndesmotic injuries associated with ankle fractures underwent single suture-button fixation and those with Maisonneuve injury underwent double suture-button fixation. The authors reported a mean time to weight-bearing of 7.7 ± 1.1 weeks (range 5–10) and a mean return to normal activities at 11.2 ± 1.8 weeks. They reported that the original technique of tying the knot over the lateral aspect of the fibular button resulted in a significantly higher rate of wound complications compared to their reported modified technique of creating a subperiosteal recess in the posterior fibula in which they buried the knot. They reported satisfactory results at 2 years postoperatively [86].

A recent prospective randomized trial comparing screw fixation with a single 3.5-mm screw ($n = 22$) vs. suture-button fixation ($n = 22$) of the syndesmosis revealed no difference in quality or maintenance of reduction between the two as seen on postoperative imaging. Additionally, there was no difference at 2-year follow-up in the incidence of ankle joint osteoarthrosis [88].

In 2013, Ebramzadeh et al. compared two suture-button devices (ZipTight [Biomet] and TightRope [Arthrex]) along with a 3.5-mm quadricortical screw fixation in a cadaveric, failure-to-load model. In 12 of 20 specimens, failure occurred via a fibula fracture. The screw construct was found to provide a significantly higher torsional strength than the ZipTight (30.1 vs. 22.2 Nm) but the difference seen between the screw and the TightRope was not significant.

The authors reported that there were no significant differences between the two suture-button constructs. Ultimately, they suggested that the torsional fixation strengths of all three constructs were above the physiologic loads that would "likely" be experienced during the healing process, citing that level ground walking generally creates syndesmotic torsional stresses below 2 Nm and "various other activities" generally create stresses less than 20 Nm [89].

One issue that arises with regard to the use of a suture button is how to determine the amount of force to put on the construct while securing the syndesmosis. Additionally, there has been debate regarding which position the foot should be in at the time of final tightening. A recent cadaveric study revealed that with the use of suture-button syndesmotic fixation, there was consistent overcompression compared to the intact state, with significant volume reduction and medial displacement of the fibula [50].

Overcompression, however, is not unique to suture-button constructs as it has been reported to occur with forceps reduction and screw fixation as well [90].

However, the clinical impact of overcompression of the syndesmosis is not known and it has been shown that this compression does not appear to affect ankle dorsiflexion/plantarflexion. Further, it has been shown that the position of the foot (i.e., plantarflexion, neutral, or dorsiflexion) during the time of compression and fixation has no significant effect on postoperative ankle motion [90–92].

Another recent cadaveric study compared a single screw to either a single suture-button construct or a divergent double-suture button construct [93]. The authors found that while all fixation techniques provided significant torsional stability, no technique provided the rotational stability and native anatomic relationships provided by the intact ligaments.

Further, the screw provided the most rigid restraint to anterior-posterior translation of the fibula with the highest amount of translation seen in the single suture-button group [94].

Although multiple studies have addressed biomechanical stability, Laflamme et al. reported on functional scores in addition to radiographic outcomes of patients randomized to either static fixation with a single 3.5-mm quadricortical screw ($n = 36$) or dynamic fixation with a single TightRope ($n = 34$).

Dynamic fixation resulted in improved Olerud-Molander functional scores at 3, 6, and 12 months (significant at 12 months). AOFAS

scores were significantly better in the TightRope group at 3 months only. There were four cases of lost reduction in the screw group compared to zero in the TightRope group.

Anatomic Repair of Syndesmotic Ligaments

There has been recent support for anatomic repair of the syndesmosis.

Schottel et al. in 2016 reported from a cadaveric model that anatomic repair using suture anchors for the deltoid ligament and PITFL was not significantly inferior to screw fixation in terms of external rotational stability [95].

Zhan et al. reported that patients who had augmented anatomic repair of the AITFL with a 5.0-mm anchor placed into tibia and tied to the fibular plate had better functional outcomes and earlier return to work than patients with screw fixation.

Additionally, there were significantly fewer cases of malreduction in the repair group (19.2% vs. 7.4%). The repair group had significantly higher overall range of motion, although they had significantly decreased plantarflexion compared to the screw group [68].

A recent topic of debate is in relation to fixation of the posterior malleolus and the role that it plays in syndesmotic reconstruction and stabilization. Even small posterior fragments in trimalleolar fractures can represent complete avulsion of the PITFL. Therefore, the previous teaching that posterior malleolar fractures that constitute less than 20% of the joint surface do not require fixation has been called into question.

Posterior malleolar fixation has been found to further stabilize the syndesmosis and decrease the risk of post-traumatic arthritis [53].

A cadaveric study by Gardner et al. found that in specimens with unstable syndesmoses, fixation of a posterior malleolus fracture restored 70% of preinjury stiffness compared to only 40% with screw fixation [96].

A prospective clinical study of 31 patients (9 who underwent posterior malleolus fixation and 14 who underwent screw fixation of their syndesmotic injury) revealed that fixation of a posterior malleolus fracture with the PITFL attached resulted in at least equivalent stability and clinical outcomes as trans-syndesmotic screw fixation [97]. This is typically performed through a posterolateral approach with the patient in a prone position [98].

Syndesmotic injuries are increasingly common in both competitive and recreational athletes. Although screw fixation has been shown to provide greater stability than newer suture-button constructs, the benefit of the earlier motion allowed by these constructs is not completely understood.

Although both of these techniques have the ability to overcompress the syndesmosis, it is unclear what effect this has on healing and ankle motion. Additionally, direct anatomic repair of syndesmotic ligaments with or without augmentation has shown promising results in terms of anatomic restoration of the joint with acceptable strength. At present, more work is needed to understand the long-term impact of newer treatments and the utility of more aggressive rehabilitation techniques.

6.8 New Ideas: "Syndhoo" [41]

There are no standardized criteria for the diagnosis and management of syndesmotic injuries, creating great ambiguity regarding optimal treatment. Future challenges are to identify clinical syndesmotic instability without the need of invasive arthroscopic procedures, especially in subtle (grade IIb) instabilities [41].

A grade II isolated syndesmotic injury is defined as a lesion to the antero-inferior tibiofibular ligament and the interosseous ligament of the ankle with involvement of the deltoid ligament on magnetic resonance scanning (MRI).

We tested 15 registered athletes between the age of 18 and 36 years, who presented with a grade II isolated syndesmotic injury (confirmed on MRI) between 1 January 2015 and 1 May 2017. All 15 athletes were independently tested by an experienced physiotherapist with the "syndhoo" device that we developed. They all had a grade II isolated syndesmotic injury with clinical and radiological signs of potential instability and therefore all were indicated for arthroscopy [37].

For every "syndhoo"-tested athlete, an arthroscopy was performed by the same experienced ankle surgeon at our Center between January 2017 and September 2017. During arthroscopy, the syndesmosis was considered positive (unstable) if a 4.5-mm arthroscopic shaver could be pushed through the distal syndesmosis, 1 cm proximal from the tibiotalar joint. The physiotherapist and surgeon were blinded to the other one's results. All patients were tested and treated between 1 and 4 weeks from the initial injury. The principle of this "syndhoo" device is to dynamically evaluate the distal tibiofibular stability during external rotation of the ankle as an extension to the available clinical tests. Cadaveric testing has shown that the distal syndesmosis is unstable when a force of 87–100 N is applied. The foot is positioned and fixed on the syndhoo board that rotates over the heel (Fig. 6.9a, b).

The board can be put in neutral position, 20° of plantar flexion and 20° of dorsiflexion (Fig. 6.9c, d).

The knee is stabilized through a patellar strap and the patient is tested in sitting position (Fig. 6.9b). With a dynamometer, the foot is passively externally rotated with the hinge positioned over the heel (Fig. 6.9e, f).

When the patient experiences clinical apprehension at a force <87 N, the "syndhoo" test is considered positive.

If the apprehension occurs during a force 87–100 N, the syndhoo test is considered equivocal.

When no apprehension occurs or the apprehension occurs with a force >100 N, the "syndhoo" test is considered negative.

Statistically, Cohen's kappa (κ) has been used to determine the inter-rater agreement between

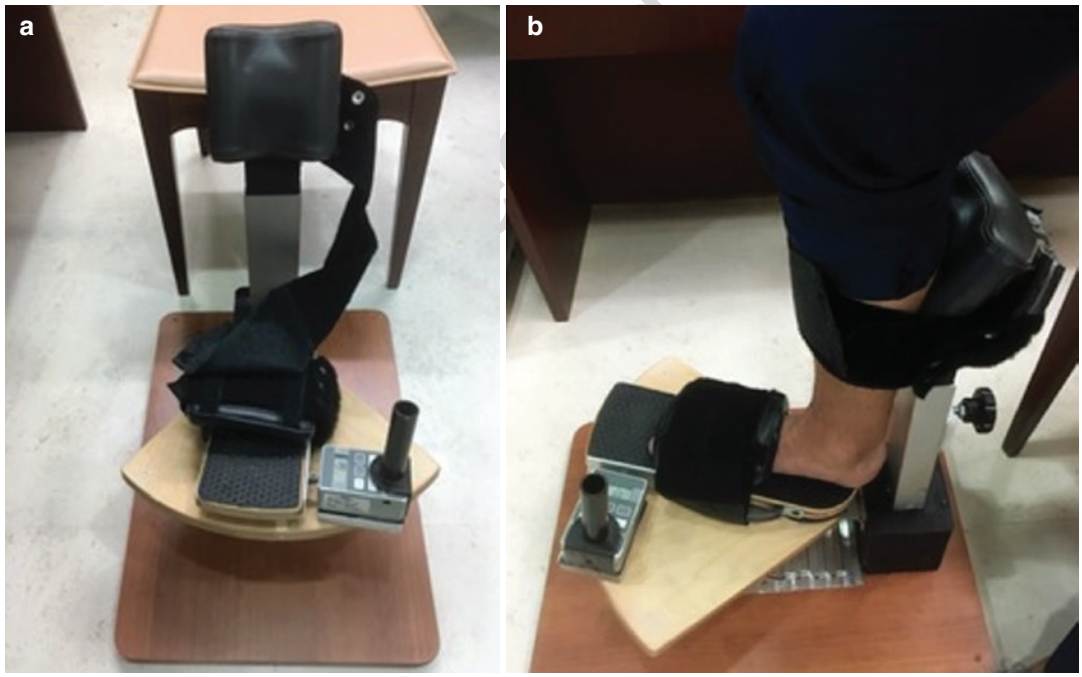

Fig. 6.9 (**a**) Image of the "syndhoo" device (front side). (**b**) Image of the "syndhoo" device from the side with the foot placed on the rotating board in neutral position. (**c**) Image of the syndhoo device from the side with the foot placed on the rotating board in 20° of plantar flexion. (**d**) Image of the syndhoo device from the side with the foot placed on the rotating board in 20° of dorsiflexion. (**e**) Image close up of the dynamometer, placed at the medial foot side of the rotating board. (**f**) Overview image of the dynamometer, linked to the rotating board

Fig. 6.9 (continued)

the arthroscopy method (as a reference) and the three "syndhoo" methods (dorsiflexion, neutral, plantar flexion).

Based on the guidelines from Altman, and adapted from Landis and Koch, Cohen's kappa (κ) is interpreted as poor agreement if less than 0.20, fair agreement if between 0.20 and 0.40, moderate agreement if between 0.40 and 0.60, good agreement if between 0.60 and 0.80, and very good agreement if between 0.80 and 1.00.

6.8.1 "Syndoo" Testing Results

"Syndhoo" dorsiflexion: When pushing manually the dynamometer in external rotation (with the board in 20° of dorsiflexion), the test is considered positive if the athlete feels apprehension at a force <87 Newton (N).

"Syndhoo" neutral: When pushing manually the dynamometer in external rotation (with the board in neutral position), the test is considered positive if the athlete feels apprehension at a force <87 Newton (N).

"Syndhoo" plantar flexion: When pushing manually the dynamometer in external rotation (with the board in 20° of plantar flexion), the test is considered positive if the athlete feels apprehension at a force <87 Newton (N).

There was very good agreement between arthroscopy and syndhoo dorsiflexion diagnosis ($\kappa = 1$, $p < 0.001$) but no significant agreement was found between arthroscopy, and "syndhoo" neutral and "syndhoo" plantar flexion ($p = 0.053$ and $p = 0.99$, respectively).

Traditionally, individuals with clinical and/or radiological suspicion of syndesmotic instability warrant an examination under anesthesia and/or diagnostic arthroscopy to confirm and treat. However, the invasive process of this has inherent risks to the patient. The described noninvasive "syndhoo" device in this chapter can be a valuable tool in the evaluation of isolated syndesmotic ankle instability.

Further studies on the correlation of this noninvasive test with clinical examination, imaging, and arthroscopic findings are needed. Ongoing work at our institution is seeking to establish the agreement between the examination described here and MR quantification of syndesmotic injury which we hope will better depict the cut-point for a positive test.

We have incorporated these finding in this chapter on novel techniques since we have found this "syndhoo" device very helpful as part of the available noninvasive options in the clinical diagnosis of syndesmotic instability [41].

6.9 Return to Play

Athletes who sustain a syndesmotic ankle sprain typically should go through much longer recovery periods than those who sustain a lateral ankle sprain [9]. Return to play (RTP) in grade I injuries is usually at 6–8 weeks post-injury, but is variable. Professional athletes with stable isolated grade II syndesmotic injuries are reported to RTP at a mean of 45 days, compared with 64 days for those with unstable grade II injuries [99]. Also, athletes with injury to both the AITFL and deltoid ligament took longer to RTP than those with an AITFL injury alone and IOL injury on MRI and PITFL injury on MRI were both independently associated with a delay in RTP [99]. In the case of surgically treated grade III injuries, the expected time frame to RTP is between 10 and 14 weeks [9, 100] although RTP as early as 6 weeks has been described in case series [101]. RTP in syndesmotic injury is permitted when able to single-leg hop for 30 s without significant pain [5]. To our knowledge, there are no specific studies on prevention of syndesmotic re-injury. Although it might be assumed that neuromuscular bracing and bracing or taping is beneficial, injury mechanisms differ and further investigation is required to increase our understanding of syndesmosis injuries and improve treatment and prevention of this significant injury [9, 28, 40].

6.10 Conclusion

Syndesmosis injuries are increasingly common in both competitive and recreational athletes. Recent advances in the diagnosis and management enable

early detection of these injuries that can avoid evolution to chronic debilitating ankle conditions.

Despite improved insights in this multifactorial pathology, more work is needed to understand the long-term impact of the newer treatments and the utility of more aggressive rehabilitation techniques.

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Subtalar Joint Instability

7

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7.1 Anatomy

The subtalar joint (STJ), also known as the talocalcaneal joint, is an important and complex joint in the hindfoot that allows articulation of the talus and calcaneus. It consists of three articular facets between the inferior surface of the talus and the dorsal surface of the calcaneus (Fig. 7.1). STJ is formed by two articular components: the anterior talocalcaneal articulation and the posterior talocalcaneal articulation [1].

The anterior talocalcaneal articulation is formed by the anterior and middle facets of anterior one-third of the calcaneum that articulate with the head of the talus and the proximal navicular surface. The joints are connected by a fibrous capsule, the talonavicular ligament (a fibrous band which connects the neck of the talus to the dorsal surface of the navicular), the plantar calcaneo-navicular ligament (a broad thick band which connects the anterior margin of the sustentaculum tali of the calcaneus to the plantar surface of the navicular, also called "spring ligament"), and the calcaneo-navicular portion of the bifurcated ligament (also called "Y shaped" ligament, a strong band which originates from the anterior surface of the calcaneus and splits anteriorly into the calcaneo-cuboid portion, which lies in the horizontal plane and attaches to the dorsal aspect of the cuboid, and the calcaneo-navicular portion, which lies in the vertical plane and attaches to the lateral aspect of the navicular) [2].

The posterior talocalcaneal articulation is formed by the posterior calcaneal facet on the inferior surface of the talus and the posterior facet on the superior surface of the calcaneus and makes up the largest articulation between the talus and calcaneus. The joint is surrounded by a fibrous capsule and synovial membrane that attach at the edges of the articular surface. However orthopedic surgeons consider the talocalcaneal joint and the talocalcaneonavicular joint to be one functional unit [2].

The subtalar joint essentially is a uniaxial joint at which the calcaneus rotates from dorsolateral to medioplantar. The axis of motion passes obliquely from a posterior, plantar, and lateral position to an anterior, dorsal, and medial position. STJ range of motion (ROM) is approximately from 25 to 30° of inversion/supination to 5 to 10° eversion/pronation [3–5]. However the

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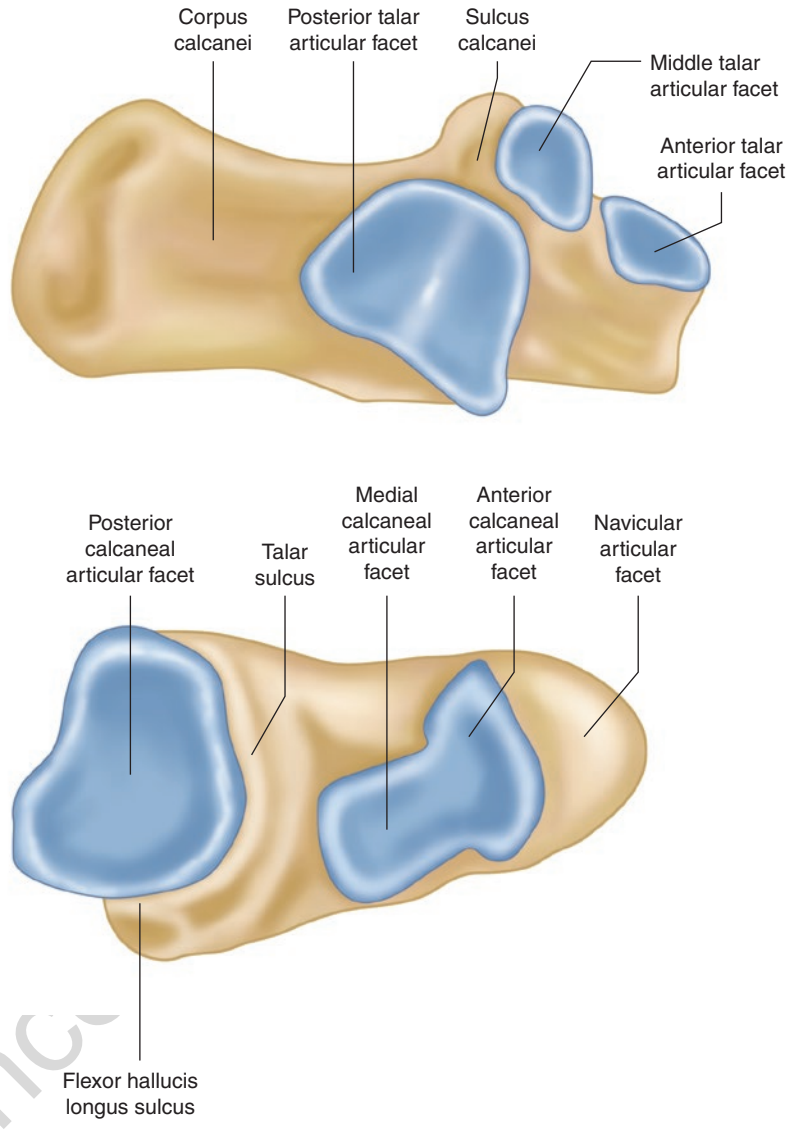

Fig. 7.1 Inferior surface of the talus and the dorsal surface of the calcaneus

STJ motions are linked to the ankle joint motions and to the midtarsal joint motions. Subtalar ligaments can be divided into intrinsic ligaments (interosseous talocalcaneal ligament—ITCL, cervical ligament—CL, lateral, posterior, and medial talocalcaneal ligament) and extrinsic ligaments (calcaneo-fibular ligament—CFL and the tibio-calcaneal fascicle of the deltoid ligament).

The ITCL is a dense, broad, and flat bilaminar bundle that descends obliquely and laterally from the sulcus tali to the calcaneal sulcus and runs through the sinus tarsi. The posterior band of ITCL lies posterior to the anterior band. ITCL attaches to the sinus tarsi anterior to the superior posterior articular facet of the calcaneus and inserts into the sinus tali just anterior to the posterior inferior articular facet of the talus [2]. It is the primary restraint of the subtalar joint and can be classified according to its shape in band type, fan type, and multiple type [6]. The ITCL can be compared with the cruciate ligaments of the knee for its stabilizing and proprioceptive function [7].

The CL is located along the antero-lateral portion of the STJ and is the strongest ligament con-

necting the talus and the calcaneus [8]. It is attached to the upper surface of the calcaneus and passes superiorly and medially to a tubercle on the inferior and lateral aspect of the neck of the talus [2]. The primary function of the CL is to resist excessive STJ supination whereas the ITCL remains taut during pronation.

The lateral talocalcaneal ligament arises from the lateral tubercle of the talus, runs obliquely inferiorly and posteriorly, and attaches to the lateral surface of the calcaneus [2].

The posterior talocalcaneal ligament arises from the lateral tubercle of the talus and inserts on the proximal and medial portion of the calcaneus [2].

The medial talocalcaneal ligament connects the medial tubercle of the talus with the posterior and medial aspect of the calcaneus [2].

The calcaneo-fibular ligament is a narrow, rounded cord, running from the tip of the lateral malleolus of the fibula downward and slightly backward to a tubercle on the lateral surface of the calcaneus. It restricts the hyperinversion of the subtalar joint.

The tibio-calcaneal fascicle of the deltoid ligament arises from the medial malleolus, descends almost perpendicularly, and inserts into the whole length of the sustentaculum tali of the calcaneus.

The extensor retinaculum significantly contributes to stability of the ankle and subtalar joint. Weindel et al. demonstrated in a biomechanical cadaver study that dissection of the inferior extensor retinaculum results in a significant increase in eversion and inversion [9].

7.2 Pathophysiology

Subtalar instability (STI) is a chronic functional talocalcaneal instability characterized by a combination of anterior movement, medialization, and varus tilt of the calcaneus [10].

Subtalar instability is a problem because it can lead to severe flatfoot with growing pain and quick fatigue while walking and running. It can lead to many orthopedic problems affecting ankle, knee, hip joint, and lower back and result in clinical presentations like anterior or posterior tibial tendinopathy, plantar fasciitis, and forefoot pain [11].

STI could be a consequence of acute subtalar injury or dislocation; however chronic tear or insufficiency of interosseous talocalcaneal ligament (ITCL), cervical ligament (CL), and calcaneofibular ligament (CFL) have been reported as the most frequent etiologies of STI [12].

Acute injury of ST joint is common in basketball and volleyball players and it is seen when the player comes to an abrupt stop [13].

Acute subtalar dislocation is a relatively uncommon injury that occurs frequently in the third decade of life in male patients after motor vehicle accidents [14, 15]. Frequently the subtalar dislocation is closed; however, between 10 and 40% of all cases, high-energy injuries may lead to open subtalar dislocation. Medial dislocations are the most common, followed by lateral and posterior.

Acute subtalar dislocation is caused by forced inversion combined with the ankle in dorsiflexion or the neutral sagittal position. The CFL is the first to be damaged, followed by the lateral talocalcaneal ligament, the cervical ligament, and finally the ITCL. Dislocation of subtalar joint is often associated with fractures of the fifth metatarsal, the talus, or the malleoli. However isolated subtalar dislocation is common in patients with aplasia of the ankle ligaments or the calcaneus facets, hypoplasia of the malleolus, recurrent ankle sprains, post-traumatic ligamentous insufficiency, and atrophy of the peroneal muscles [16]. Broca distinguished three types of subtalar dislocation: (1) the medial dislocation; (2) the lateral dislocation; and (3) the posterior dislocation. Direction of the rest foot in relation to the talus was the determinant element to classify dislocation as medial, lateral, or posterior [17]. Malaigne and Burger described an additional type of subtalar dislocation, the anterior dislocation [17]. After an acute dislocation, conservative treatment with closed reduction under general anesthesia and an ankle brace for 3–6 weeks, followed by physical therapy, is recommended. However the interposition of posterior tibialis tendon after the rupture of the flexor retinaculum or the interposition of the extensor retinaculum makes the dislocation not reducible. In this case an operative treatment is required [4].

Chronic tear or insufficiency of ligaments could be a consequence of recurrent ankle sprains [18].

Subtalar instability is frequently accompanied by ankle instability. On the other hand, lateral ankle instability may be combined with subtalar joint involvement in up to 25% of the cases [10].

7.3 Diagnosis

Clinical symptoms of ankle and subtalar instability are very similar and therefore a correct diagnosis is not easy. A feeling of uncertainty during walk on uneven ground is a common finding. Other symptoms include recurrent swelling, painful stiffness of the subtalar joint, and diffuse pain in the hindfoot and onto the sinus tarsi.

In the acute phase lateral ecchymoses, swelling, and tenderness in the area of the sinus tarsi can be found. In contrast to chronic ankle instability female patients with STJ instability may prefer high-top shoes [18].

The instability of the subtalar joint has been assessed clinically by a manual inversion stress test. An increased amount of inversion is revealed stressing the hindfoot. However, after acute injury it may be problematic to recognize an increased amount of calcaneal inversion compared with the intact side due to pain-induced limitations [19].

Radiographic examination of STJ instability involves stress Broden views [20]. To perform the stress Broden view, the examiner internally rotates the foot, the beam is centered on the talonavicular joint, and the tube is angled from 30 cephalad. This positioning allows the surgeon to view different portions of the posterior facet of the STJ. Separation of the posterior facet of the calcaneus and talus greater than 7 mm may indicate chronic subtalar instability [20].

CT scan may be helpful. Some investigators have recommended its use because of the inaccuracies of stress radiographs [21, 22]. CT allows an accurate analysis of any type of osseous deformity or osteoarthritis.

MR imaging has been shown to have significant role in the detection of injured structures [22]. Moreover, MRI can be useful in evaluating the joint surfaces for osteochondral defects and identifying peroneal tendon injury. With MRI, a partial or complete tear of components of the ligaments contributing to subtalar stability may be diagnosed as well as an acute involvement of the subtalar joint by bone marrow edema in T2-weighted sequence.

Arthrography of the ankle and STJ can also be used for the evaluation of ruptured ligaments and associated pathologic condition. Sugimoto et al [23] attributed to arthrography a sensitivity of 92% and a specificity of 87% for the diagnosis of CFL rupture in patients with recurrent ankle instability.

7.4 Treatment

The treatment of acute STJ injury consists of wearing an ankle–foot orthosis within the shoe for 5–6 weeks [24]. In chronic injury nonoperative treatment is essential and involves physical therapy directed at the soft tissue envelope and dynamic stabilizers, taping, proprioceptive training, stretching of the Achilles tendon, and lateral wedging of the shoe or insole up to 0.5 mm for 12–16 week [18]. If conservative treatment is unsuccessful, operative treatment may be an option to restore stability and function to the joint. However, normal subtalar joint kinematics are not restored by tenodesis ligament reconstruction [25, 26]. Techniques for surgical reconstruction generally are divided into anatomical and nonanatomical reconstruction, such us tendon transfer procedures.

Broström first introduced a direct anatomic repair of the ruptured ATFL and CFL, with good long-term results and functional recovery (Fig. 7.2) [27]. The Gould modification of the Brostrom procedure associated the direct repair of the lateral ligaments with extensor retinaculum reinforcement (Fig. 7.3) [27]. Brostrom Evans procedure adds to Brostrom repair the transfer of the anterior third of the peroneus brevis tendon to provide supplemental lateral static restraint [28], but it increased stiffness and had poor long-term patient satisfaction [29]. Moreover it has been suggested that the

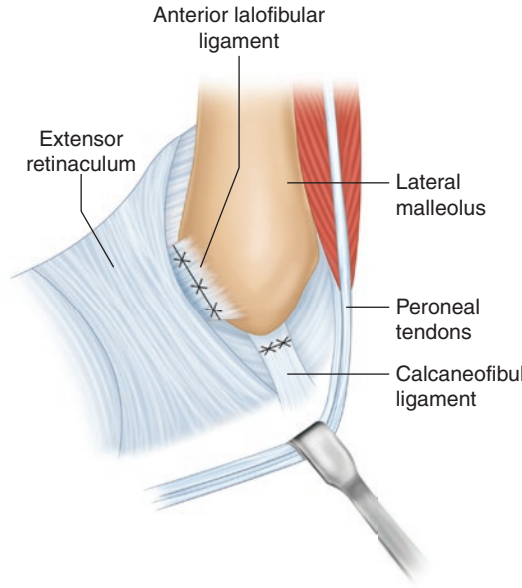

Fig. 7.2 Brostrom procedure

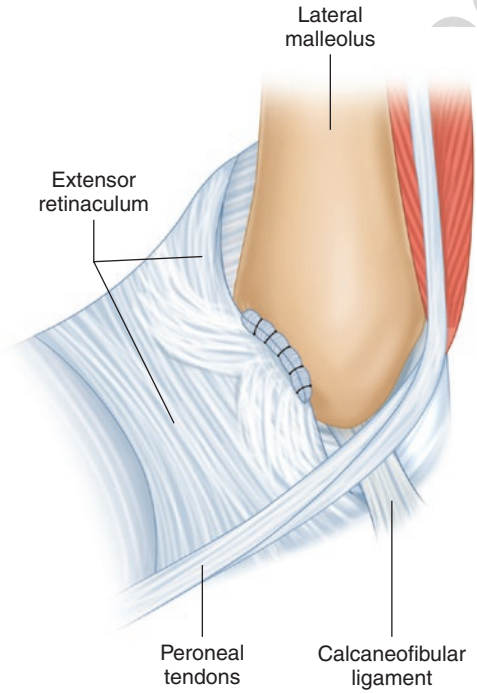

Fig. 7.3 Gould modification of the Brostrom procedure

Evans procedure is ineffective for SJI [30]. However, especially for STJ instability, tenodesing procedures may be considered advantageous because of the reduction of hindfoot motion. For this reason Chrisman-Snook procedure has become the procedure of choice for patients with isolated STJ instability [24]. The Chrisman-Snook tenodesis consists in the use of a split peroneus brevis tendon: the proximal part of the tendon is passed through the fibula in an anterior to posterior direction and finally the tendon is fixed to the calcaneus near the original insertion of the CFL. Other procedures addressed in the literature include ITCL reconstruction, ligamentous reconstruction using the entire peroneus brevis tendon to recreate the ATFL and CFL, and triligamentous reconstruction procedures to address the ruptured ATFL, CFL, ITCL, and cervical ligament [31]. Kato performed an ITCL reconstruction with a partial Achilles tendon graft with good functional results and a very low rate of postoperative complications [32]. Pisani used the anterior half of the peroneus brevis tendon for reconstruction of ITCL with an open surgical technique [33]. Liu described an arthroscopic approach with a gracilis tendon from the ipsilateral knee as a graft with controversial results [34].

Surgery is a successful solution for patients with STJ instability; however, hindfoot malalignment can contribute to subtalar joint instability and dysfunction and can be a cause of surgery's failure [35].

7.5 Surgical Technique of Brostrom-Gould Procedure

Brostrom procedure is performed with the patient placed in the lateral decubitus position. The borders of the fibular malleolus and the location of the anterior talo-fibular and calcaneo-fibular ligaments are identified. The skin incision is inferiorly to the tip of the fibula ending just posterior to the lateral malleolus and extends across the body of the ATFL and CFL. Careful dissection is critical to avoid damage to dorsal cutaneous and sural nerves.

After the identification of the intra-capsular ATFL the ligament is incised and the midportion removed. Dissection is then directed toward the distal portion of the fibula. The peroneal sheath is incised, peroneal tendons are retracted, and CFL is identified. The lax portion of the ligament is removed and the remaining portions saturated with a nonabsorbable suture. The foot is dorsiflexed and evefied and the ATFL ligament is saturated. Finally the extensor retinaculum is identified and its lateral border is brought superficial to the ATFL repair and sutured to the fibular periosteum. The subcutaneous tissue and skin are then closed. The patient is maintained in a non-weight-bearing orthosis. After about 1 month the ankle is protected with an air stirrup brace, and range of motion exercises are begun [36, 37].

7.6 Surgical Technique of Chrisman-Snook Procedure

The procedure is performed with the patient placed in the lateral decubitus position. The skin incision is made from the mid-calf laterally along the course of the peroneal tendons beneath the lateral malleolus and turning down to the base of the fifth metatarsal, reminding a single "hockey stick"; however some authors prefer a three incisional approach incision. In this case the first incision is placed over the peroneal tendons posterior to the distal fibula, the second over the sinus tarsi, and the third laterally over the posterior tubercle of the calcaneus. The peroneal brevis tendon is identified and split. Once the tendon is split, half of it is transected proximally, so the distal half may be pulled into the anterior incision. The tendon graft is passed subcutaneously from the base of the fifth metatarsal to superiorly to the sinus tarsi region, and after through the distal fibular using a tendon passer. The foot is placed in an ankle-neutral STJ-everted position and the peroneal brevis tendon is sutured to the anterior fibular periosteum. A second subcutaneous tunnel is made from the lateral malleolus to the lateral wall of the calcaneus and the tendon is inserted into the calcaneus using an anchoring device [37].

7.7 Conclusion

Subtalar joint pain and instability is a common problem. The estimated number of unknown cases with chronic subtalar instability might be substantially higher than the number of patients where we actually recognize this diagnosis. A high degree of suspicion is necessary for the correct diagnosis. Moreover weight-bearing X-rays including Broden views, CT scan, MR imaging, and arthrography could be helpful. The treatment of acute injuries is conservative and has good outcomes. The situation is less clear for operative approaches although tenodesing procedures had showed good clinical outcomes. There is a definitive need for prospective and controlled studies to get a more reliable answer regarding subtalar joint pathology.

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Lisfranc Complex Injuries

8

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8.1 Introduction

Lisfranc or tarsometatarsal (TMT) joint complex injuries involve disruption of one or more of the osseous or ligamentous stabilizers of the transverse arch of the midfoot [1, 2]. The TMT joint complex encompasses the bases of the first through fifth metatarsals and their respective articulations with the three cuneiform bones and the cuboid bone [3]. The Lisfranc joint is stabilized by dorsal, interosseous, and plantar ligaments that tether the lateral border of the medial cuneiform to the medial border of the second metatarsal base. These three ligaments are collectively known as the "Lisfranc ligament" [3, 4].

Injuries to the Lisfranc or TMT joint complex are generally rare and almost 20% are missed or misdiagnosed on initial imaging [3, 5, 6]. Lisfranc injuries often occur as a result of axial loading of the plantarflexed foot and may involve any of the joints in the TMT complex [3, 7]. Injuries may occur via high- and low-energy mechanisms [3, 8]. However, low-energy mechanisms constitute a significant number of Lisfranc injuries seen in the athlete. Injuries to the Lisfranc joint are potentially career-ending for the athlete, as they often result in significant long-term morbidity such as post-traumatic osteoarthritis, anatomic deformity, and functional disability [1, 3, 8, 9]. Early diagnosis and appropriate management of a Lisfranc injury is therefore essential [2]. While non-operative management is a feasible option for stable injuries, surgical treatment is typically recommended for unstable injuries [8, 9].

The objective of this chapter is to discuss the current treatment options for Lisfranc injuries. However, the literature regarding operative management of Lisfranc injuries is in need of more high-quality, randomized controlled trials before any definitive recommendations regarding optimal surgical techniques can be made.

8.2 Clinical Evaluation

Prior to intervention, a surgeon should obtain a detailed history, with special emphasis placed on the mechanism of injury [10]. Lisfranc injuries can occur in acute traumatic settings from both high- and low-energy mechanisms. In athletes, Lisfranc injuries often present with subtle signs following a low-velocity mechanism [8]. The position of the foot and the direction of force applied at the time of injury are key aspects of the history. Injuries classically occur with axial loading of the foot in a hyper-plantarflexed position [3, 8].

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Medial plantar ecchymosis of the midfoot is a hallmark of Lisfranc injury [8, 10]. Other additional findings include midfoot edema and tenderness to palpation. Passive flexion of the metatarsal (MT) heads as well as passive abduction-adduction through the forefoot may demonstrate instability within the TMT joint. Special tests such as pronation-abduction of the forefoot and the TMT compression test may elicit pain in the injured region of the midfoot [9]. Examination should always include a thorough neurovascular assessment as dislocation of the second metatarsal can compromise blood flow through the dorsalis pedis artery. Additionally, diffuse swelling may lead to compartment syndrome [3, 8, 10].

8.3 Radiographic Evaluation

Weight-bearing radiographs of both the injured and uninjured foot should be obtained in addition to the standard non-weight-bearing AP, oblique, and lateral views of the foot [5, 10]. It is recommended that radiographs include imaging of the ankle, as concomitant injuries may be missed [11].

On radiographic imaging of a Lisfranc injury, there will be intra-articular displacement throughout the TMT joints, the intercuneiform joints, and/or the naviculo-cuneiform joint which is distinct from an uninjured radiograph [10] (Fig. 8.1). Any displacement of more than 2 mm in any plane around the TMT joint should raise suspicion for a Lisfranc injury [8]. The "fleck sign" indicates an avulsion of the second metatarsal base into the interval between the first and second metatarsals. This radiographic sign is pathognomonic of a Lisfranc injury [3, 9, 10] (Fig. 8.2). Additionally, the lateral radiograph may reveal either dorsal or plantar displacement of the affected joints as well as an overall flattening of the medial column [3].

Stress radiographs may be necessary in patients with indeterminate weight-bearing images. Advanced imaging is useful when there

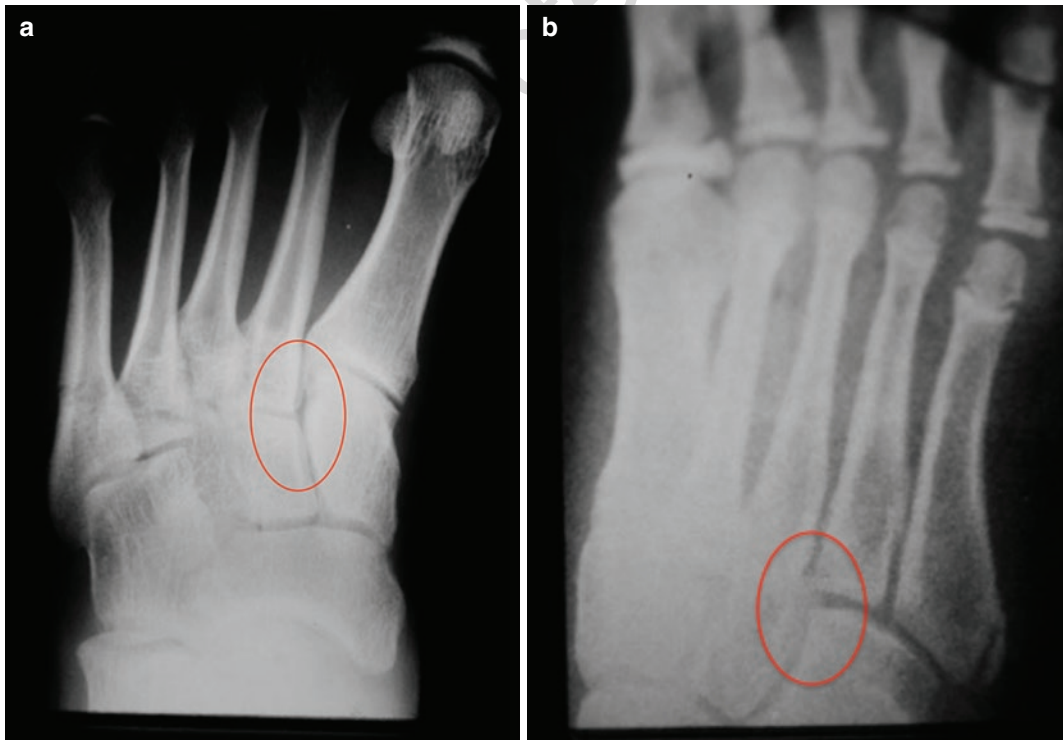

Fig. 8.1 Normal radiographic findings of the Lisfranc joint. (**a**) **AP**: Alignment of the medial border of the second MT with the medial border of the middle cuneiform. (**b**) **Oblique**: Alignment of the medial border of fourth metatarsal with the medial border of the cuboid bone

is a high index of suspicion for a Lisfranc injury in the setting of inconclusive plain radiographs or in patients who are unable to perform weight-bearing imaging [5, 12, 13]. CT scan may identify occult fractures, assess intra-articular extension of fractures, and detect subtle subluxation of pertinent joints [13]. MRI may be useful in the evaluation of the extent of soft tissue damage associated with purely ligamentous Lisfranc injuries [12]. Advanced imaging may also provide additional benefit in preoperative planning for severely comminuted osseous injuries [8].

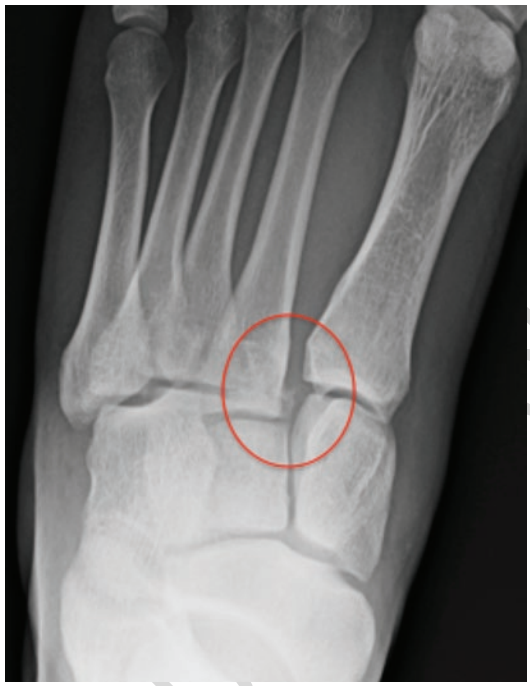

Fig. 8.2 The "fleck" sign

8.4 Lisfranc Injury Classification

There are a variety of classification systems for Lisfranc injuries, although none have demonstrated significant efficacy in determining optimal management or predicting outcomes.

In 1909, Quenu and Kuss were the first to use standardized terminology to describe Lisfranc injuries using a system based on mechanism of injury and the direction of the metatarsal dislocation [3, 12]. The terminology was later modified in 1982 by Hardcastle et al. who observed that the level of joint displacement seemed to have a greater influence on prognosis than mechanism of injury [3, 5, 11, 14]. In 1986, Myerson et al. used the scaffold of the earlier classifications to develop a system based on the columnar structure of the foot. The medial column consists of the first TMT and medial naviculo-cuneiform joints. The middle column comprises the articulations between the second and third TMT joints as well as the articulations between the middle and lateral cuneiforms and the navicular. The lateral column encompasses the articulations between the fourth and fifth metatarsals and the cuboid bone [3, 10, 14]. The Myerson Classification emphasizes the strong prognostic implications of column-specific midfoot motion and is currently the most commonly used system [3]. However, the current classification systems often only describe the high-energy or traumatic subset of Lisfranc injuries [7]. Thus, more recently in 2002, Nunley and Vertullo developed a classification system to specifically describe the more subtle, low-energy Lisfranc injuries occurring in athletes [3, 7, 8, 15] (Table 8.1).

Table 8.1 Lisfranc injury classifications

Quenu and Kuss (1909)		Hardcastle (1982)		Myerson (1986)		Nunley and Vertullo[a] (2002)	
Homolateral	All MTs displaced in the same direction	A	Complete displacement of all MTs	A	Total incongruity	I	Negative radiographs Increased uptake on bone scan
Isolated	Displacement of only one or two MTs	B	Displacement of one or more MTs	B	Partial incongruity B1: Medial B2: Lateral	II	1–5 mm diastasis between first and second MTs No loss of midfoot arch height
Divergent	MTs displaced in different directions	C	Divergent	C	Divergent C1: Partial C2: Total	III	>5 mm diastasis Loss of midfoot arch height

[a]Classification criteria are based on comparison with the uninjured contralateral foot

While classification systems effectively standardize terminology and provide a method to communicate injury patterns, many surgeons use clinical signs of instability in lieu of structured classifications to guide their medical decision-making. Clinically, Lisfranc injuries may be classified based on stability: unstable injuries present with mild to marked displacement (>2 mm) and typically require surgery while stable injuries with no or minimal displacement (>2 mm) are variably amenable to non-operative management [8].

8.5 Non-operative Treatment

All Lisfranc injuries in the acute setting should be managed following the standard PRICE-M approach: protection with immobilization, rest with weight-bearing restrictions, ice, compression, elevation, and medications for analgesia.

Following confirmation with either stress radiographs, CT scan, or MRI, stable Lisfranc injuries, whether osseous or ligamentous, can be managed non-operatively for the duration of the treatment protocol [8–10]. Other indications and contraindications for non-operative treatment of Lisfranc injuries are described in (Table 8.2). If there is mild displacement, a closed reduction should be achieved using axial traction and direct manipulation of the metatarsal bases. Percutaneous Kirschner wire (K-wire) fixation may be performed to provide stability to the reduction of simple Lisfranc injuries [5]. When used, K-wires should be directed obliquely across the metatarsal base and into the adjacent tarsal bone. Due to the oblique placement, loss of correction and migration of the metatarsal heads may be better avoided [16].

The non-operative treatment timeline should be individualized for each patient (Table 8.3). Cast immobilization is indicated once there is significant reduction in soft tissue swelling [15]. Patients should be evaluated every 2 weeks with weight-bearing plain radiographs to assess

Table 8.2 Indications and contraindications of management for Lisfranc injury

	Indications	Contraindications
Non-operative management	Stable ligamentous injury No static or dynamic displacement Stable osseous injury None to minimal displacement Latent instability in nonathletes Pes cavus deformity	Unstable Lisfranc injuries TMT joint misalignment First and second metatarsal diastasis >2 mm Latent instability in athletes
Operative management	Emergent injuries[a] Open fracture-dislocation Vascular compromise Acute neuropathy Compartment syndrome Unstable ligamentous injury[d] Unstable osseous injury Irreducible fracture-dislocation[a] Static malalignment Latent malalignment Intercuneiform displacement >2 mm Displacement between medial cuneiform and second MT >2 mm Latent instability in athletes Comminuted fractures[b] Athletes Pes planus deformity[b]	Poor surgical candidates Poor wound healing Significant soft tissue injury Vascular insufficiency Medical comorbidities Socioeconomic factors Psychiatric illness Nonambulatory patients

[a]Absolute indication
[b]Relative indication

Table 8.3 Lisfranc injury treatment timeline

Time	Non-operative management[a]	Operative management
Acute injury	PRICE-M protocol[b] Non-weight-bearing Immobilization CAM boot Short-leg cast After reduction of edema	PRICE-M protocol Non-weight-bearing Immobilization CAM boot Short-leg cast Delay surgery for 1–2 weeks after reduction of edema[c]
0–2 weeks	Weight-bearing radiographs If stable Non-weight-bearing Immobilization Short-leg cast If unstable Refer for orthopedic evaluation	Operative intervention performed Immediate postoperative period Non-weight-bearing Plaster splint
2–6 weeks	Weight-bearing radiographs Non-weight-bearing Immobilization Short-leg cast	Removal of sutures Non-weight-bearing Immobilization Short-leg cast CAM boot
6–8 weeks	Weight-bearing radiographs Non-weight or heel-weight-bearing Immobilization Short-leg cast CAM boot	Heel-weight-bearing[d] Immobilization Short-leg cast CAM boot
8–10 weeks		Removal of K-wires in the lateral column [1] Partial weight-bearing[d] Immobilization Short-leg cast CAM boot
10–12 weeks	Weight-bearing radiographs Partial weight-bearing Stiff-sole shoe Semi-rigid arch support orthotic	Weight-bearing radiographs Progressive weight-bearing as tolerated CAM boot Semi-rigid arch support orthotic
12–16 weeks	Weight-bearing radiographs Full weight-bearing Stiff-sole shoe Semi-rigid arch support orthotic Gradual return to sport	Weight-bearing radiographs Athletes <200 lb Full weight-bearing Stiff-sole shoe Semi-rigid arch support orthotic Athletes >200 lb Progressive weight-bearing as tolerated CAM Boot Semi-rigid arch support orthotic
24 weeks	Full return to sport Semi-rigid arch support orthotic	Athletes <200 lb Full return to sport Semi-rigid arch support orthotic Athletes >200 lb Full weight-bearing Stiff-sole shoe Semi-rigid arch support orthotic

[a]Return/persistence of pain or tenderness to palpation should immediately prompt phase regression and secondary evaluation with advanced imaging.

[b]PRICE-M stands for protection (immobilization), rest (weight-bearing restriction), ice, elevation, medication (analgesia)

[c]Non-emergent Lisfranc injuries

[d]Dependent upon patient weight and fixation construct

alignment and stability of the Lisfranc joint. At 6–8 weeks post-injury, patients may be transitioned into a low profile Controlled Ankle Movement (CAM) boot or short-leg cast. If used, K-wires may also be removed at this time. However, the patient will continue non-weight-bearing or heel-weight-bearing restrictions until week 8 or 10. After 10 or 12 weeks, patients may be weaned from the CAM boot or short-leg cast into a stiff-sole shoe with well-molded arch support. Over the course of 2 week, patients will transition to partial weight-bearing [8]. Patients may continue to increase weight-bearing intensity every 2 weeks. Full weight-bearing is not recommended prior to 12 weeks post-injury. Physical therapy may be prescribed to assist with strengthening and gait training [3]. Return of pain or tenderness to palpation at any time during treatment should prompt phase regression and secondary evaluation with imaging [12]. Recovery from a Lisfranc injury may take up to 4 months. Life-long use of a semi-rigid arch support is often recommended [3].

8.6 Non-operative Complications

Complications of non-operative management are attributed to difficulty in obtaining adequate reduction and relative instability of non-operative methods in achieving immobilization of the TMT joint. Closed reduction is often obstructed by bony fragments and soft tissue between the fractured or dislocated structures [16]. Casting provides poor immobilization of the disrupted Lisfranc joint when the integrity of the capsular and ligamentous structures is compromised [1]. Due to failure to maintain reduction and subsequent irritation due to increased motion at the affected joint, non-operative management has been associated with symptomatic degeneration and reflex sympathetic dystrophy syndrome. K-wire fixation has been associated with loss of reduction due to proximal migration of the metatarsals as well as osteolysis and infection along the pin tract [5, 16].

8.7 Non-operative Outcomes

Outcomes following non-operative management of Lisfranc injuries vary based on the severity of injury. Injuries involving a mild degree of TMT displacement tend to have fair outcomes. However, this may not be the case in athletes. Curtis et al. report treatment failure and inferior results following non-operative management in athletes with minimal Lisfranc instability [9]. Closed reduction and casting is reliably unsuccessful in the majority of moderate to severe cases [1, 4, 11, 17]. Furthermore, due to articular damage sustained at the time of injury, many patients develop painful, symptomatic midfoot arthritis and may require fusion of the TMT joint [4, 13].

Although the most invasive nonsurgical option, closed reduction and percutaneous pinning with K-wire has also been conceded as ineffective for unstable Lisfranc injuries due to the high rate of treatment failure [18, 19]. K-wire fixation is recognized as inferior in achieving rigid reconstruction of the Lisfranc joint when compared to cortical screw fixation [2].

8.8 Operative Treatment

Absolute indications for operative management of a TMT joint complex injury include open injuries, acute vascular compromise, neurologic damage, compartment syndrome, and unstable fracture-dislocations [8]. Other indications and contraindications for surgical management of Lisfranc injuries are described in Table 8.2.

Acute, unstable Lisfranc injuries with minimal displacement may be treated electively with surgery in the outpatient setting [8]. Surgery is often delayed for at least 2 weeks to allow for resolution of the associated edema and healing of the damaged soft tissue envelope [3, 10, 19]. Acute, unstable Lisfranc injuries with moderate to severe displacement should be treated surgically as soon as clinically possible. Immediate surgical intervention with external fixation or ORIF is particularly warranted if the acute injury

is open or accompanied by neurovascular compromise or compartment syndrome [19]. Chronic unstable or severely comminuted Lisfranc injuries may require primary TMT arthrodesis.

The primary goal of operative management is to restore stability and biomechanical function to the midfoot. As such, maintenance of the anatomic relationships between the bony and soft tissue structures that stabilize the TMT joint complex should be prioritized intraoperatively in order to promote optimal postoperative outcomes.

8.8.1 Preoperative Planning

The patient is positioned supine on a flat Jackson table with a soft bump placed underneath the ipsilateral hip. The bump provides internal rotation to the lower extremity, which allows the foot to remain in optimal, neutral alignment throughout the procedure. All bony prominences are well padded and the contralateral limb is secured to the table. The entire length of the ipsilateral limb should be draped out to allow manipulation of the lower extremity during surgery. A tourniquet for the lower extremity is often utilized during the operation. A tourniquet cuff may be placed on the thigh or calf and inflated or deflated intraoperatively as necessary. The preference at our institution is to place a sterile tourniquet on the ankle using a 4-inch non-latex elastic bandage. The foot may be placed on a sterile radiolucent triangle or a bump to allow for further manipulation intraoperatively. Our preference is to use a sterile bump under the ipsilateral ankle.

General anesthesia or regional anesthesia using a spinal, popliteal fossa, or ankle block may be employed. The authors prefer general anesthesia in conjunction with an ankle block. A local anesthetic (1% lidocaine with 0.25% Marcaine) may be injected into the surgical incisions either preoperatively or postoperatively to provide additional analgesia.

Fluoroscopy is used to identify Lisfranc joint instability, confirm reduction of the TMT joint fracture-dislocation, guide hardware trajectory, and assess the adequacy of anatomic fixation.

Various types and combinations of hardware have been employed for fixation of unstable Lisfranc injuries (Table 8.4). The authors prefer to use K-wires, standard AO screws, and dorsal

Table 8.4 Hardware for operative fixation of Lisfranc injuries

Hardware	Indications	Advantages	Disadvantages
Kirchner (K) wires	Fourth TMT joint Fifth TMT joint	Preserves natural motion of the lateral column	High rates failure when used alone
Standard AO screws	Lisfranc joint Intercuneiform Medial column Middle column	Strong Rigid	Iatrogenic cartilage damage Hardware failure Removal of hardware
Bio-absorbable polylactide screws	Lisfranc joint Intercuneiform Medial column Middle column	Strong Rigid No removal of hardware	Iatrogenic cartilage damage Hardware failure
Extra-articular dorsal plate	ORIF Intra-articular cartilage Multiple unstable TMT joints Adjunct to screw fixation Primary arthrodesis Severely comminuted fractures Significantly damaged intra-articular cartilage	Strong Rigid Preserves cartilage	Plantar gapping Hardware irritation Longer operating time Non-union Mal-union
External fixation	Open injuries Significant edema	Strong Rigid Temporary stabilization	Infection Delayed treatment

Table 8.5 Surgical techniques for Lisfranc injuries

Method	Hardware	Indications	Contraindications
Closed reduction, percutaneous fixation	K-wire	Stable closed injuries Low energy trauma	Unstable injuries Open injuries
Closed reduction, external fixation	External fixator	Stable closed injuries High energy trauma Significant edema	Unstable injuries Open injuries
Open reduction, external fixation	External fixator	Open injuries High energy trauma Compartment syndrome	Stable injuries
Open reduction, internal fixation	K-wires Standard AO screws Extra-articular dorsal plate Bio-absorbable polylactide screws Combination	Unstable injuries Moderate to severe displacement (>2 mm) Moderate to severe angulation (>15°) Athletes Low energy trauma Failed closed reduction and percutaneous fixation	Stable injuries Reducible with splint Significant edema
Primary arthrodesis	K-wires Extra-articular dorsal plate Standard AO screws Combination	Medial column injuries >50% articular cartilage damage Severely comminuted fractures High energy trauma Unstable, purely ligamentous Failed ORIF	Lateral column injuries

extra-articular plates. However, the type and combination of hardware used varies based on the individual injury pattern as well as patient-specific demographic factors.

8.8.2 Operative Techniques

ORIF and primary arthrodesis are the most widely used techniques of operative management for Lisfranc injuries [17]. ORIF and primary arthrodesis reliably return stability to the Lisfranc joint; however, it is debated which surgical technique optimally restores anatomic function to the midfoot [2]. The indications and contraindications of ORIF versus arthrodesis are detailed in Table 8.5.

Primary ORIF is the currently accepted technique for the management of displaced, unstable Lisfranc injuries and is often indicated for treatment of athletes with low-energy injuries, regardless of severity [1, 20]. TMT arthrodesis has been traditionally viewed as a salvage procedure following failure of ORIF [18, 20]. Yet, for various reasons, there has been an increasing trend in arthrodesis as the primary method of fixation [1].

Arthrodesis may be categorized as either complete or partial. Complete arthrodesis consists of fusion across all TMT joints of the foot [18]. However, some argue that loss of motion due to fusion across the medial, middle, and lateral columns of the midfoot would result in a biomechanical deficit [1]. An in vitro study of midfoot biomechanics demonstrated that the three columns of the midfoot vary with respect to inherent motion at each articulation. On average, the lateral column demonstrates approximately 11.1° of motion during supination-pronation while the medial and middle columns only demonstrate 1.5° and 2.6°, respectively [21]. As such, partial arthrodesis is a hybrid fixation-fusion method that attempts to address the column-specific biomechanical differences of the midfoot [2, 18]. Partial arthrodesis may be defined as fusion of the medial and middle columns while the lateral column is either provisionally fixed or left free [18].

8.8.2.1 Open Reduction and Internal Fixation

Surgical Approach

The choice of incision for Lisfranc ORIF is guided by the injury pattern and required exposure (Table 8.6). The authors prefer a dual-incision approach, as it allows access to the

Table 8.6 Surgical approaches to Lisfranc injuries

Incision	Landmark	Approach	Exposure	Dangers
Dorsomedial[a]	Second MT First MT interval	Medial to EHL	1st TMT	Dorsal medial cutaneous nerve (branch of superficial peroneal nerve)
		Between EHL and EHB	First TMT Second TMT Lisfranc ligament	
		Between EHB and second EDL tendon Superficial to dorsalis pedis artery and DPN	Second TMT joint Third TMT joint Lisfranc ligament	Dorsalis pedis artery Deep peroneal nerve
Dorsolateral	Fourth MT Third MT interval	Between EDL and EDB	Third TMT joint Fourth TMT joint Fifth TMT joint	Superficial peroneal nerve branches
Medial	Medial border of first TMT joint	Tibialis anterior tendon insertion	First TMT joint NCJ joint Lisfranc screw Intercuneiform screw Medial plating of first TMT	Dorsal medial cutaneous nerve (branch of superficial peroneal nerve) Tibialis anterior tendon

NCJ Naviculo-cuneiform joint, *DPN* Deep peroneal nerve

[a]Can be extended proximally to access the naviculo-cuneiform joint

medial, middle, and lateral columns of the foot [14, 18].

In the dorsomedial approach to the midfoot, a 15-blade is used to make a 4–5 cm longitudinal incision between the first and second TMT joints on the dorsomedial aspect of the foot [1, 19]. Skin hooks or sens are used to apply gentle traction on the epidermis during dissection. Great care should be taken at the distal most aspect of the incision in order to preserve the integrity of the medial branch of the dorsal medial cutaneous nerve. Following skin exposure, the inferior extensor retinaculum is incised. The exposure continues in the plane between the extensor hallucis longus (EHL) and extensor hallucis brevis (EHB). The EHL tendon sheath is incised dorsally, while the EHL tendon is retracted laterally, and the exposed floor of the EHL tendon sheath is incised. A medial full-thickness flap is created by extending this incision to the medial margin of the first TMT joint. A lateral full-thickness flap is created in a subperiosteal dissection toward the lateral margin of the second TMT joint. The lateral full-thickness flap may be used to protect the adjacent neurovascular bundle throughout the procedure.

A dorsolateral incision can be made to provide access to the third, fourth, and fifth TMT joints, as necessary [1]. For Lisfranc injuries resulting in lateral column instability, our authors use a dorsolateral incision that is parallel over the fourth metatarsal. During incision and dissection, it is important to maintain the integrity of a wide skin bridge between the dorsomedial and dorsolateral incisions in order to avoid necrosis of interarching tissue [18]. After blunt dissection, incision of the inferior extensor retinaculum reveals the underlying extensor digitorum communis (EDC) tendon and the medial margin of the extensor digitorum brevis (EDB) muscle. The EDC and EDB tendons are retracted laterally to expose the third TMT joint capsule. Full-thickness subperiosteal flaps are developed in a similar fashion as the dorsomedial incision, with medial extension toward the lateral aspect of the second TMT joint and lateral extension toward the medial aspect of the fourth TMT joint.

Lastly, a medial incision can be made along the medial utility line to assist with reduction and screw placement across the Lisfranc joint. If indicated, fixation of the intercuneiform joints, the first TMT joint, and the naviculo-cuneiform joint

can also be performed through this incision. Using a 15-blade, a 3-cm longitudinal incision is made on the medial border of the first MT base. Dissection is performed along the fiber lines of the tibialis anterior tendon down to the level of the insertion.

Intraoperative Assessment

Once appropriate exposure has been obtained, the fracture-dislocation is debrided of hematoma and irrigated to allow for further assessment of articular damage and to ensure an anatomic reduction. If more than 50% of the medial and middle column joints show evidence of chondral damage, primary midfoot arthrodesis may be used instead of ORIF. There is significant debate regarding primary arthrodesis of the lateral column given the functional advantages of its inherent mobility.

Reduction

Depending on the specific injury pattern of the TMT joint complex, several reduction techniques may be employed. The first MT joint is generally reduced with a supination-external rotation maneuver relative to the proximal bones of the foot. Distinct crests on the dorsal aspects of the first MT and the medial cuneiform should be aligned as closely as possible. Alignment of these dorsal landmarks can guide accurate reduction of the joint.

A K-wire is passed along the intended path of the trans-articular screw or extra-articular plate, across the first MT and the medial cuneiform or across the second MT and the medial cuneiform, to provide provisional fixation. Temporary reduction was confirmed via intraoperative fluoroscopy [1].

Fixation

Once anatomic reduction has been achieved, a variety of options exist for definitive fixation of Lisfranc injuries. In athletes and patients participating in high impact activities, our authors preference is to use either a traditional technique with trans-articular screws or a joint-sparing approach with dorsal extra-articular plates.

Final fixation is performed in a medial to lateral orientation [1, 2, 19]. Trans-articular screws or a dorsal extra-articular plate may be used for definitive stabilization of the first TMT joint [1, 9, 19]. The first trans-articular screw is placed retrograde, starting at the dorsal crest of the first MT metadiaphysis and aimed plantarly toward the medial naviculo-cuneiform joint. The retrograde screw should be countersunk to avoid violation of the cortex and hardware prominence. A second trans-articular screw is then placed in an antegrade manner. Starting at the dorsal edge of the medial cuneiform along the Chopart joint, the antegrade screw is aimed toward the plantar aspect of the first metatarsal metadiaphysis. If an extra-articular plate is used, it is positioned and fixed in the same manner as the trans-articular screws [1].

Attention is then turned to fixation of the Lisfranc joint. A pointed reduction clamp is used to span the joint, with one tine placed on the medial aspect of the medial cuneiform and the other tine placed on the lateral border of the second MT [1]. Special care should be taken to ensure that there is no dorsal or plantar malreduction. It has been observed that plantar displacement of greater than 2 mm may lead to transfer metatarsalgia. Next, anatomic reduction is confirmed with fluoroscopy. A K-wire is passed along the anticipated path of the fixation, beginning at the medial cortical shelf of the medial cuneiform and angling through the proximal metaphysis of second MT. A common error is to aim too plantarly when performing this step. The second MT serves as the "keystone" in the "roman arch" structure of the midfoot; as such, the K-wire should be aimed slightly more dorsally [2]. A trans-articular screw or an extra-articular plate is placed along the trajectory of the provisional fixation.

The second TMT is provisionally reduced and stabilized with a K-wire. Definitive fixation of the second TMT joint is achieved using a trans-articular screw or an extra-articular plate [19]. If necessary, the third TMT is secured in a similar fashion to that of the second TMT.

If the intercuneiform joints are involved in the injury complex, these are also reduced and fixed to ensure complete stabilization of the Lisfranc joint. A trans-articular screw is passed through

the cuneiforms and is oriented parallel to the Chopart joint [1, 9]. As the intermetatarsal ligaments are often intact between the third, fourth, and fifth metatarsals, reduction may be obtained indirectly, which allows for percutaneous fixation of these joints [19]. At the conclusion of the procedure, final fluoroscopic images are obtained. Radiographs should demonstrate anatomic reduction of the articular surfaces and appropriate placement of the hardware (Figs. 8.3 and 8.4).

Wound Closure

Wounds are copiously irrigated and suction is used to achieve further visualization of the operative field. The dorsomedial incision is closed first. The floor of the EHL tendon sheath and the associated subperiosteal flaps are repaired with deep absorbable suture, 2-0 or 3-0 vicryl. Through the dorsolateral incision, the subperiosteal flaps and the inferior extensor retinaculum are repaired using the same deep absorbable suture, 2-0 or 3-0 vicryl. A layered superficial closure of both incisions is performed next. The subcutaneous tissue is closed using 2-0 or 3-0 absorbable vicryl suture. The skin is closed superficially with 3-0 monofilament suture, monocryl, via a vertical mattress or simple interrupted stitch. If an intercuneiform screw was placed, a simple superficial closure of the medial incision with 3-0 monocryl or 3-0 nylon may be adequate.

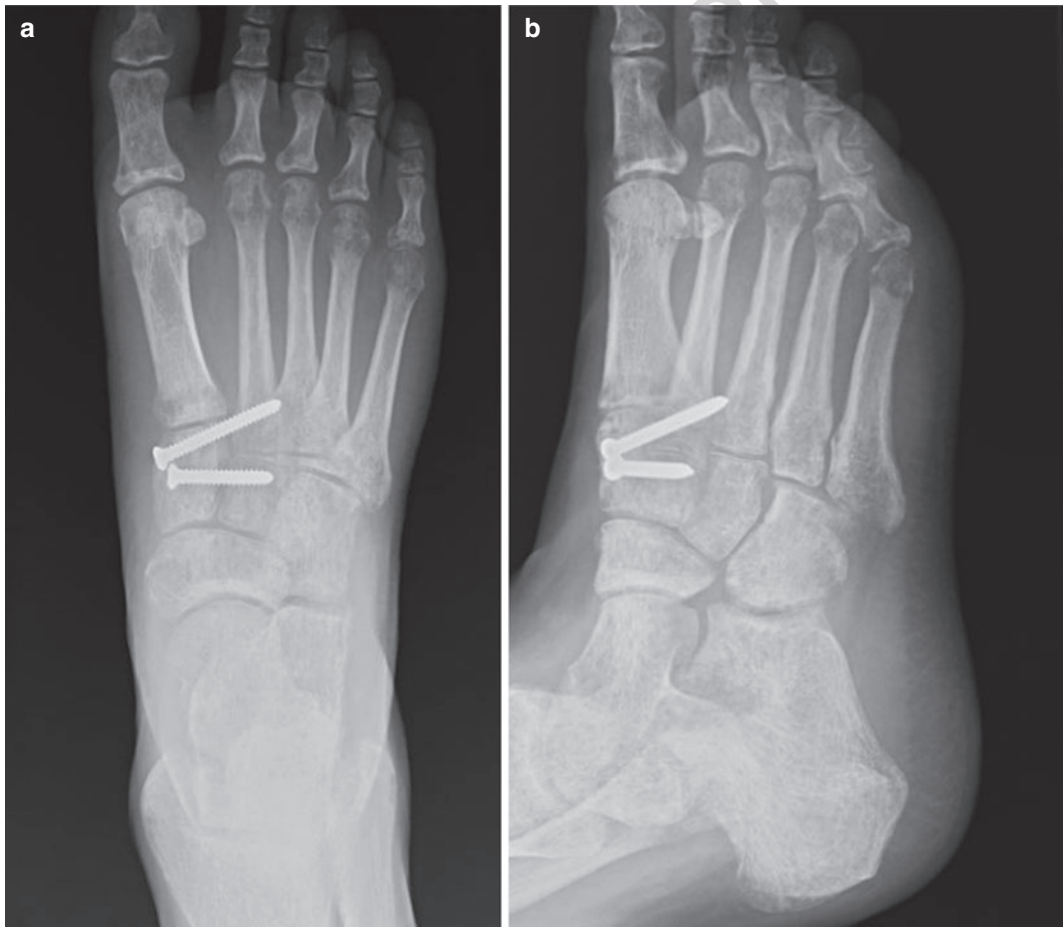

Fig. 8.3 AP (**a**) and oblique (**b**) plain films following open reduction and internal fixation of the Lisfranc joint using trans-articular screws

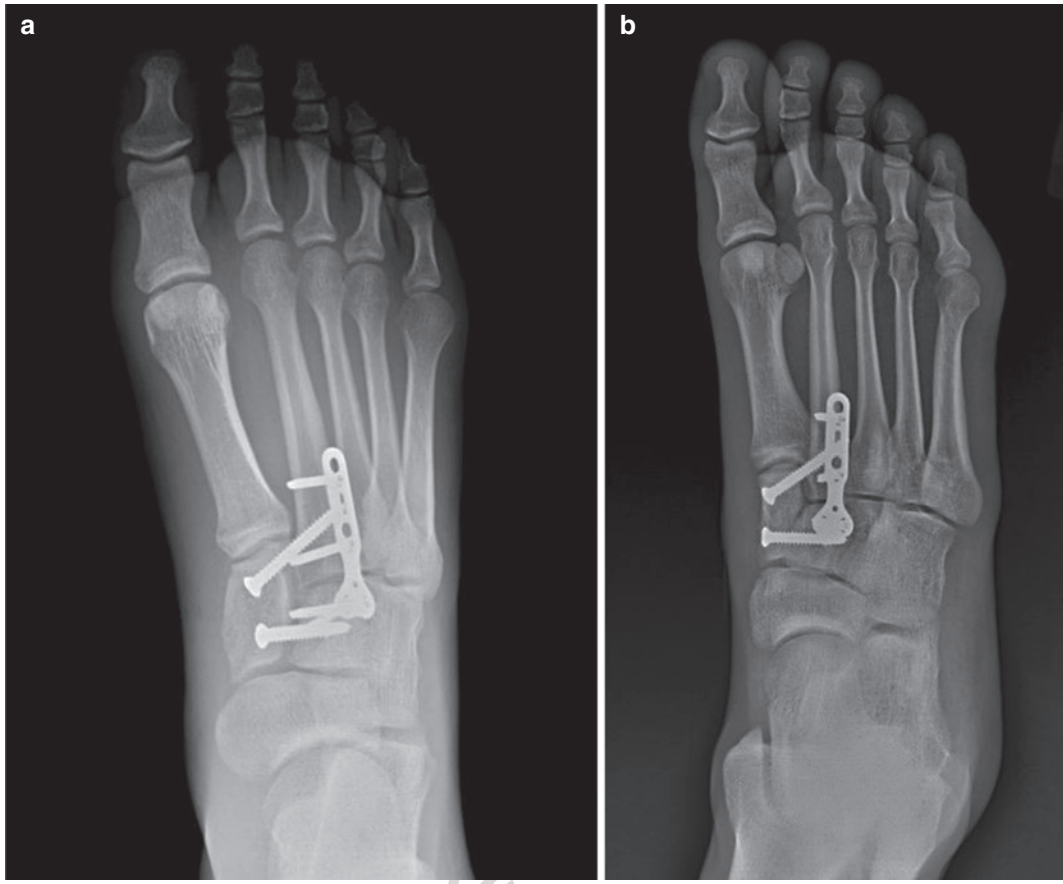

Fig. 8.4 AP (**a**) and oblique (**b**) plain films following open reduction and internal fixation of the Lisfranc joint using a combined technique with trans-articular screws and a dorsal extra-articular plate

8.8.2.2 Primary Arthrodesis

Surgical Approach

The surgical approach for primary arthrodesis is similarly guided by the injury pattern and required exposure. A dual-incision approach is also commonly used for primary arthrodesis in order to access the medial, middle, and lateral columns of the foot.

The dorsomedial, dorsolateral, and medial aspects of the midfoot are incised using the same techniques as described above for ORIF [1].

Intraoperative Assessment

Once appropriate exposure has been obtained, the fracture-dislocation is debrided and irrigated to allow for further assessment of articular damage. Use of a small laminate spreader may allow for better visualization of the involved joint. If more than 50% of the articular surface demonstrates evidence of chondral damage, primary midfoot arthrodesis is indicated. Articular cartilage is removed from the affected joints via controlled movements with a rongeur, osteotome, or curved curette [1]. Special care must be taken to ensure that the subchondral plate is not violated. The exposed subchondral bone can be further perforated in a controlled punctate fashion to allow for cancellous bleeding, which is thought to promote a higher likelihood of fusion. Bone graft from the calcaneus may also be used to promote successful fusion [6, 18].

Reduction

Depending on the injury pattern and number of joints involved, several reduction techniques may be used. The same reductions techniques as those for ORIF may be used during primary arthrodesis.

Alignment should be confirmed using fluoroscopy [6]. A K-wire should be passed along the intended path of the screw to provide provisional fixation.

Fixation

Once anatomic reduction has been achieved, a variety of options exist for definitive fixation of Lisfranc injuries. Fixation is commonly achieved using a solid screw construct across multiple midfoot joints. However, dorsal extra-articular plates may also be used for fusion. Stabilization of the medial column is the recommended first step in fixation as it provides a foundation for subsequent fixation of the lesser metatarsals. Fixation of the medial column is traditionally achieved via placement of a trans-articular screw from the medial cuneiform to the first metatarsal via lag technique or via a dorsal extra-articular plate positioned in the same manner.

Attention is then turned to fixation of the second metatarsal. A pointed reduction clamp is used to ensure anatomic reduction of the second metatarsal into the keystone position. As in ORIF, special care should be taken to ensure that there is no dorsal or plantar malalignment. Anatomic reduction should be confirmed with fluoroscopy. A trans-articular screw or an extra-articular plate is placed from the medial cuneiform to the base of the second metatarsal [1].

If complete arthrodesis is desired, additional trans-articular screws or extra-articular plates may be placed across the remainder of the TMT joint. If partial arthrodesis is desired, percutaneous fixation of the lateral column may be achieved using K-wires. However, depending on the injury pattern, the third-fourth, and fourth-fifth intermetatarsal ligaments may be intact and reduction of the lateral may have been achieved indirectly after fixation of the medial and middle columns. In that case, the lateral column may be left free [18] (Fig. 8.5). Final fluoroscopic images should demonstrate anatomic reduction of the articular surfaces and appropriate placement of all hardware.

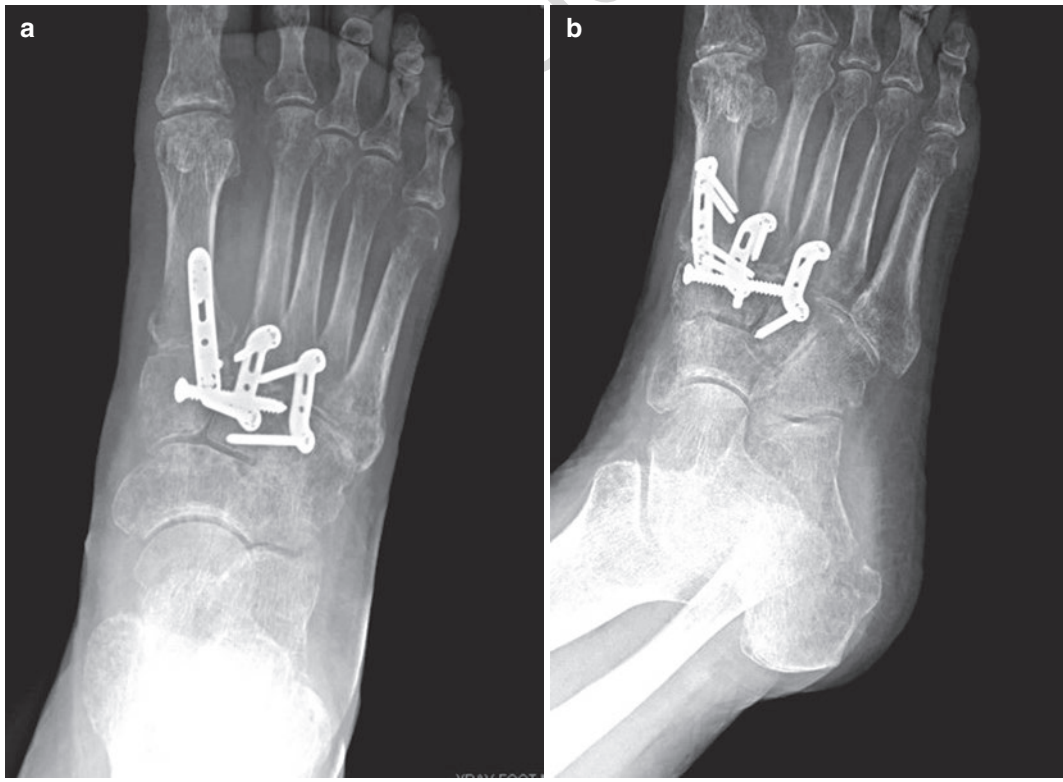

Fig. 8.5 AP (**a**) and oblique (**b**) plain films follwing partial arthrodesis of the Lisfranc joint

Wound Closure

Wounds are copiously irrigated and suctioned for further visualization of the operative field prior to closure. The dorsomedial, dorsolateral, and medial incisions are closed using 2-0 vicryl for the deep closure and 3-0 nylon for superficial closure in the same fashion as detailed above for ORIF.

8.8.3 Postoperative Management

The postoperative timeline is individualized for each patient (Table 8.3). Typically, sterile dressings and a well-padded, bulky posterior short-leg splint are applied in the operating room [19]. Sutures and splint are removed 2 weeks postoperatively. The patient is transitioned into a CAM boot or short-leg cast. If K-wires were used, they are removed around 6 weeks postoperatively [18, 19]. Progressive weight-bearing occurs in a step-wise manner after 6 weeks. Full weight-bearing is not permitted until 10–12 weeks postoperatively, at which point weight-bearing radiographs can be performed. When appropriate, weight-bearing images should confirm maintenance of reduction and appropriate bone healing [1, 3].

Removal of hardware in the postoperative period is highly debated. There is currently no consensus regarding timing, necessity, and role that hardware removal plays in overall patient outcomes [1]. Some surgeons believe that cortical screws involved in medial column fixation should remain implanted indefinitely [19]. Alternatively, other surgeons advocate for routine removal of any and all hardware at 18 weeks to 6 months following the procedure [13, 18–20]. Under the rationale that removal of hardware potentially restores the natural motion of the midfoot, it has been suggested that athletes may benefit from removal of hardware while nonathletes may not [1, 8]. Furthermore, hardware removal among athletes may be influenced by individual weight, such that those >200 pounds should undergo removal of hardware after 24 weeks while those <200 pounds may undergo hardware removal at 12–16 weeks [7, 8].

8.9 Postoperative Complications

The most common complication following operative management of Lisfranc injuries is posttraumatic arthritis, regardless of surgical technique [17]. In a prospective, randomized study, Mulier et al. reported that 94% of patients demonstrated degenerative changes at an average follow-up of 30.1 months. However, surgeons debate whether iatrogenic disruption of the articular surface compounds the pre-existing cartilage damage sustained at the time of injury [3]. Further studies are needed to assess the extent of intraoperative damage during Lisfranc fixation and whether it contributes to the severity of subsequent osteoarthritis [4, 18, 19].

Osteoarthritis is significantly associated with injuries that have not been anatomically reduced at the time of fixation [5, 16, 17]. Adib et al. found that only 35% of patients with anatomic reduction developed osteoarthritis while 80% of those who with nonanatomic reduction developed degenerative changes [17]. However, patients with purely ligamentous Lisfranc injuries demonstrate a higher prevalence of osteoarthritis (40%) compared to combined osseous-ligamentous injuries (18%), despite achieving anatomic reduction [19].

ORIF has also been associated with hardware failure, missed concomitant injuries, deep vein thrombosis, and superficial wound infection as compared to arthrodesis [17]. Persistent pain, midfoot deformity, and symptomatic hardware have also been frequently reported [20, 22]. Primary arthrodesis has also been linked to a greater incidence of pseudoarthrosis, delayed union, and non-union as compared to ORIF [1, 17, 18]. Ly et al. reported specific instances of delayed union and non-union requiring a bone stimulator and revision arthrodesis with bone graft, respectively [20].

8.10 Postoperative Outcomes

Outcomes following Lisfranc injuries are influenced by a variety of factors such as injury pattern, patient-specific demographic factors, diagnostic accuracy, and appropriate manage-

ment. High-energy traumatic mechanisms and concomitant injuries demonstrate worse outcomes compared to low-energy mechanisms and isolated injuries [13]. Delayed diagnosis and prolonged time to treatment is associated with persistent pain, functional disability, progressive post-traumatic osteoarthritis, and need for salvage arthrodesis [6].

Outcomes following postoperative management also vary based on the surgical technique employed. Following ORIF, fixation of the affected Lisfranc joint in anatomic reduction is an essential factor in determining long-term prognosis [19]. Increased average width between the first and second metatarsal base after ORIF has been associated with worse outcomes among patients with severe Lisfranc injuries [18]. As such, maintenance of accurate reduction is of equal importance, regardless of the severity of injury [2, 11]. Fortunately, anatomic reduction of the Lisfranc joint following rigid fixation appears to be well maintained over the long term. Henning et al. reported that 100% of patients who underwent Lisfranc ORIF maintained anatomic reduction at 2-years follow-up [1]. When anatomic reduction of the midfoot is both achieved and maintained, normal dynamic walking patterns may be restored in the injured foot [22].

Restoration of adequate midfoot function following ORIF has been frequently demonstrated. In a study of patients with radiographically confirmed anatomic reduction, there was a mean American Orthopaedic Foot and Ankle Score (AOFAS) of 78.3 at 42.6 months follow-up [13]. Similarly, Kuo et al. found positive postoperative outcomes, reporting a mean midfoot AOFAS of 77 and a mean Musculoskeletal Function Assessment (MFA) Score of 19 at an average follow-up of 52 months [19]. Patient-reported outcomes following ORIF of Lisfranc injuries also demonstrate positive results. Arntz et al. document that greater than 90% of patients report excellent or satisfactory outcomes following ORIF of the Lisfranc joint using a standard AO technique [3, 17].

While ORIF of Lisfranc injuries generally demonstrates favorable outcomes, the technique often requires second surgery for removal of hardware, whether due to patient dissatisfaction or surgeon preference. Kuo et al. reported that 50% of patients underwent subsequent arthrodesis at an average time of 12 months from initial ORIF due to persistent pain associated with post-traumatic arthritis [19]. Ly et al. reported that 30% of Lisfranc ORIF patients underwent a second surgery for removal of prominent or painful hardware at an average of 6.75 months postoperatively [20]. In a systematic review of the literature, Sheibani-Rad et al. reported an overall higher rate of reoperation among patients after ORIF (75–79%) compared to arthrodesis (17–20%). However, many of the studies included in the systematic review describe scheduled removal of hardware at specified time intervals following the index procedures; and thus, it is possible that the higher rates of operation may be simply due to study design [17]. Further studies are needed in order to provide evidence-based recommendations regarding the specific implications of hardware removal on patient outcomes following ORIF.

Like ORIF, arthrodesis has also demonstrated favorable outcomes. In a study conducted by Henning et al., 94% of patients who underwent primary arthrodesis of the Lisfranc joint maintained anatomic reduction and achieved solid fusion at 2-year follow-up [1]. Due to the high rates of success and nature of the technique, arthrodesis rarely requires additional surgery for hardware removal or revision [1, 20].

Primary arthrodesis appears to have particularly favorable functional outcomes with respect to operative management of purely ligamentous Lisfranc injuries. Ly et al. report significantly higher mean midfoot AOFAS at 2-year follow-up among patients with purely ligamentous Lisfranc injuries who underwent primary arthrodesis compared to those who underwent ORIF, 88 and 68.6, respectively [20]. Purely ligamentous Lisfranc injuries have also shown favorable patient-reported outcomes following primary arthrodesis. Patients with ligamentous injuries reported a return to 92% of their pre-injury level at 2 years following primary arthrodesis. At 2 years, patients also reported an average Visual Analog Pain Scale (VAPS) score of 1.2 compared to an

average VAPS score of 4.2 among open reduction patients [20].

Both ORIF and arthrodesis are reasonable primary surgical interventions for Lisfranc injuries, and it appears that most patients may experience positive outcomes regardless of the surgical technique employed [17]. Mulier et al. demonstrated no significant difference in pain, foot function, and cosmesis among patients who underwent either ORIF or partial arthrodesis in which only the first through third TMT joints were fused while the fourth and fifth TMT joints were left free [18]. In a more recent study, Henning et al. similarly found no statistical difference in Short Musculoskeletal Function Assessment (SMFA) scores, Short Form Survey 36 (SF-36) scores, and satisfaction rates between primary ORIF and primary arthrodesis patients at an average follow-up of 53 months [1].

8.11 Conclusion

Injury to the Lisfranc joint is rare and commonly missed or misdiagnosed. These injuries may cause significant damage to the midfoot resulting in disabling morbidity. Thus, timely identification and appropriate treatment of Lisfranc injuries are important. Stable Lisfranc injuries with minimal displacement are amenable to a trial of non-operative management. However, non-operative management in the competitive athlete is recommended with caution, as there is a higher likelihood of treatment failure. Unstable injuries with moderate to severe displacement require prompt surgical management in both the athlete and nonathlete. Although ORIF has been accepted as the standard for operative management, primary arthrodesis has become an increasingly favorable option among surgeons. Arthrodesis appears to have a unique application in that studies cite superior outcomes in purely ligamentous Lisfranc injuries as compared to ORIF. However, both surgical techniques are reasonably controversial in nature. ORIF has been associated with high rates of reoperation due to planned removal of hardware, and primary arthrodesis has been associated with a loss of natural biomechanical function within the midfoot. While anatomic reduction is highly recognized as an essential factor in promoting positive outcomes, there is currently no consensus regarding the ideal operative method for the treatment of Lisfranc injuries.

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Part II

Cartilage

9 Cartilage Techniques for Osteochondral Lesions of the Talus

Eoghan T. Hurley, Yoshiharu Shimozono, and John G. Kennedy

9.1 Introduction

Osteochondral lesions of the talus (OLT) are a common ankle pathology and have been shown to occur in over 65% of chronic ankle sprains and 75% of ankle fractures [1, 2]. OLT can be a significant source of pain and disability and may have a potential to progress to arthritis. Conservative management, including physiotherapy, injections, and a period of non-weight-bearing, may relieve symptoms in the short term, but they often recur due to inadequate healing of the lesion and require surgical treatment.

The surgical management of OLT is largely dependent on the size of the lesion, the occurrence of cysts, and whether the patient has failed previous surgeries. Surgery can be broadly divided into reparative and replacement procedures [3]. Reparative procedures include bone marrow stimulation procedures (BMS) such as microfracture [4]. Replacement procedures include autologous osteochondral transplantation (AOT) and osteochondral allograft transplantation [5]. Autologous chondrocyte implantation (ACI), matrix-induced autologous chondrocyte implantation (MACI), autologous matrix-induced chondrogenesis (AMIC), and scaffolds as adjuncts to surgery have become popular in recent years, but further studies are required to substantiate their widespread use [6]. Biological adjuncts, including platelet-rich plasma (PRP) and concentrated bone marrow aspirate (CBMA), have been shown to have promising evidence and may be utilized alongside surgery to improve healing potential [7].

Despite the advances in the treatment of OLT in the last few years, no gold standard treatment exists and surgical treatment should be individualized to the patient in order to optimize outcomes [8].

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9.2 Microfracture

9.2.1 Indications

Microfracture is a reparative technique, where the subchondral bone in the defects is perforated with awls to release the mesenchymal stem cells and growth factors from bone marrow, leading to the

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formation of fibrous cartilage repair tissue. Microfracture is indicated for smaller lesion which is typically less than 150 mm^2 in area or 15 mm in diameter [9, 10]. However, a recent systematic review by Ramponi et al. demonstrated that microfracture may be optimal for lesions smaller than 107.4 mm^2 in area and/or 10.2 mm in diameter [11]. Ankle stability, joint alignment, lesion size, the presence of a cyst, previous cartilage repair procedure, and uncontained lesion are all prognostic factors when performing microfracture [9, 10]. There are several disadvantages with microfracture, including the quality of fibrocartilage which is inferior to native hyaline cartilage, permanent damage to the subchondral bone, and deterioration of the fibrocartilage over time [12].

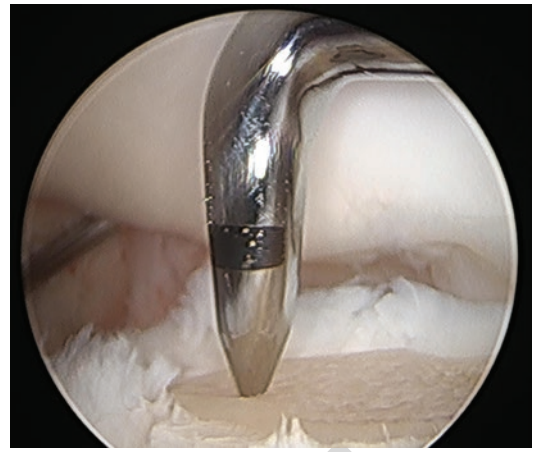

Fig. 9.1 Arthroscopic image of the microfracture awl penetrating the subchondral bone plate

9.2.2 Technique

Microfracture is typically performed arthroscopically using anteromedial and anterolateral portals. After inspection of the ankle joint, the OLT is prepared prior by debriding all unstable cartilage by shaving or curettage until there is a stable rim of articular cartilage. The calcified cartilage layer of bone should be removed; however, care should be taken not to disrupt the subchondral bone excessively.

Once the defect site is prepared, an awl <1 mm is used to perforate the subchondral bone. A smaller awl may result in less damage to the subchondral bone and may be preferable. Additionally, the distance between the awl apertures should be 3–4 mm apart to minimize damage to the subchondral bone (Fig. 9.1). After the holes have been created, the tourniquet should be turned off to assess for bleeding and fat droplet extrusion. Biological adjuvants, including PRP or CBMA, may be added, which may improve fibrocartilage repair tissue.

9.2.3 Outcomes

Microfracture has been shown to result in favorable short-term outcomes in several systematic reviews, with typically >85% of patients resulting in good to excellent clinical outcomes [8, 13]. In regard to return to play sports following microfracture, Hurley et al. found in a systematic review that 86.8% of patients returned to sport at previous levels, with a mean return at 4.5 months [14].

Despite successful outcomes in the short to mid-term, there is a concern about deterioration of the fibrocartilage repair tissue over time, which may potentially affect the clinical outcomes in the longer term [12, 15, 16]. Ferkel et al. found deterioration of clinical scores in up to 35% of patients within 5 years following BMS [12]. Lee et al. found that only 30% of patients who underwent BMS showed lesion integration at second look arthroscopy at 12 months postoperatively [17]. In addition, van Bergen et al. reported that one-third of patients progressed ankle arthritis by one grade on plain radiographs at a mean follow-up of 141 months [18].

Recent studies have focused greater attention on the subchondral bone, which provides significant joint loading [15, 19]. Seow et al. found in a systematic review that there was permanent alteration of the subchondral bone following BMS in preclinical studies [15]. This subchondral bone alteration will reduce its mechanical support and may contribute to fibrocartilage deterioration. Therefore, techniques minimizing damage to the subchondral bone will be important for cartilage longevity. In a translation animal model Orth et al. found that the use of small-diameter awls

offers better articular cartilage repair than large-diameter awls on histological exam [20]. Gianakos et al. evaluated different microfracture awl sizes in a cadaver talus model, and found that smaller awl sizes may help diminish the amount of subchondral bone microarchitectural disturbances [21]. Additionally, biologics may play a role in reducing the deterioration of the fibrocartilage, although the long-term evidence on this is still limited.

9.2.4 Particulated Juvenile Cartilage Allograft

PCA (DeNovo NT; Zimmer Biomet, Inc.) is a scaffold containing juvenile chondrocytes and particulated juvenile cartilage, typically harvested from donors less than 3 years old. PCA is theoretically advantageous as an adjunct to microfracture, as their high metabolic activity level and differential gene expression may have the potential to reproduce more hyaline cartilage than adult chondrocytes (Fig. 9.2).

The supporting evidence for PCA is limited; however several in vitro studies have found PCA has a superior chondrogenic potential to adult cartilage [19]. These studies showed improvement in histological, biochemical, and biomechanical analyses, but not in gene expression [19]. Karnovsky et al. performed a retrospective comparative study of the results of patients treated with microfracture and PCA, and those treated with microfracture alone, at a mean follow-up of 30 months [22]. The authors found both groups still showed fibrocartilaginous growth that did not appear normal on MRI, and there was no difference in functional outcomes between the two groups. The current role of PCA remains unclear, and further long-term high-level studies are needed.

9.2.5 Micronized Cartilage Allograft

MCA (BioCartilage; Arthrex, Inc) contains an allogeneic extracellular matrix, including type II collagen, proteoglycans, and cartilaginous growth factors. MCA is theoretically advantageous as an adjunct to microfracture, by inciting the migration of stem cells to the defect site of the defect, while MCAs facilitate chondrogenesis by acting as a tissue network promoting cell interaction (Fig. 9.3).

The evidence supporting MCA is still limited, although the results of early literature appear promising. Fortier et al. found that alongside microfracture, MCA with PRP improved the quality of cartilage repair tissue compared to

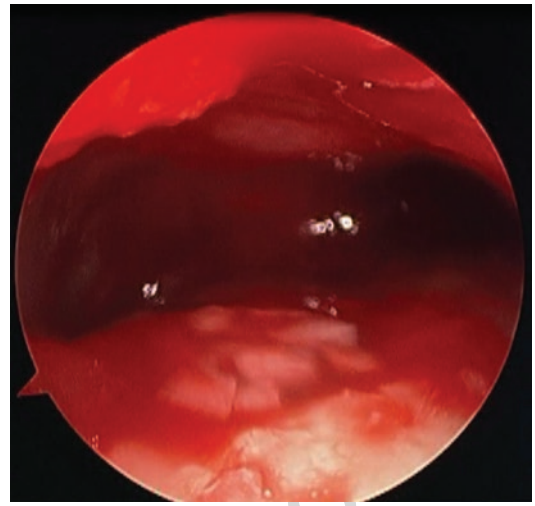

Fig. 9.2 PCA application into the defect, mixed with CBMA or PRP

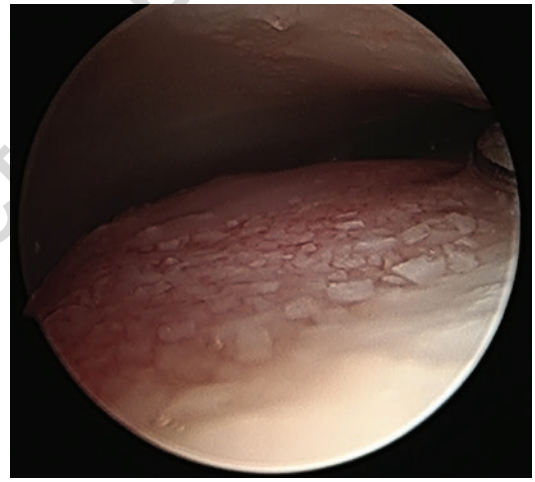

Fig. 9.3 MCA application into the defect, mixed with CBMA or PRP

microfracture alone in an equine model [23]. Desai et al. reported on the results of nine patients treated with microfracture and MCA at a mean follow-up of 12 months [24]. Seven patients had excellent outcomes, and two patients reported good outcomes, although no quantitative outcome measures were noted. However, no comparative studies comparing MCA with microfracture to microfracture alone have been reported. Therefore, long-term high-level studies are warranted to justify its current widespread use [19].

9.3 Autologous Osteochondral Transplantation

9.3.1 Indications

AOT is a cartilage replacement technique where a graft is harvested from the host, and transferred into a prepared site at the defect in the talus. As AOT replaces the local subchondral bone, it may result in the restoration of the native biological environment leading to improved functional outcomes and survivorship over BMS. It is typically indicated in primary cystic lesions, lesions >10 or 100 mm^2, and revision procedures following a failed primary procedure [11, 25–27]. A recent systematic review by Ramponi et al. found that AOT is indicated in lesions greater than 107.4 mm^2 in area and/or 10.2 mm in diameter [25]. Lesion containment, the requirement greater than two grafts, previous BMS, and body mass index can be prognostic factors when performing an AOT [25, 28–30]. There are several disadvantages to AOT, including donor site morbidity, the possible need for an osteotomy to approach the lesions, and differences in cartilage biology/mechanics between the host and graft tissues.

9.3.2 Technique

The OLT may be accessed by a medial or lateral osteotomy depending on the location of the lesion. In the case of a medial OLT, a medial malleolar osteotomy may be utilized to adequately visualize the lesion (Fig. 9.4). A Chevron osteotomy is preferred for this approach as it provides appropriate alignment, stability, a large surface area for healing, and greater visualization [5]. However, an anteromedial lesion may only require a standard arthrotomy for visualization. Anterolateral lesions may be exposed via standard arthrotomy of the ankle joint, although if it is in a central or posterior position an anterolateral tibial osteotomy may be required. After the lesion is visualized, a trephine is utilized to remove the damaged cartilage and underlying bone at the recipient site. A depth of 12–15 mm is the optimal depth to drill the lesion site.

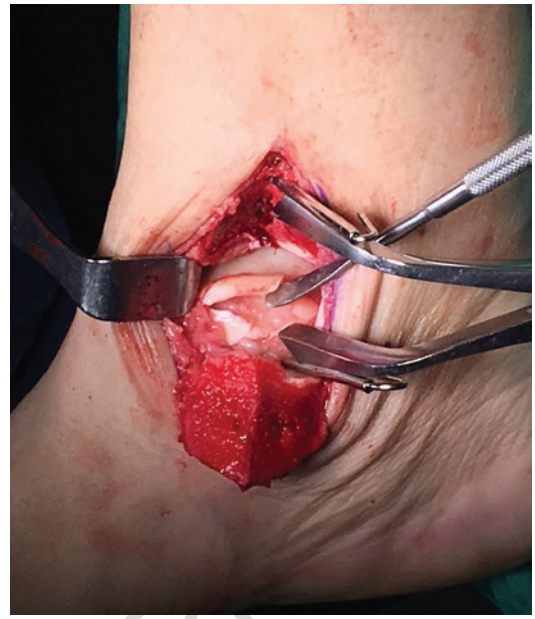

Fig. 9.4 A medial malleolar osteotomy utilized to adequately visualize the lesion

Multiple donor sites exist for graft harvesting; however, our preferred technique is to harvest from a non-weight-bearing portion of the ipsilateral femoral condyle. This site is utilized as it is technically undemanding to access and the variation in topography closely matches the talar dome. It also has a large surface area, allowing for at least three grafts to be harvested without compromising the patellofemoral articulation. Additionally, the superior aspect of the lateral femoral condyle experiences less pressure than other articular surfaces. There is a low incidence

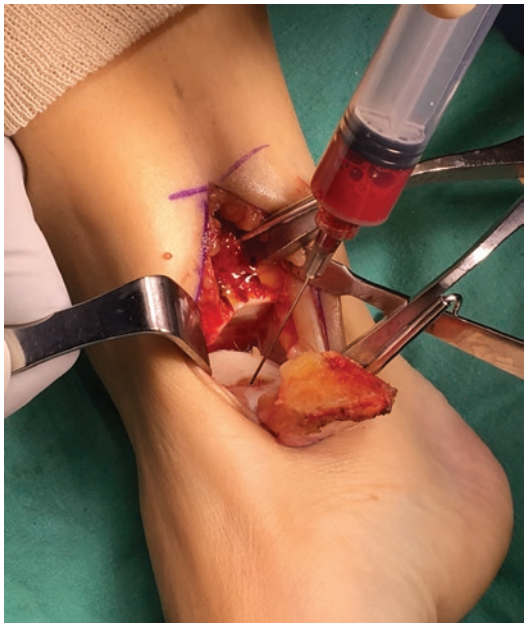

Fig. 9.5 Application of PRP or CBMA into the defect site

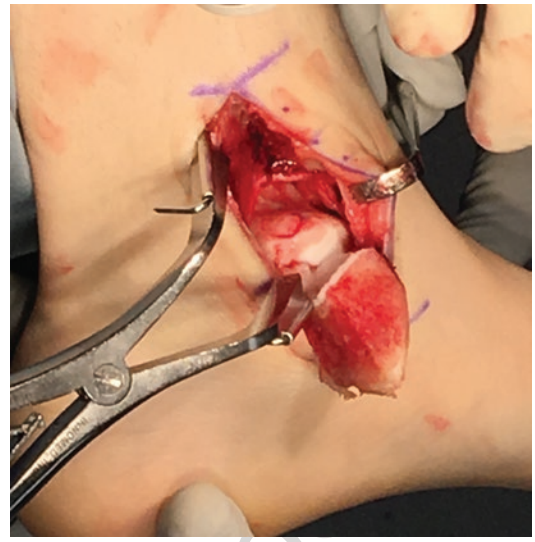

Fig. 9.6 The osteochondral graft transplant being placed into the created recipient site

of donor site complications, typically less than 5% in large series [5, 31–33]. Larger lesions may require two grafts, which should be "nested" next to each other to reduce risk of fibrocartilage formation and synovial fluid inflow between the grafts [5, 34].

Prior to graft implantation, biological adjuvants, including PRP or CBMA, are added, which may facilitate biological integration of graft and host interface (Fig. 9.5). The AOT plug is then transferred to the prepared recipient site. Congruency of the implanted graft is essential as the final graft position should be as flush as possible to match the surrounding cartilage, and care should be taken during surgery to achieve an articular surface as closely as possible to the native talus (Fig. 9.6) [35].

9.3.3 Outcomes

The clinical outcomes following AOT have been shown to be excellent in multiple studies, and a recent systematic review by Shimozono et al. found 87% of patients had good to excellent outcomes at mid-term follow-up [33]. Fraser et al. found that in athletes, 90% of professional athletes and 87% of recreational athletes were able to fully return to pre-injury activity levels at a mean of 24 months follow-up [36]. However, Paul et al. showed patients engaging in high-impact and contact sports required partial modification of sporting activities and a reduced level of participation [29]. Additionally, several studies have shown improved radiological outcomes following AOT, with a low incidence of joint space narrowing [33]. There is still lack of evidence regarding the long-term outcomes of AOT for OLT.

Complications remain a concern with AOT; Shimozono et al. found in a systematic review that 10.6% of patients had complications, with the most common being donor site morbidity [33]. Yoon et al. found that while 9% patients had early donor site morbidity all of these resolved at 48 months follow-up, and Fraser et al. found an early donor site morbidity of 12.5% but this decreased to 5% at a mean of 41-month follow-up [27, 37]. Shimozono et al. found that the overall rate of reoperations was 6.2%; however, only 1% of patients were considered a clinical failure at mid-term follow-up [33]. The osteotomy may be a concern for some surgeons; however, studies have found minimal morbidity when performing

an osteotomy to access the talar dome [17, 38]. Lamb et al. found that in 62 patients a chevron-type medial malleolar osteotomy provided satisfactory healing on T2 mapping MRI and only four patients reported some pain postoperatively [39]. Additionally, postoperative cysts have been shown to occur in up to 65% of patients following AOT, although the clinical significance of this remains unclear. Savage-Elliott et al. found that clinical influence of postoperative cyst formation was not significant in the short term [40]. Finally, the congruency of the graft is paramount to restore contact mechanics in the ankle [35]. Fansa et al. found that implantation of the osteochondral graft in the most congruent position possible restored the force, mean pressure, and peak pressure on the medial region of the talus comparable to intact levels [35].

9.4 Osteochondral Allograft Transplantation

9.4.1 Indications

Osteochondral allograft transplantation is a cartilage replacement technique similar to AOT in which the graft is harvested from a cadaver. There are two types of osteochondral allograft: bulk type and cylindrical plug type. Bulk allograft is generally considered as a salvage surgery if previous surgeries fail, but can be performed as a first-line procedure for excessive large lesions whose successful outcomes cannot be expected by other procedures. Osteochondral allograft transplantation using cylindrical plug has similar indications to AOT, but is usually indicated in preference to AOT in knee osteoarthritis, history of knee infection, and patients concerned with donor site morbidity in the knee. Patient counseling is important in deciding on autograft or allograft, and the pros/cons must be discussed with the patient. There are several disadvantages to allograft, including potential higher failure rate, increased cost, disease transmission, and differences in immunology/cartilage biology between the host and cadaveric tissues [41, 42].

9.4.2 Technique

The recipient site for osteochondral allograft transplantation may be accessed and prepared in a similar manner to AOT. However, bulk allograft may require an anterior approach in the majority of cases. Additionally, bulk allograft may require more extensive preoperative imaging utilizing 3D-CT planning to accurately determine the sizing of the graft needed.

AOT can be harvested from either cadaveric knees or ankles, and there is no consensus over which is the optimal site. Cadaveric talus may be preferable as the cartilage biology, tissue mechanics, and topography may more closely match the recipient site. Fresh nonfrozen allografts less than 28 days old may be preferable to maintain chondrocyte viability, as less than 70% chondrocyte viability is associated with poor outcomes and osteochondral allograft transplantation loses approximately 30% viability at 28 days [43, 44]. Prior to graft, biological adjuvants, including PRP or CBMA, can be utilized, as Oladeji et al. have found that utilizing CBMA in allograft improves radiographic integration [45]. The osteochondral allograft transplantation should be placed in a manner as congruent as possible to AOT, in order to as closely match the local biomechanics and of the local joint. Additionally, bulk allograft requires screw fixation in order to secure the graft, and in this instance a headless screw is preferable.

9.4.3 Outcomes

Studies have found mixed clinical outcomes following osteochondral allograft transplantation for OLT. The results of osteochondral allograft transplantation differ whether it is bulk or cylindrical plug allograft, as bulk allograft may experience poorer outcomes due to larger size of the lesions treated. VanTienderen et al. found in a systematic review of 91 OLTs treated with bulk allograft that at a mean of 45 months follow-up the average AOFAS score improved from 48 to 80 and the mean VAS score improved from 7.1 to

2.7 [42]. Raikin et al. found in 15 patients treated with bulk allograft at a mean of 54 months that the mean VAS score improved from 8.5 to 3.3 and the mean AOFAS score improved from 38 to 83, with 11 patients reporting good/excellent results [46]. However, two patients required conversion to arthrodesis [46]. On plain radiographs, some evidence of collapse or resorption of the graft was found in 67% of patients [46]. El-Rashidy et al. showed using cylindrical plug allograft in the treatment of OLT significantly improved clinical outcomes at a mean follow-up of 3 years, although there was a 10.4% failure rate over this time [47]. Ahmad et al. found similar clinical outcomes following cylindrical plug allograft and autograft for OLT at 35.2 months [48]. However, 18.8% of patients in allograft group required revision surgery due to non-union at the graft/host integration site.

Complications including failure and reoperations remain a concern with osteochondral allograft transplantation. VanTienderen et al. found in their systematic review that 13.2% of patients were considered clinical failures and 25% required reoperation [42]. The cause of the early failure is likely a combination of chondral wear, chondral fissuring, and cyst formation in the graft's subchondral bone, due to poor graft/host bone incorporation. Additionally, differences in the cellular biology between the graft/host and the chondrocyte viability may be a cause for the higher failure rates. Neovascularization may also play a role in the failure of allograft, as Neri et al. found that only 10 out of 15 osteochondral allografts showed gene expression matching the recipient, indicating blood supply between the graft/host interface [41].

9.5 Autologous Chondrocyte Implantation

9.5.1 Indications

ACI is a two-step cartilage reparative technique where autologous chondrocytes are harvested from a non-weight-bearing area and culture expanded in vitro. ACI is then placed into a prepared site at the defect in the talus and covered in an autologous periosteal membrane. The aim of this procedure is to regenerate damaged cartilage with hyaline-like tissue. ACI is indicated in larger lesions or revision procedures following a failed primary procedure. There are several disadvantages to ACI, including two steps to the procedure, cost, and potential failure rates.

9.5.2 Technique

ACI is a two-step procedure, whereby in the first step chondrocytes are harvested from the ankle, the osteochondral fragment itself, or the ipsilateral knee [49]. These cells are then expanded and cultured in vitro for 2–3 weeks.

Once the cells are prepared, the patient returns for the second step where the chondrocytes are implanted, either arthroscopically or via an open incision. The OLT recipient site is first prepared, where it is debrided to the subchondral bone and any cysts present are removed. In larger subchondral cystic defects, a "sandwich" technique can be utilized. This is where after cyst debridement, the autologous bone graft obtained is placed into the defect creating a smaller defect, followed by placement of a periosteal patch. The periosteal patch is taken from the distal or proximal tibia and is made 1–2 mm larger than the defect to account for shrinkage. The patch is then secured over the defect, cambium side down, with sutures and fibrin glue.

9.5.3 Outcomes

ACI has been shown to result in good clinical outcomes, and a recent systematic review by Niemeyer et al. found a clinical success rate of 89.9% in 213 patients at a mean follow-up of 32 months [6]. Giannini et al. reported on the clinical and MRI outcomes of ten patients following ACI for OLT at 10-year follow-up [50]. The authors showed in patients with a mean

lesion size of 3.1 cm^2 treated with ACI at a mean follow-up of 119 months that the AOFAS score improved from 37.9 preoperatively to 92.7 postoperatively with well-modeled restoration of the articular surface on MRI. Additionally, Giannini et al. found that in 46 patients at a mean follow-up of 87.2 months there were only three failures [51]. Battaglia et al. evaluated 20 patients following ACI at a mean follow-up of 5 years and found that, on MRI evaluation, all patients showed a T2 mapping value consistent with normal hyaline cartilage [52].

ACI has a low rate of complications specific to the procedure, and most complications are those associated with ankle arthroscopy or osteotomy, particularly non-union, scar tissue formation and nerve damage as this is a two-stage procedure. However, there is a concern of periosteal hypertrophy due to overgrowth of the repair tissue, which may require debridement.

9.6 Scaffolds

9.6.1 Matrix-Induced Autologous Chondrocyte Implantation

Matrix-induced autologous chondrocyte implantation (MACI) is where a biodegradable polymer scaffolds embedded with chondrocytes is utilized as a scaffold. MACI is a third generation version of ACI and a two-step procedure. However, it is advantageous as it is a self-adherent scaffold, and avoids complications related to the graft harvest.

Aurich et al. reported on the results of 19 patients treated with MACI and observed improvement of the AOFAS score from 58.6 to 80.4 at a final follow-up of 24 months [53]. Additionally, they found 81% of patients returned to play sports after MACI for OLT, including 56% returning to their pre-injury level. Similarly, Magnan et al. showed improvement in the mean AOFAS score from 36.9 to 83.9 in 36 patients, with 18 returning to sport within 2 months [54].

9.6.2 Autologous Matrix-Induced Chondrogenesis

Autologous matrix-induced chondrogenesis (AMIC) is where a porcine collagen I/III matrix is utilized at the site of the defect following microfracture and is a one-step procedure. The supporting theory is that this porcine collagen matrix supports the growth of cartilage following microfracture.

The literature on AMIC is limited to a few small case series, but the results seem promising. Valderrabano et al. reported in a series of 26 patients that 84% of patients had normal/near normal signal intensity of the repair tissue compared with the native cartilage on MRI [55]. However, Wiewiorski et al. observed a significant difference in T1 relaxation times between AMIC repair tissue and the surrounding cartilage, suggesting lower glycosaminoglycan content in the repair tissue [56].

9.6.3 Bone Marrow-Derived Cell Transplantation

Bone marrow-derived cell transplantation (BMDCT) is a combination of CBMA and scaffold material and is a one-step procedure. BMDCT is theoretically beneficial as the mesenchymal stem cells and the growth factors in CBMA support the scaffold in chondrogenesis, to develop hyaline-like cartilage at the site of the defect.

Similar to AMIC, the clinical evidence supporting the use of BMDCT is limited albeit promising. Vannini et al. reported on 140 athletes treated with BMDCT at a mean of 48 months follow-up and found the overall mean AOFAS score improved from 58.7 to 90.9 [57]. The authors also showed that 72.8% of athletes were able to return to pre-injury level of sports. Buda et al. evaluated 80 patients treated with ACI or BMDCT at 48 months follow-up [58]. There was no significant difference in clinical outcomes, but the rate of return to sports was

slightly higher with BMDCT, although the difference was not statistically significant. However, this shows that BMDCT may be a viable alternative to ACI, with the advantage of being a one-stage procedure.

9.7 Biologics

9.7.1 Platelet-Rich Plasma

PRP may be considered as adjuncts to surgical therapies in the treatment of OLT to improve the local healing potential. PRP is an autologous blood product that contains at least twice the concentration of platelets above the baseline value, or >1.1 × 10^6 platelets/μl. PRP contains an increased number of growth factors and bioactive cytokines, including transforming growth factor, vascular endothelial growth factor, fibroblast growth factor, and platelet-derived growth factor [59]. PRP is harvested by drawing venous blood from a peripheral site, and then is put in a preparation kit where it is spun to formulate PRP. This may be performed in either the office or in the operating room.

There is strong basic science evidence to support the use of PRP in cartilage repair. Smyth et al. performed a systematic review and found that 18 of 21 (85.7%) basic science literature studies reported positive effects of PRP on cartilage repair, establishing a proof of concept [7]. Smyth et al. also showed that the application of PRP at the time of AOT implantation in a rabbit model improved the integration of the osteochondral graft at the cartilage interface and decreased graft degeneration [60]. Similarly, Boayke et al. found using PRP alongside AOT in a rabbit model that there was increased TGF-β1 expression at the graft/host interface compared to saline-treated controls, and thus PRP may play a chondrogenic role [61].

Several randomized controlled trials have shown a benefit of PRP in the treatment of OLT and ankle osteoarthritis. Guney found PRP at the time of surgery improved the AOFAS scores and pain-related scores of BMS in the treatment of OLT compared to a placebo control [62]. Additionally, Gormeli et al. and Mei-Dan et al. both found that PRP improved the clinical outcomes and pain scores of patients with ankle osteoarthritis compared to hyaluronic acid in the short term [63, 64].

9.7.2 Concentrated Bone Marrow Aspirate

CBMA may be considered as adjuncts to surgical therapies in the treatment of OLT to improve the local healing potential in a similar manner to PRP. CBMA is an autologous blood product harvested from the long bones, typically the iliac crest or the tibia. CBMA contains a similar growth factor and cytokine profile compared to PRP, with the addition of interleukin 1 receptor antagonist protein in CBMA, which is a potent anti-inflammatory agent [65]. CBMA may be harvested in either the office or in the operating room. However, as CBMA harvest can be painful and may be difficult to perform in the office, we typically only harvest this in the operating room.

Fortier et al. have shown that CBMA improves both the histological and radiological outcomes in the repair of cartilage defects in an equine microfracture model, compared to a control without CBMA [66]. Fortier et al. found increased fill of defect and improved integration of repair tissue with surrounding cartilage [66]. In addition, Saw et al. found in a goat model that CBMA and hyaluronic acid (HA) improved defect coverage and repair tissue following BMS compared to HA alone [67].

Hannon et al. found patients who underwent BMS with CBMA in the treatment of OLT had comparably good mid-term clinical outcomes, but improved MOCART scores compared to BMS alone [68]. While the clinical evidence is limited in the use of CBMA in the treatment of OLTs, Chahla et al. performed a systematic review and showed CBMA was a promising treatment in the treatment of osteochondral defects in the knee [69].

9.8 Summary/Conclusion

The surgical management of OLT remains controversial. Based on the current available clinical evidence, both reparative and replacement procedures have a role in the surgical treatment of OLT and have been shown to result in good clinical outcomes. MACI, which is a next-generation technique of ACI, has become increasingly utilized in recent years. Additionally, biological adjuncts and scaffolds have increasingly gathered attention and provided promising clinical results. However, further high-level studies are still needed to develop standardized clinical guidelines for the treatment of OLT.

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10 Tissue Engineering for the Cartilage Repair of the Ankle

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10.1 Introduction

The cartilage in the ankle is a highly specialized tissue, known to be unique both in biology and anatomy, thinner than in the knee, but with a higher cell density, metabolic activity and more resistance to chronic inflammation [1]. For these reasons the ankle joint is, although often involved in sports injuries, less prone to osteoarthritic progression than other joints, and many osteochondral lesions remain clinically silent. However, larger osteochondral lesions and osteochondritis dissecans of the talus can rapidly develop unstable joint fragments, cyst formation and deterioration of the subchondral bone leading to deformation and collapse of the talus. Young, active people constitute the majority of the patients developing postresidual pain after either acute sprain or repetitive trauma that is why it is crucial that the chosen treatment method has good long-term functional outcomes. There are many treatment possibilities for osteochondral lesions (OCLs) of the talus; nevertheless a gold standard is yet to be established [2]. A systematic review by Verhagen et al. has shown that nonsurgical treatment of OLCs of the talus seems to be successful in only 45% of the cases and for that reason it is not advised [3]. Microfracture has been considered a primary line of treatment in the majority of lesions, and even though short-term results have been promising, some long-term follow-up studies have shown fair and poor results from 47.7% up to 54% [4, 5]. What is more, in our randomized study comparing microfracture, chondroplasty and osteochondral autograft transplantation, we have seen an incomplete healing on a control MRI 12 months after microfracture [6]. Ferkel et al. reported that the promising clinical outcome after microfracture deteriorated in 35% of the treated patients over a period of 5 years [7]. The primary reason of long-term failure may be the poor biomechanics and biological quality of subsequently forming fibrous cartilage, rich in type I collagen. The autologous chondrocyte implantation (ACI) was the next step in the development of osteochondral lesion treatment, and it has demonstrated good clinical outcomes [8–10]. However, the procedure has been considered demanding and required two surgeries. Evolution of tissue engineering and biomaterial science provided a substrate for the development of different scaffolds for cartilage repair. Firstly, used with chondrocytes that were seeded onto the matrix, still that did not

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eliminate the need for chondrocyte harvest and cultivation. Subsequently, a need for a "biological solution to a biological problem" idea has led to the use of bone marrow aspirate concentrate (BMAC) and a hyaluronic acid-based scaffold (HA) in a one-step procedure [11].

10.2 Scaffolds for Tissue Engineering

Scaffolds are designed to host and support the cells used for cartilage regeneration. Materials used in matrices development are either naturally occurring (i.e. hyaluronan, collagen, chitosan) or synthetic (i.e. polystyrene, polylactic acid) [12]. The physical structure and the macro- and micro-architecture also vary, and liquid scaffolds entrap the cells, whereas a multilayered fibre or mesh supports implanted cells allowing their adherence [13, 14]. There are crucial criteria that characterize a good scaffold [14]. Firstly, the material must be biocompatible, and the scaffold itself and the breakdown products should not create an immune response. Secondly, the sufficient porosity of the material is important, so that it allows the cells ingrowth. Finally, the mechanical resistance to shear forces acting in the joint and scaffold stability are of great importance. Among the natural and synthetic materials that have been investigated, only a few have been used in ankle lesions.

The hyaluronan-based scaffolds are entirely based on the benzylic ester of hyaluronic acid, which is a natural glycosaminoglycan, widely distributed in connective tissues. Because of its molecular structure and multifunctional activity, it has proven to be an ideal material for tissue engineering. The network of 15–20-μm-thick fibres forms a scaffold that provides a good support that allows contact of seeded cells, subsequent cluster formation and extracellular matrix deposition. Good clinical results have been achieved in a two-step procedure using the matrix-induced autologous chondrocyte implantation technique and the use of a hyaluronic acid-based scaffold [15–17], as well as in a one-step procedure with the use of BMAC (Hyalofast, Anika Therapeutics Inc., Massachusetts, USA) [11]. Another type of scaffold used in treatment of OCL of the talus consists of collagen I and III and is a bilayer matrix that has been used in first-generation ACI and in combination with microfracture providing a good outcome [18, 19]. A scaffold used in treatment of OCL that varies in structure from collagen- and hyaluronan-based scaffolds mimics the trilateral morphology of the osteochondral unit. The superficial layer is made of type I collagen, while the lower layer consists mainly of magnesium-enriched hydroxyapatite. Although presenting clinical improvement in the treatment of OCL in the talus, it has shown limited tissue regeneration [20, 21].

10.3 Bone Marrow in Cartilage Repair

Using BMAC for cartilage regeneration is a valuable technique, offering a chance to avoid two surgeries and expensive chondrocyte cultivation. BMAC has proven to be a good material for cell-based therapy in cartilage regeneration with a potential to differentiate into osteogenic and chondrogenic cells [22–24]. Moreover, many studies and publications have proven that BMAC has the ability to restore healthy and functional tissues even in cases of high-grade articular cartilage injury [11, 25–27]. The bone marrow aspirate (BMA) is usually harvested from an ipsilateral iliac crest prior to the main procedure. A sharp trocar with an aspiration needle is placed in the bone between the cortices, about 3–5 cm deep. An average total aspiration volume of 60 mL is harvested, using a standard syringe. Frequently used centrifugation systems include the "RegenKit Extracell BMC" (Regen Lab, Le Mont-sur-Lausanne, Switzerland), "Arthrex Angel®"(Arthrex, Naples, United States), "Harvest Technologies system" (Plymouth, MA) or the "Cobe 2991 Cell Processor" (Terumo BCT, Paris, France) [28].

The aspirate is then prepared and centrifuged to obtain a concentrated product. The rationale behind the process is to increase the proportion of mesenchymal stem/progenitor cells (MSCs) in plain BMA, which is in between 0.001 and 0.01% of the nucleated cells [29]. The process of centrifugation not only results in a higher proportion of MSCs but also higher concentration of platelets and disrupts cell components increasing free

growth factors that might be predominantly relevant for the regenerative processes. The average processing time takes around 15 min, but a newly introduced bone marrow retrieval system (Marrow Cellution®) may reduce time and cost of the procedure and avoid regulation problems regarding cell manipulation by centrifugation. Combination of gradual aspiration through a system of lateral holes reduces the peripheral blood harvest, which results in an aspirate consisting a greater amount of fibroblast-like colony-forming units (CFU-f) without the centrifugation step.

10.4 Scaffold and Stem Cell Surgical Technique

The first and crucial decision in the surgical treatment of OCL of the talus is if the defect is accessible through an anterior approach or a medial malleolar osteotomy is needed in case of the medial talar dome OCL. Lesions on the lateral side are usually more accessible in plantar flexion and only in rare cases require a fibular osteotomy, which is a technically challenging procedure. Figure 10.1 shows basic surgical procedures to access chondral lesions of the talus [30]. The second decision is if an osseous reconstruction is necessary in addition to the cartilage repair procedure. In that case, cancellous bone can be harvested from the tibia or from the iliac crest with a coring drill instrument to provide a stable bony reconstruction [31]. Defects that are deeper than 5 mm are considered indicated for cancellous bone filling as has been stated in the latest published recommendations of a consensus group [32]. For chondral defects without bony defect, the same group also recommended the use of a biomaterial to facilitate cartilage tissue formation and support fill of the defect, especially in defect sizes bigger than 10 mm in diameter. The treatment options are the application of a biomaterial, mostly hyaluronan-based scaffold, filled with bone marrow aspirate concentrate (BMAC) preferable without microfracture. The bone marrow harvested from the iliac crest is a source of cells that provide a biological regenerative potential in the defect without disturbing the subchondral bone. However, a thorough debridement of the defect and removal of any unstable fragments in the cartilage or bone is mandatory for a successful outcome. The surgical application technique requires bone marrow aspiration followed by its concentration, as well as the seeding of the scaffold and the implantation procedure. Trials investigating BMAC in combination with scaffolds used this approach for type II chronic talus cartilage lesions of >1.5 cm^2 [22, 33].

Firstly, bone marrow is harvested and centrifuged to obtain a concentrated product (Fig. 10.2a). We advocate the use of batroxobin enzyme (Plateltex Act, Plateltex SRO, Bratislava, Slovakia), to activate BMAC and to produce a sticky clot material (Fig. 10.2b) that makes the application into the defect easier. A standard

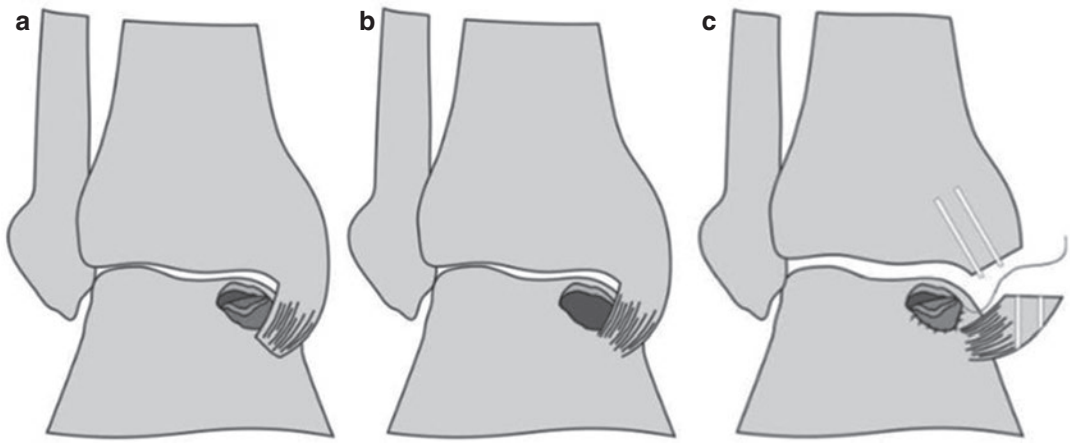

Fig. 10.1 Surgical procedures to access osteochondral lesions of the talus (**a**) delaminated piece of cartilage, (**b**) debrided defect and (**c**) malleolar osteotomy and suturing

ankle arthroscopic procedure is performed, and the lesion site is visualized (Fig. 10.3a), debrided until healthy bone (Fig. 10.3b) and clear cartilage edges are visible and measured. According to the measurements, a scaffold is cut to fit into the defect side. For a 2 × 2 cm hyaluronan scaffold,

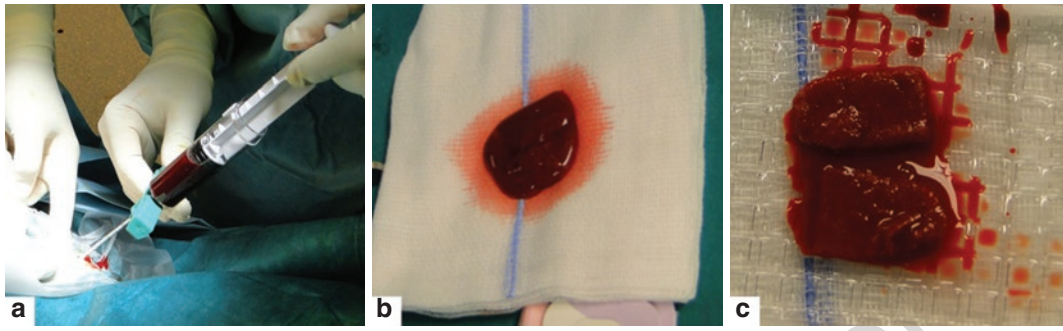

Fig. 10.2 Preparation of the HA-BMAC. (**a**) Harvesting bone marrow from the ipsilateral iliac crest using a sharp trocar (**b**) bone marrow aspirate concentrate (BMAC) after activation with batroxobin enzyme forms a sticky clot (**c**) hyaluronic acid-based (HA) scaffold combined with BMAC clot ready for implantation

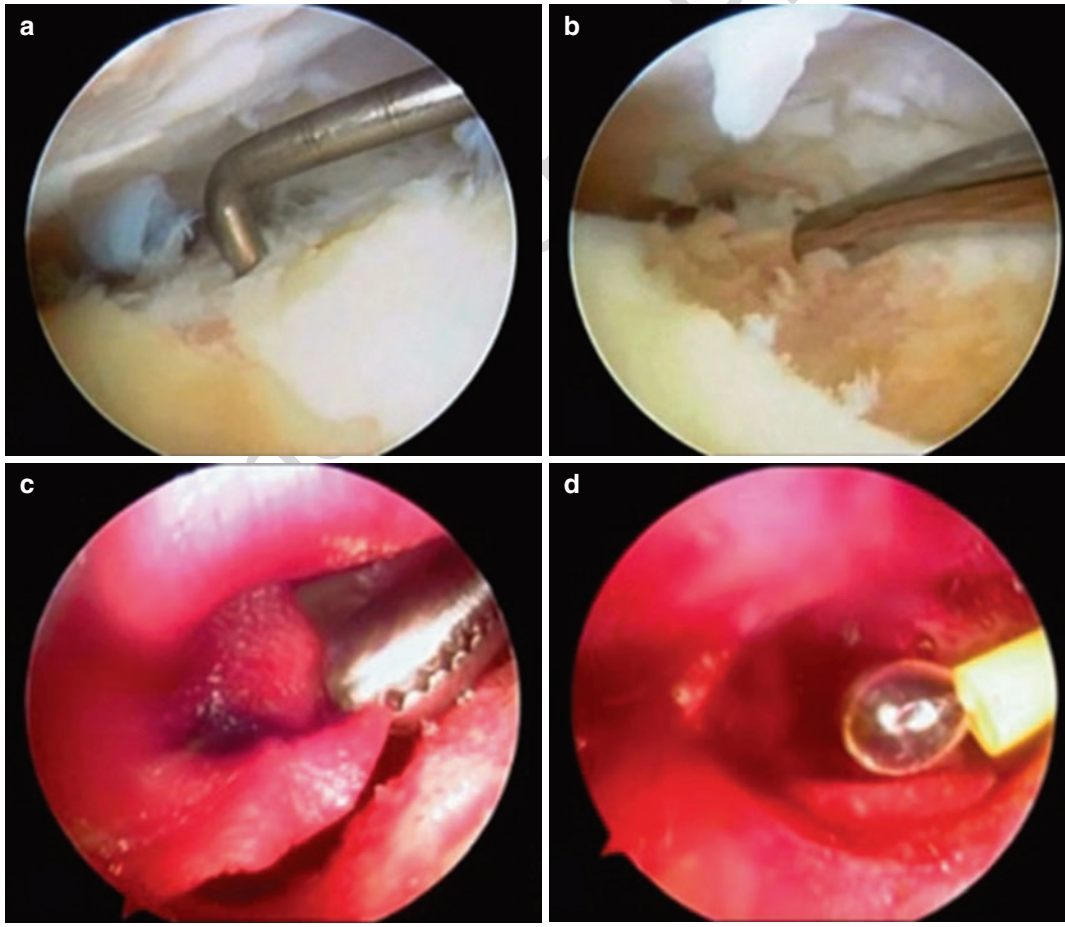

Fig. 10.3 Arthroscopic procedure with the use of HA-BMAC osteochondral lesion of the talus. (**a**) Identification of the lesion on the talar dome, (**b**) lesion debridement with a curette, (**c**) placement of the HA-BMAC into the lesion and (**d**) adding fibrin glue to secure the scaffold

approximately 2–3 mL of BMAC is needed to seed the matrix (Fig. 10.2c). The seeded matrix is then placed onto the debrided or bone augmented defect side (Fig. 10.3c). It is recommended to use a cannula or halfpipe-like instrument in order to safely transport the matrix into the joint. This surgical step might be challenging, sometimes a slight widening of the arthroscopic approach is necessary, but special devices have been designed to aid this crucial step. After scaffold placement some authors add platelet-rich plasma or platelet-rich fibrin (Fig. 10.3d).

Alternatively, in cases of bigger defects or problems with the arthroscopic technique, the scaffold can be properly placed using an open approach. Finally, the ankle is moved under visual control to ensure the correct placement and stability of the implanted scaffold. In cases of malleolar osteotomy, the bone fragment is reduced and fixed with screws; the holes for screw placement should be predrilled before the osteotomy to achieve a full anatomical reconstruction.

10.5 Conclusion

For treatment of osteochondral lesions of the talus, the addition of biologics, primarily BMAC, is recommended by the evidence level C studies. Giannini et al. showed significant improvements in AOFAS score and histological and immunohistochemical appearance up to 24 months post-treatment [33]; in a follow-up trial, the AOFAS score decreased at 36 and 48 months post-treatment and plateaued at 72 months [22]. Vannini et al. presented another insightful result; the authors could show that around 97% of patients could return to activity and 73% returned to sports at a preinjury level [34]. Based on the current evidence, the use of biomaterial and biological augmentation with BMAC can be used in the treatment of osteochondral lesions of the talus. Nevertheless, more long-term results are needed to fortify these recommendations.

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11 New and Emerging Techniques in Cartilage Repair: Matrix-Induced Autologous Chondrocyte Implantation (MACI)

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Autologous chondrocyte implantation (ACI) was originally developed based on the work of Smith, who cultured chondrocytes ex vivo [1]. Grande et al. treated full-thickness cartilage defects with expanded chondrocytes and showed viable chondrocytes and hyaline-like repair tissue on histology [2]. Initially successful in treating osteochondral defects (OCD) in the knee [3], ACI has subsequently been adopted for treatment of osteochondral lesions of the talus (OLT).

ACI is a two-stage procedure in which healthy chondrocytes are harvested arthroscopically from nonessential areas such as the loose osteochondral fragment, the periphery of the OLT, or the ipsilateral knee intercondylar notch. The authors prefer to use the ipsilateral intercondylar notch due to decreased cartilage-forming capacity of the excised osteochondral fragment [4] and concern for creating a new OLT by biopsy of the talus [5]. The harvested chondrocytes are sent to a commercial laboratory to be cultured and expanded into millions of cells. In the second stage, the isolated and expanded chondrocytes are implanted into the prepared OLT under a harvested periosteal patch that is sealed with 6-0 sutures and fibrin glue.

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More recently, modifications have been made to the original technique to try to reduce its associated pitfalls. Matrix-induced autologous chondrocyte implantation (MACI) obviates the need for periosteal patch harvest by using a biodegradable scaffold to retain chondrocytes and theoretically reduce leakage [6]. Periosteal donor site morbidity and postoperative patch hypertrophy can thus be avoided. The cultured chondrocytes are dispersed on a porcine collagen type I/III scaffold which is then implanted onto the osteochondral lesion. This procedure will be further discussed in detail later in this chapter.

Currently, MACI is approved by the Food and Drug Administration for use in the knee. Contraindications include a history of hypersensitivity to aminoglycosides or porcine material, malalignment that would increase stress on the graft, advanced osteoarthritis, history of inflammatory arthritis, and uncorrected blood coagulation disorders.

There are still no large comparative blinded studies of MACI for OLTs, and the evidence for its use is currently limited to level IV case series.

11.1 Patient Selection

Patients with OLTs who have failed extensive nonsurgical management including physical therapy, bracing, casting, and nonsteroidal anti-inflammatory medication should be considered

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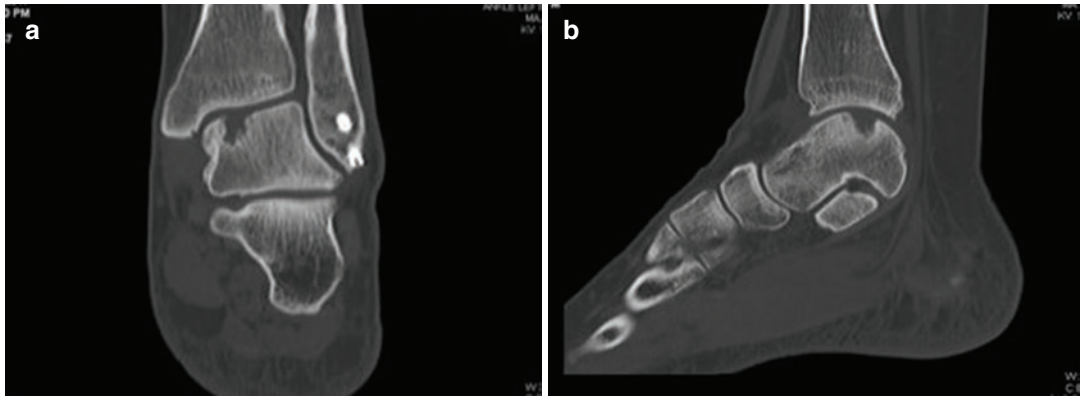

Fig. 11.1 Preoperative CT of a left ankle with a cystic osteochondral lesion. (**a**) Coronal view demonstrating medial location of the cystic osteochondral lesions. (**b**) Sagittal view showing the cystic osteochondral lesion in approximately the middle of the medial talus

for a cartilage transplant. MACI should be considered for an OLT between 1.07 and 4 cm^2. MACI is also indicated for patients who have failed marrow stimulation procedures. Additionally, if the lesion is deeper than 6 mm, bone graft augmenting of the lesion should be considered [7]. Worse outcomes have been reported when the lesion is more than 7 mm in depth, and this should be kept in mind when indicating patients with these lesions for MACI [8]. Unipolar lesions involving only the talus are preferred.

11.2 Imaging

Preoperative imaging should include standard weight-bearing anteroposterior, mortise, and lateral plain films. Stress radiographs with Telos device should be performed if ligamentous injury is suspected.

Magnetic resonance imaging (MRI) should be routinely performed. It is useful to evaluate the articular cartilage, subchondral bone, and periarticular soft tissues.

Computed tomography (CT) with 3D reconstructions is helpful for localizing and accurately measuring the size of the lesion, especially if there are associated cysts. CT images represent the true size of the lesion, absent the obscuring bone edema often seen on MRI (Fig. 11.1a, b).

11.3 Equipment

For the first phase, supine ankle arthroscopy is performed through anteromedial, anterolateral, and posterolateral portals using 30 and 70° 2.7 mm arthroscopes with noninvasive soft tissue distraction [9]. A 1.9 mm 30° arthroscope may be used for tight joints. For the cartilage harvest in the knee, ring curettes and arthroscopic graspers are used, as well as a cannula for graft harvest.

11.4 Positioning

In the first phase, the ankle arthroscopy is performed in the supine position. All padding should be removed from the leg of the table and the nonoperative leg should be padded independently, allowing for clearance between the operative ankle and the table. The knee and hip are both flexed at around 45° and held in place with a Ferkel Thigh Holder (Smith & Nephew). The ankle is distracted with a sterile soft tissue distractor (Guhl Non-Invasive Ankle Distractor, Smith & Nephew).

In the second phase, when the open procedure is done, the ankle is positioned based on the surgical approach to the lesion.

11.5 Surgical Procedure

The first phase of the procedure includes a diagnostic ankle arthroscopy using previously described techniques to evaluate the lesion and confirm that MACI treatment is appropriate. Debridement of non-involved parts of the ankle can then be performed, but the lesion should be left alone until the second stage. At the same time, the cartilage biopsy is removed from the ipsilateral knee intercondylar notch. From standard knee arthroscopy portals, cartilage is harvested from the lateral aspect of the intercondylar notch by a sharp ring curette (a 200–300 mg sample is necessary). Care is taken to avoid detaching the cartilage completely so that it does not float free in the knee joint. A tissue grasper is then used to remove the cartilage piece with a gentle twisting motion and removed out a cannula to prevent its entrapment in the anterior soft tissue. The tissue is then stored in packaging provided by and as instructed by the Vericel Corporation (Vericel, Cambridge, Massachusetts).

The second phase of the procedure is the implantation phase. This is typically performed at a minimum of 6–12 weeks after the cells have been cultured and placed onto the membrane. In most cases, the implant is available for 5 years after biopsy. Implantation can be performed by open surgery or occasionally by arthroscopically. The size and location of the lesion will often dictate which approach is optimal. Arthroscopic results have so far been promising, with multiple studies showing good short- and medium-term results [6, 10, 11].

11.5.1 Traditional Open Surgery

A tourniquet is placed to ensure the surgical field remains bloodless. The location of the lesion determines the positioning of the patient. Medial lesions are positioned supine and a bump is placed under the contralateral hip. A medial malleolar osteotomy is performed to gain access to the lesion. First, the medial malleolus is predrilled for two 4.0 mm cancellous lag screws or a medial malleolar hook plate (Fig. 11.2). Then, under fluoroscopy, a guide pin is used as a cutting guide for the saw to assist in making the osteotomy in exactly the correct plane (Fig. 11.3). Imaging should confirm that the saw will exit lateral to the extent of the OLT so that the entire lesion can be accessed.

If the lesion is lateral, it can be accessed by predrilling the fibula for two interfragmentary lag screws and then making an oblique fibular

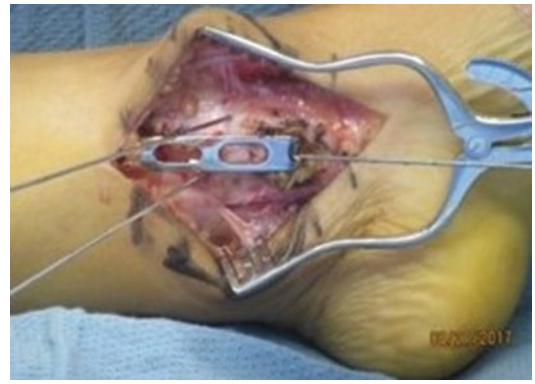

Fig. 11.2 Predrilling the medial malleolus for a medial malleolar osteotomy, utilizing a hook plate for reduction after insertion of the MACI graft

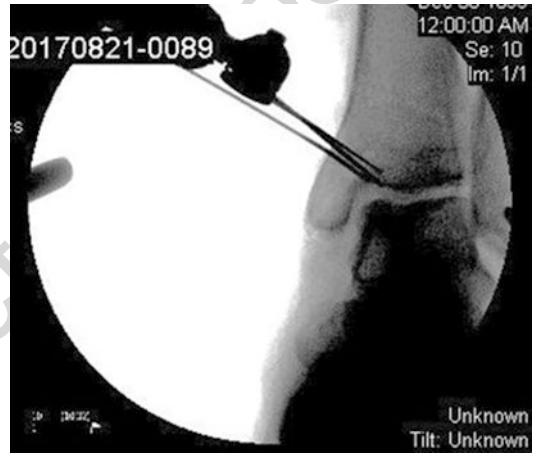

Fig. 11.3 Fluoroscopic X-ray demonstrating inserting guide pins in the correct planes. The saw tip is then placed on the guide pins to assist with the appropriate location of the medial malleolar osteotomy

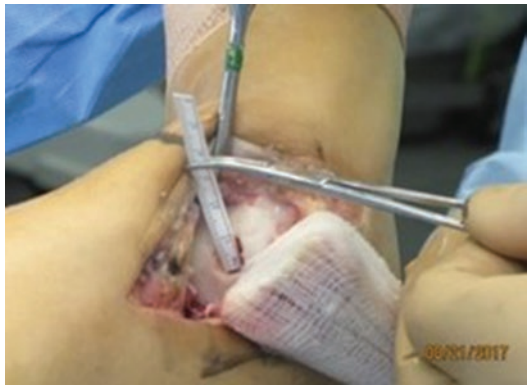

Fig. 11.4 After excising the osteochondral lesion, the defect is measured with a ruler to get the exact dimensions for preparation of the MACI graft

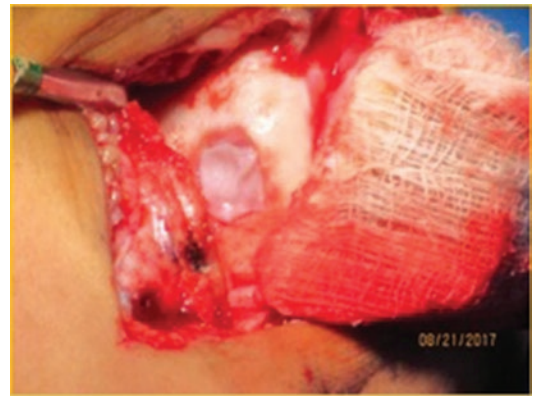

Fig. 11.5 The MACI graft is self-adherent but sometimes a few stitches are used to further secure it to the osteochondral lesion bed. Pictured is the graft in the medial talar dome of a left ankle prior to fibrin glue insertion

osteotomy. Releasing the anterior talofibular ligament and the anterior capsule allows for the fibula to be rotated posteriorly. A cuff of tissue is left on the fibula for latter Brostrom-type repair of the lateral ligaments.

After adequate exposure is obtained, the OLT can then be debrided to stable vertical margins. A 15 blade can be used to obtain sharp vertical borders. The subchondral bone must be left intact in order to prevent osseous bleeding. After deflating the tourniquet, the lesion is dried with thrombin-soaked pledgets. The lesion is then measured to determine the exact size of the defect (Fig. 11.4). A sterile suture foil package is then pressed into the defect to form a template. The MACI membrane is then cut to match this template. After further drying with thrombin, the membrane is then placed onto the lesion and pressed gently into place, ensuring that the cell side is facing down into the lesion (Fig. 11.5). Fibrin glue is then applied to the membrane, sealing it in place. After the fibrin has been set (5–7 min), range of motion testing should then be performed to ensure that the MACI graft is stable. 6-0 Vicryl sutures can be applied for additional security, but are rarely needed because the membrane is self-adherent.

The osteotomy is then reduced and repaired. An additional transverse screw at the proximal portion of the medial malleolar osteotomy is used for additional fixation due to the oblique nature of the osteotomy. If a hook plate is utilized, it is secured with compression across the medial malleolar osteotomy (Fig. 11.6a, b). Lateral osteotomies can be fixed with a neutralization one-third tubular plate after placing the interfragmentary lag screws. The incisions are then closed with 3-0 Vicryl sutures, followed by 4-0 nylon vertical mattress sutures. The leg is then placed in a well-padded short-leg cast that is subsequently split with the cast saw in the recovery room.

11.6 Arthroscopic Technique

Due to the less technically demanding nature of the MACI procedure, it is reasonable to perform entirely arthroscopically, thereby avoiding the morbidity of an osteotomy. The all arthroscopic second stage procedure is performed with the same setup and through the same portals as the first stage. After debridement of any loose cartilage fragments and synovitis, debridement of the lesion should occur at this time, using arthroscopic different-angled curettes to obtain stable vertical borders. The lesion is then accurately measured and the MACI graft cut to size. Next, the arthroscopic fluid flow is stopped and all fluid is drained from the ankle joint. Thrombin-soaked pledgets are inserted from the portal closest to the lesion and used to dry the lesion.

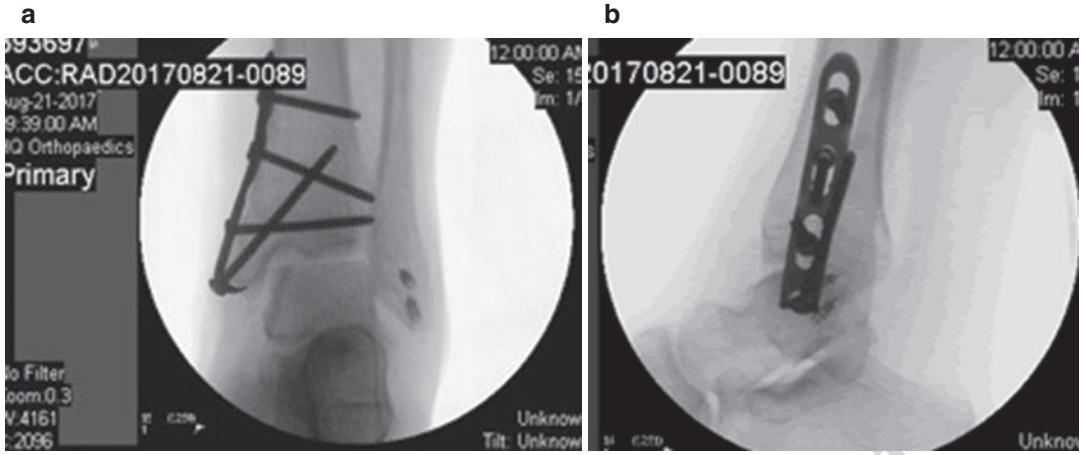

Fig. 11.6 Reduction of the medial malleolar osteotomy. (**a**) AP X-ray demonstrating the appropriate location of the hook plate and screws to reduce and compress the medial malleolar osteotomy. (**b**) Lateral radiograph demonstrates the plate located in the medial of the distal tibia and medial malleolus

Arthroscopic forceps or a specialized cannula delivery system can be used to deliver the matrix into the ankle joint [12]. A probe and a freer elevator can then be used to place the matrix onto the lesion and precisely fit it into the lesion. Fibrin glue should then be placed over the matrix with a commercially available applicator. Once the fibrin is set (5–7 min), the ankle is then taken through extension and flexion to ensure that matrix is stable. All instruments should then be removed and the portals closed with 4-0 nylon vertical mattress sutures.

11.7 Postoperative Protocol

The importance of a comprehensive protocol for postoperative care and rehabilitation cannot be overstated. The physician, patient, and physical therapist must work as a team and be in close contact during the process. The goals are to promote effective healing of the surgical site and cartilage graft and to then return the patient to a high level of function. There is a paucity of good evidence in the literature, so much of the information is based off of animal models as well as accepted information of cartilage physiology [13]. It is also reasonable to extrapolate information from ACI/MACI in the knee. Most authors agree that supervision by a skilled physical therapist is necessary and on the general concepts to follow [14].

The rehabilitation process can be divided into four phases [15]:

1. Phase 1 is the "healing phase," surgery to week 6.
2. Phase 2 is the "transitional phase," weeks 6–12.
3. Phase 3 is the "remodeling phase," weeks 12–32.
4. Phase 4 is the "maturation phase," weeks 32–52.

The following protocol is for first-generation ACI, but for newer techniques, quicker advancement can be considered because they don't rely on periosteal patch graft containment.

Phase 1: Surgery to Week 6

Cast and sutures are removed at 2 weeks postsurgery. A compression stocking is applied, and the patient is placed in a controlled ankle motion (CAM) walking boot. They are allowed to start partial weight bearing up to 30 lb. Range of motion exercises are initiated at week 2 and focus on the sagittal plane. At 4 weeks stationary bike with no resistance is begun. Weight bearing is increased toward full weight bearing in a CAM boot at week 6, and osteotomy healing must be checked. They are transitioned to a lace-up

figure-of-eight brace and supportive athletic shoes when the osteotomy is healed. Formal physical therapy is then initiated. Phase 1 is designed to recover full range of motion while protecting the healing graft. Motion and light compressive forces are needed for healthy chondrocytes [16].

Phase 2: Weeks 6–12

The musculature around the ankle is strengthened under close direction of the physical therapist. Resistance can be added to the stationary bicycle, and proprioceptive exercises are begun. Isometric followed by eccentric strengthening exercises are included in this phase. The basis for this increase in resistance is that the implanted chondrocytes are maturing and can undergo increased compressive loading. An increase in strength and proprioception are needed in order to progress to more demanding activities.

Phase 3: Weeks 12–32

Patients can now increase their activity level and strengthening. Both walking speed and duration can be increased, as long as their pain and swelling allows. No jogging or running is allowed. Strengthening exercises in weight-bearing positions are started. This phase serves to increase strength and endurance while maintaining range of motion, which are needed for sports-specific training. The graft is still maturing in this phase, and 30 min of weight-bearing exercise without pain and swelling is necessary in order to graduate to phase 4.

Phase 4: Weeks 32–52

Cross-training and return-to-sport activities are begun. By 8 months, the graft should tolerate high-impact activities. The therapist can supervise an increase in intensity and duration, with symptoms such as pain and swelling guiding progression. Due to extended periods of immobilization and slow progression in the prior phases, the patient may be generally deconditioned, and generous rest periods between sessions should be standard. Unrestricted activity can begin 12 months after surgery, bearing in mind that the graft continues to mature and remodel for up to 2 years from the time of surgery [17].

11.8 Results

Previously, we have reported on our results of ACI of the talus [11, 18]. Outside the United States, Schneider and Karaikudi did MACI on 20 patients, with a mean follow-up of 21 months. The mean size of the lesions was 233 mm^2. The AOFAS scores improved from 60 to 87, but there were two failures and six patients with no improvement in pain [6]. Magnan et al. treated 30 patients with MACI, with a mean OLT size of 236 m^2. The AOFAS score improved from 37 to 84, with a follow-up of 45 months. However, only 50% of the patients returned to their previous sporting activity [19]. More recently, Kreulen et al. reported on 7-year follow-up of nine patients who had failed previous arthroscopic treatment for an osteochondral lesion of the talus. The average OLT size was 129 mm^2. The AOFAS score went from 62 to 78, and the SF-36 score showed significant improvements in physical functioning, lack of bodily pain, and social functioning, compared with preoperative data [20]. Brittberg et al. studied MACI versus microfracture of the knee in a prospective randomized trial and published results in 2014 and 2018 in the same group. At an average follow-up of 5 years, the symptomatic knee cartilage defects 3 cm^2 or greater treated with MACI were significantly improved over microfracture [21, 22].

11.9 Complications

Infection, bleeding, wound breakdown, neurovascular injury, and continued symptoms are possible in any foot and ankle surgery. Graft and patch hypertrophy are specific complications of ACI, but are decreased in second- and third-generation ACI techniques such as MACI [23]. If an osteotomy is performed, nonunion and hardware-related pain are possible complications.

Pearls:

1. If performing an osteotomy, ensure that direct perpendicular access to the entirety of the lesion is maintained. The osteotomy site should exit the plafond lateral to a medial OLT and medial to a lateral OLT.

2. A thorough debridement of all diseased tissue from the lesion is necessary, and stable vertical walls should be obtained.
3. Concomitant ankle malalignment or instability must be corrected.
4. Be prepared to perform an open procedure with or without an osteotomy in the event that the lesion proves to not be amenable to arthroscopic MACI graft placement.

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Osteochondral Lesions of the Talus

12

P. A. D. van Dijk and C. N. van Dijk

12.1 Introduction

Intra-articular ankle pathologies are a relatively common source of ankle disability, most often associated with osteochondral lesions (OCL) of the talus [1]. Historically, numerous terms have been used to describe what is known being referred to as OCL, including osteochondritis dissecans, transchondral talus fracture, and osteochondral talus fracture.

OCL consist of both pathology to the subchondral bone and its overlying cartilage. The incidence of these lesions in patients with acute lateral ankle ligament ruptures varies around 5–7% [2–4]. Typically, talar OCL lead to swelling, pain, functional impairment, and disability. While most traumatic cartilage lesions have a good prognosis and patients will become asymptomatic eventually, traumatic osteochondral lesions have a poor healing tendency.

Despite constant improvement in the treatment of symptomatic OCL, proper selection of the most suitable treatment remains a challenge. In order to optimize management, adequate knowledge of the clinical presentation and appropriate diagnostics of OCL is essential. This chapter gives an overview of the pathophysiology and classification, patient history, clinical examination, and diagnostics of OCL of the talus. Moreover, it provides a step-by-step description of anterior ankle arthroscopy technique in treating these lesions.

12.2 Classification of Talar Osteochondral Defects

Several classifications for OCL have been described in literature, with the first classification being from Berndt and Harty [5]. Years later, Ferkel and Sgaglione proposed a classification bases on CT findings [6]. Anderson described an MRI-based classification and Cheng and Ferkel proposed an arthroscopy-based classification [7,

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8]. In a more recent consensus statement, the ISAKOS consensus group concluded that there is only limited need for these classification systems, and proposed a therapy-based classification:

1. Asymptomatic lesions or low symptomatic lesions: conservative treatment.
2. Symptomatic lesions up to 15 mm: debridement and drilling/microfracturing/bone marrow stimulation.
3. Symptomatic lesions larger then 15 mm: fixation.
4. Cystic lesions in tibial roof or large talar cystic lesions: retrograde drilling and bone transplantation.
5. Failed primary treatment: osteochondral transplant, hemicap or calcaneal osteotomy.

In patients with category 4 or 5 OCL, debridement and bone marrow stimulation can also be considered [9–11].

12.3 Pathophysiology of Pain Related to Talar Osteochondral Lesions

Both traumatic and nontraumatic talar OCL exist. Lateral lesions are associated with trauma in 98% of cases; in medial lesions this is only 70% [12]. The nontraumatic etiology concerns idiopathic OCL, associated with ischemia, necrosis, and genetical predisposition. In this matter, OCL of the talus have been described in identical twins and siblings [13–15]. In 10–25% of patients, the lesion is found bilateral with most OCL being asymptomatic [16, 17]. After a traumatic event, however, they can become symptomatic.

Traumatic lesions may develop after repetitive loading of the damaged articular cartilage surface, leading to local cellular degeneration or death by the disruption of collagen fibril ultrastructure and thickening of the subchondral bone [18]. In animal experiments, van der Vis et al. have shown that oscillating fluid pressure can lead to osteolysis [19]. Fluid pressure-induced bone resorption is a powerful bone-resorptive stimulus. In situations with net bone loss, there is ongoing bone formation adjacent to bone resorption [20]. This bone resorption due to hydrostatic pressure leads to subchondral cysts surrounded by a newly formed calcified zone [21].

Numeral theories have been proposed as a cause of OCL pain, including synovial pain, elevated joint pressure, elevated interosseous (venous) pressure, and bone pain:

- Synovial pain would imply tenderness on palpation of the inflamed and thickened synovium. During physical examination, the synovium is relatively easily accessible on both the anteromedial and anterolateral joint line. Patients with a symptomatical OCL, however, usually present with absence of recognizable tenderness on palpation of the synovium, implicating that the synovium is not the main cause of pain in these patients [22].
- In order for articular pressure to cause pain, the joint must be filled with synovial fluid. In patients with a talar OCL, there can be some ankle effusion but this is not sufficient to give a relevant rise in intra-articular pressure. These patients normally demonstrate remarkably low levels of effusion. Therefore, elevated joint pressure is not a plausible cause of pain.
- Several authors studied the relationship between painful osteoarthritis and intraosseous venous pressure. Their research suggested that blockage of flow in the periarticular veins can lead to high interosseous venous pressure, with osteotomy or cortical fenestration resulting in a remarkable reduction in interosseous pressure [23, 24].
- It has been determined that nerve fibers are widely distributed in bone tissue. The nerves in the bone marrow, for example, show apparent regional distribution with different densities. They are often associated with blood vessels and show a beaded appearance [25, 26]. Local fluid pressure is a powerful stimulus for the nerve endings in the bone marrow.

Interstitial water is expressed from the cartilage matrix as it is compressed, leading to stimulation of the nerves.

- Cartilage consists of cells (chondrocytes), collagen (arcade structure), and water. The water content of human articular cartilage is determined to be 79% [27]. Under dynamic load (0.1 s), the deformation of talar cartilage has been determined to reduce 20% of its thickness, releasing water into the joint space. Under static load (30 min), up to 55% of the thickness of the cartilage will be reduced [27]. In case the subchondral bone plate is damaged, (released) water can penetrate into the richly innervated subcortical spongiosa [28]. In this way, loading of the damaged talar cartilage by the distal tibia will result in local fluid pressure towards the subchondral bone plate. If the subchondral bone plate is damaged, fluid can enter the underlying bone, leading to bone resorption and eventually cyst formation [29].

Extensive review of the literature revealed that pain associated with talar OCL does not arise from the cartilage lesion itself. It is most probably caused by repetitive high fluid pressure during dynamic loading such as walking, resulting in stimulus of the highly innervated subchondral bone underneath the cartilage lesion [22, 28].

12.4 Patient History and Clinical Examination

In the acute situation, diagnosis of talar OCL is often delayed because of a relatively low index of suspicion. In chronic cases, careful patient history and clinical examination is the key to proper diagnosis of an OCL of the talus. On examination, these patients can show surprisingly little abnormality. In most cases, there is a normal range of motion with the absence of recognizable pain on palpation or swelling during physical examination. Deep ankle pain during or after activity is the most important sign indicating an OCL of the talus [30]. Other findings include a clear history of ankle trauma, weakness and instability of the ankle, and swelling and stiffness of the ankle joint [31–34].

12.5 Additional Diagnostics

In general, routine radiographs of the ankle consist of an AP and lateral radiographs. The X-ray may show an area of detached bone, surrounded by radiolucency. In most cases, however, the damage is too small to be visualized on initial radiographs [31, 32, 35]. By repeating radiographics in a later stage, the abnormality sometimes becomes apparent. With conventional radiographs having only moderate sensitivity, additional imaging is recommended in diagnosing talar OCL [30].

A heel-rise view with the ankle in a plantarflexed position may reveal a posteromedial or posterolateral defect. For further diagnostic evaluation, a CT scan and MRI have demonstrated a similar level of accuracy ($p = 0.33$) [36]. For preoperative planning, however, a CT scan has the advantages of detection and characterization of the bony lesion. Based on the findings of a study by van Bergen et al, a recent international consensus statement suggested that the preferred CT settings are helical or spiral CT with ultrahigh-resolution axial slices with an increment of 0.3 mm and a thickness of 0.6 mm, and a coronal and sagittal reformation of both 1 mm [30, 35]. The consensus panel advised CT scan for preoperative planning [30].

12.6 Treatment

There are various published surgical techniques for the treatment of symptomatic OCL. These are based on one of the following three principles:

1. Debridement and bone marrow stimulation (microfracturing, abrasion arthroplasty, drilling)
2. Securing a lesion to the talar dome (retrograde drilling, fragment fixation)

3. Preservation of hyaline cartilage (osteochondral autografts, allografts, autologous chondrocyte implantation)

The effectiveness of the different principles varies greatly in literature and no superior treatment strategy is yet defined. Several systematic reviews of the literature have been published [37–41], with the two most recent systematic reviews concluding that there is no superior treatment in treating both primary and secondary OCL of the talus [42, 43]. Worldwide, bone marrow stimulation is most often used in the treatment of primary lesions, because it is highly effective, it is relatively inexpensive compared to other (implantation) techniques, it has low morbidity, it provides a quick recovery, and it provides early return to sports [43].

The choice of the management strategy when treating a talar OCL depends mostly on the duration of complaints, size of the lesion, and whether we deal with a primary or secondary OCL. In general, asymptomatic or low symptomatic lesions are treated conservatively with rest, ice, temporarily reduced weight bearing, and, in case of giving way, an orthosis.

For mechanically unstable lesions, fixation is preferred in (semi) acute situations in which the fragment is 15 mm or larger. In adolescents, refixation of a lesion must always be considered even in fragments that are smaller than 15 mm. Large talar cystic lesions can be treated by retrograde drilling and filling the defect with a bone graft. In case of failed primary treatment, an osteochondral transplant can be considered. The technique of osteochondral transplant should be reserved for secondary cases with a malleolar osteotomy often being needed in order to adequately gain access to the lesion.

The type of surgical treatment influences the perioperative exposure. Most primary lesions can be treated by ankle arthroscopy. For posteromedial lesions in a stable ankle some authors recommend the use of either malleolar osteotomy, "grooving" of the anteromedial distal tibia, or drilling through the medial malleolus [12, 17, 44]. In the experience of the senior author of this chapter, however, 90–95% of all OCL can be treated by means of anterior arthroscopy by bringing the foot in hyperplantarflexion. It should be noted, however, that skills and experience are required [45]. Posterior lesions in the most posterior quarter of the talar dome, which cannot be reached by hyperplantarflexion, can be treated by means of a two-portal endoscopic hindfoot approach.

12.7 Anterior Ankle Arthroscopy: A Step-by-Step Description of the Procedure

From anterior to posterior, the talar dome can be divided into four equal parts. When the OCL is located in one of the three anterior parts of the talar dome, it can be treated by a routine anterior ankle arthroscopy. In cases the lesion is located in the most posterior quarter of the talar dome, the defect should be approached by a posterior ankle arthroscopy or by means of a medial malleolar osteotomy [46]. For anterior ankle arthroscopy technique, two portals are created:

12.7.1 Anteromedial Portal

The anteromedial portal is made first since it is easy to access, especially when the ankle is in a dorsiflexed position. In this position, the point of entry is easily reproducible and the risk of neurovascular damage is minimal. The portal is placed just medial to the anterior tibial tendon at the level of the joint line. At this level, a depression (soft spot) can be palpated with the ankle in hyperdorsiflexion. In the horizontal plane, the depression is located between the anterior tibial rim and the talus. In the vertical position, the anterior tibial tendon is the landmark. The anterior tibial tendon should be palpated in the dorsiflexed position. Note that in this dorsiflexed position, the tendon moves 1 cm lateral. The location of the anteromedial portal can now be marked on the skin just medial from the anterior tibial tendon. Care must be taken not to injure the saphenous vein and nerve transversing the ankle joint along the anterior

edge of the medial malleolus. By moving the ankle joint from the plantarflexed position to the dorsiflexed position, the talus can be felt to move in relation to the distal tibia. The palpating finger gets locked into the soft spot in the hyperdorsiflexed position. A small longitudinal incision is made through the skin only, just medial from the anterior tibial tendon. Blunt dissection is performed with a mosquito clamp through the subcutaneous layer and through the capsule into the ankle joint. With the ankle in forced dorsiflexed position, cartilage damage is avoided. In this forced dorsiflexed position, the arthroscope shaft with the blunt trocar is introduced. When the trocar is felt to contact the underlying bony joint line, the shaft with the blunt trocar is gently pushed further into the anterior working area in front of the ankle joint towards the lateral side. The anterior compartment is irrigated and inspected.

12.7.2 Anterolateral Portal

Next, the anterolateral portal is made. It is placed just lateral to the tendon of the peroneus tertius or slightly proximal to the joint line and is made under direct vision by introducing a spinal needle. In the horizontal plane, it is situated at the level of the joint line or slightly proximal to it. In the vertical plane, it is located lateral to the common extensor tendons and the peroneus tertius tendon. Care must be taken to avoid damage to the subcutaneous superficial peroneal nerve, which can often be palpated or visualized by placing the foot in forced hyperplantarflexion and supination. The intermediate dorsal cutaneous branch of the superficial peroneal nerve crosses the anterior aspect of the ankle joint superficial to the common extensor tendons. Damage to this branch can be avoided by staying just lateral to the extensor tendons. Once the lateral branch is identified, its position can be marked with a marking pen on the skin.

It should be noted that the location of the anterolateral portal might vary depending on the location of the lesion in the ankle joint. For the treatment of anteromedial ankle pathology, the anterolateral portal can be placed slightly above the level of the ankle joint and as close to the peroneal tertius tendon as possible. For the treatment of lateral pathology, the anterolateral portal is placed at the level of the joint line and more laterally. After a small skin incision has been made, the subcutaneous layer and capsule are divided bluntly with a mosquito clamp. Most important is to apply a nick and spread technique.

12.7.3 Surgical Procedure

Routinely, the procedure is performed without distraction. The standard anteromedial and anterolateral portals are created as described above. In case of a medial OCL, the 4 mm arthroscope is moved over to the anterolateral portal and the instruments are introduced through the anteromedial portal. For an anterolateral lesion, the arthroscope remains in the anteromedial portal and the instruments are introduced through the anterolateral portal. In case of osteophytes, these are removed first. Synovitis is removed with the ankle in plantar flexion, after which the OCL can be identified.

Not only can the lesion be palpated with a probe, but it should also be possible to visualize at least the most anterior part of the lesion. It can be helpful to distract the joint by means of a soft-tissue distractor [47].

After removal of the fragment, a 3.5 or 4.5 mm synovator is now introduced into the lesion. After it has been debrided, the arthroscope is moved over to the portal opposite the defect to check the completeness of the debridement. It is important to remove all dead bone and overlying, unsupported, unstable cartilage. Every step in the debridement procedure should be checked by regularly switching portals in order to perform a precise and complete debridement, with removal of all loose fragments. Introduction of the instruments and the arthroscope is performed with the ankle in the fully dorsiflexed position, thus preventing iatrogenic cartilage damage. After full debridement, the sclerotic zone is perforated with a microfracturing technique.

12.8 Rehabilitation

After arthroscopic debridement and drilling, patients are encouraged to make active plantarflexed and dorsiflexed ankle movements. Partial weight bearing is allowed of up to 6 weeks, and full weight bearing after 6 weeks. Running on even ground is permitted after 12 weeks. Full return to sporting activities is advised at 5 months after surgery.

12.9 Pearls and Pitfalls

- Pain associated with talar OCL is caused by repetitive high fluid pressure during dynamic loading such as walking, resulting in stimulus of the highly innervated subchondral bone underneath the cartilage lesion.
- On physical examination, patients with a talar OCL often show surprisingly little abnormality. Deep ankle pain, during or after activity, is the most important sign to indicate an OCL of the talus.
- For appropriate preoperative planning, a CT scan is the preferred strategy.
- Management of talar OCL depends on the size of the lesions, duration of complaints, and whether it is a primary or secondary lesion.
- 90–95% of all primary OCL can be treated by means of anterior arthroscopy by bringing the foot in hyperplantarflexion.
- Active range of motion immediately after arthroscopic surgery is important for optimal treatment outcomes.

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13 Lift, Drill, Fill, and Fix (LDFF): A New Arthroscopic Treatment for Talar Osteochondral Defects

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13.1 Introduction

An osteochondral defect (OCD) of the talus is an injury to the articular cartilage of the talus and its underlying subchondral bone. Although a number of studies described vascular and genetic etiologies of the particular injury, the lesions are most frequently caused by traumatic events, such as ankle fractures and sprains [1–4]. The injury can rigorously affect daily activities of patients leading to a deterioration of the quality of life [5]. The treatment protocol is usually initiated by a conservative protocol by means of shared decision-making (SDM) [6–9]. However, in case of persistence of symptoms, one can opt for one of the many existing surgical procedures. For the primary and smaller defects, a bone marrow stimulation (BMS) procedure can be carried out. However, this surgical intervention may solely be reserved for defects that are less than 107.4 mm^2 in area [10]. Moreover, a number of studies showed that the clinical efficacy of BMS deteriorates over time, most probably due to the fact that osteoarthritis of the ankle joint is being observed at long-term follow-up [11, 12]. The osteoarthritis can be caused by a depressed subchondral bone plate, which is frequently present at midterm [13–15]. Furthermore, the procedure does not aim at mirroring the natural congruency of the ankle joint, being an alternative or combined explanation of the declining clinical efficacy [16–18]. For larger and secondary defects, more aggressive surgical treatment options are probably necessary in order to relieve the patient's symptoms. Even though a recent systematic review by Lambers et al. [19] stated that the authors could not identify a best surgical strategy for these type of lesions, osteo(chondral) transplantation procedures and chondrocyte implantation procedures seem to be effective strategies for secondary defects.

As an alternative to performing bone marrow procedures, cartilage implantations, and osteo(chondral) transplantations, one could execute a fixation procedure for large primary defects as well. A fixation procedure would theoretically preserve the cartilage of the affected region of the talar dome, prevent the degradation of the subchondral bone, and restore the natural congruency of the joint and it would be possible to treat

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large defects with this surgical intervention. In this chapter we present a novel promising arthroscopic internal fixation technique, known as the Lift, Drill, Fill, and Fix (LDFF) technique. Furthermore, we describe the historical perspective of talar osteochondral defects.

13.2 Historical Perspective

The treatment of talar osteochondral defect was most probably initiated in 1743, when Hunter [20] mentioned the following: "From Hippocrates down to the present age, ulcerated cartilage is a troublesome disease; when destroyed, it is not recovered." In 1856, Monro [21] reported on the presence of cartilaginous loose bodies in the ankle joint, and it was the year of 1870 when Paget described these lesions in the knee joint [22]. However, when it comes to the first description of something similar to "osteochondral defects," it was the German surgeon Franz König who was the first to utilize the term "osteochondritis dissecans" when referring to loose bodies originating from the articular surfaces of different joints [23]. The German reasoned that the underlying etiology of these corpora libera was of necrotic nature, thereby accompanying some type of inflammation. Actually, the first description of the term osteochondritis dissecans in the ankle was executed by Kappis, who found great similarities of osteochondral defects in the knee to the ones recognized in the ankle joint [24]. A decade thereafter, in the year of 1932 it was Rendu [25] who published on the etiology and the treatment of an intra-articular fracture of the talus. The terminology and the knowledge behind the etiology and therefore indirectly the treatment of talar osteochondral defects took a turn, when in 1953 Rödén et al. [26] indicated that talar OCDs located on the lateral side of the talar dome were secondary to trauma. This finding suggested that the definition of osteochondritis dissecans seemed to be a misnomer as rather the primary underlying mechanism of etiology was of a traumatic nature. In 1959, the famous article by Berndt and Harty [27] was published. They indicated that not solely lateral lesions of the talar dome could be a consequence of traumatic events, but also medial lesions of the talar dome were prone to be secondary to traumatic events, thereby posing that generally speaking the etiology of the majority of the talar osteochondral defects is posttraumatic. Currently, different descriptions for talar osteochondral defects are being utilized: osteochondral defects, osteochondral lesions, osteochondritis dissecans, transchondral talar fractures, osteochondral talar fractures, talar dome fractures, and flake fractures of the talus. Since the influential publication in 1959, a great number of different surgical techniques have been developed and subsequently published in the literature ever since.

13.3 Arthroscopic LDFF: Indications, Contraindications, and Preoperative Planning

The indication for an arthroscopic lift, drill, fill, and fix procedure is a large (anterior-posterior or medial-lateral diameter >10 mm on computed tomography (CT) scan) primary, acute, or chronic osteochondral defect of the talus [28]. Additionally, the patient needs to have undergone and subsequently failed a conservative protocol for a minimum of 6 months. It should be mentioned that a symptomatic displaced fragment in all patients or a non-displaced fragment in a skeletally mature patient can be fixed as soon as possible; this, so that one minimizes potential intra-articular damage and one maximizes the healing potential [28]. Contraindications for the procedure are loose chondral lesions, ankle osteoarthritis grade II or III, advanced osteoporosis, infectious pathology, and malignancy [28]. There is no contraindication concerning a particular age of the patient as no violation of the growth plate takes place during the arthroscopic LDFF procedure. As preoperative planning for assessment of the talar OCD location, the size, the morphology, and the degree of displacement of an osteochondral fragment, a preoperative CT scan in maximum plantar flexion is advisable to assess the right accessibility of the talar OCD [29–31].

13.4 Surgical Technique: Arthroscopic Lift, Drill, Fill, and Fix (LDFF) Procedure

The arthroscopic fixation procedure has a number of surgical steps that will be outlined hereafter: lift, drill, fill, and fix (LDFF) [32]. The operation is carried out as an outpatient procedure either under general or spinal anesthesia and the patient is positioned in a supine position with slight elevation of the ipsilateral buttock by placing a support at the contralateral side of the patient's pelvis. In order for the surgeon to be able to plantar- or dorsiflex the injured ankle by leaning against the foot sole, the heel of the affected foot is positioned on the end of the operating table. By means of this special position, the surgeon can use the operating table as a lever in case maximum plantar flexion is necessary. When this is required, the surgeon can use a noninvasive soft-tissue distraction device. The surgeon then pays attention to creating the commonly used anteromedial and anterolateral arthroscopic portals. When these have been created, the ankle joint can be visualized. In order to create a proper facilitation of access to the ankle joint, the distal tibial rim is removed. Subsequently, by means of a probe the precise location of the osteochondral defect on the talar dome can be assessed. The first step of the LDFF procedure is the lifting step. In order to prepare for the first step of the LDFF technique, a beaver knife is used to create a sharp osteochondral flap (Fig. 13.1a, b)**.** It should be mentioned that the orthopedic surgeon should pay great attention to leaving the posterior side of the flap purely intact. This flap should be left intact and may then be used as a lever which facilitates an anterior lift by means of a chisel (*lift*) (Fig. 13.1c).

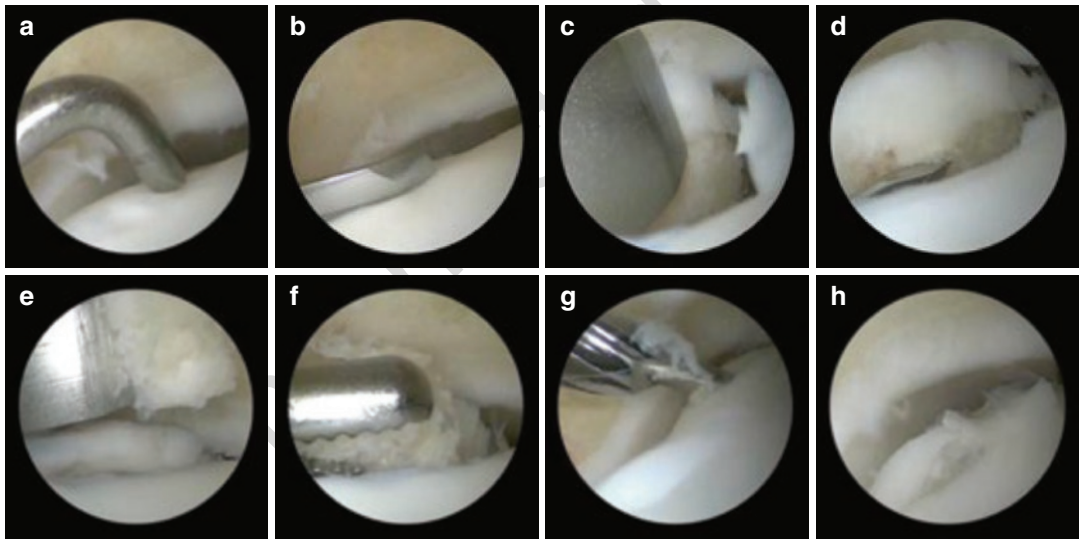

Fig. 13.1 Arthroscopic images of the lift, drill, fill, and fix (LDFF) procedure, a medial osteochondral lesion of the left talus. (**a**) The surgeon palpates the diseased cartilage with a probe in order to identify the precise location of the talar osteochondral defect; this step can be performed when the ankle is held in plantar flexion. (**b**) The orthopedic surgeon creates an osteochondral flap by utilizing a beaver knife. (**c**) The flap is lifted by a chisel (lift). (**d**) The drilling step consists of drilling the bone flake of the fragment with a Kirschner wire and a shaver blade; this so that one promotes revascularization of the subchondral bone. It needs to be mentioned that one has to be careful not to loosen the iatrogenically created osteochondral fragment at its posterior side. (**e**) A 4 mm chisel is utilized to harvest cancellous bone from the distal tibial metaphysis. (**f**) Thereafter, the harvested cancellous bone is transported into the osteochondral defect by an arthroscopic grasper (fill). (**g**) In order to prepare one of the last steps of the procedure (fix), a cannulated system is utilized to perform predrilling and tapping of a compression screw. (**h**) An absorbable screw 1–2 mm recessed relative to the surrounding hyaline cartilage is placed. Due to the diameter and the compression strength, one prefers the non-cannulated screw (*figure reproduced with permission from* Reilingh et al. [33])

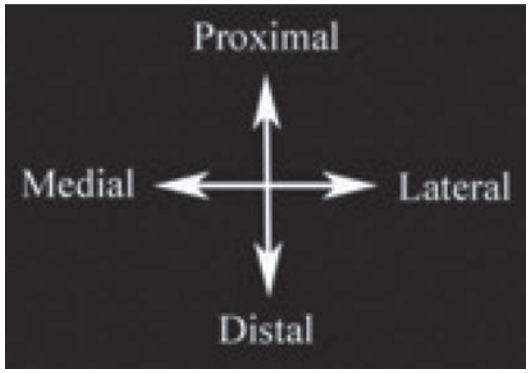

Fig. 13.1 (continued)

The step hereafter is the second step, namely the *drilling* step. During this step, one aims at the promotion of revascularization. The surgeon debrides the osteosclerotic area of the bed and osteochondral fragment (Fig. 13.1d). It is important that the surgeon pays clear attention to debriding and puncturing any subchondral cysts that may be present in selected cases. The subsequent penultimate step of the LDFF procedure is the step during which the debrided and drilled defect will be filled with bone *(fill)*. Cancellous bone is harvested from the distal tibial metaphysis by means of a chisel, after which these harvested bony flakes are transported into the defects by means of a grasper (Fig. 13.1e, f). The last step of the LDFF procedure (*fix*) consists of fixating the fragment that has been created during the first step of the LDFF. A clinically important condition prior to initiating the fixation procedure itself is that the surgeon needs to have achieved a correctly aligned osteochondral fragment. For the fixation procedure itself, Bio-Compression (Arthrex Inc., Naples, USA) or metal screw(s) can be used (Fig. 13.1g, h). Additional bioabsorbable dart(s) or pin(s) can be utilized to prevent rotation.

13.5 Arthroscopic Osteochondral Fragment Fixation: Postoperative Management

A short-leg, non-weight-bearing cast is applied at the operation theatre for a period of 4 weeks postoperatively. When having completed this 4-week period of immobilization, the ankle is placed in a short-leg walking cast in a neutral flexion and neutral hindfoot position—having full weight bearing allowed. One removes the cast at 8 weeks postoperatively. A referral to a physiotherapist for functional physiotherapy is performed in order to help the patient concerning functional recovery and range of motion (dorsiflexion and plantar flexion) exercises. This, so that the patient can progress to full weight bearing in a time frame of about 2 weeks. The important aim of the whole medical team is to supply a well-designed personalized after-treatment protocol in which it is key to focus on balance, proprioception, and ankle functionality. By these means, one can progress to a normal ambulation pattern and achieve full strength as well as propriocepsis. Depending on the patient, running abilities, and personal wishes, as well as sport-specific training, the team can subsequently prepare the patient for a timing of return to sports. It is advised to personalize after treatment after the 3-month period based on the clinical exam and the CT scan of the patient. In general, the patient should be advised to prevent performing any type of activities that consist of peak mechanical forces around the ankle (walking on toes, running, etc.) until after 6 months postoperatively. After these 6 months, the team can gradually start the preparation of return to sports, such as football, running, and other high-impact sports.

13.6 Arthroscopic LDFF: Results

In 2016, the first results of the arthroscopic LDFF procedure were published. This publication consisted of a patient group of seven patients whose clinical and radiological results were analyzed at short term (mean follow-up 12 months, SD 0.6) [32]. The mean preoperative size of the defects was 15.7 mm (SD 3.0) in the anteroposterior direction, 9.6 mm (SD 3.2) in the mediolateral direction, and 6.7 mm (SD 1.4) in the craniocaudal direction. In each and every patient, the LDFF procedure resulted in significant improvements in both American Orthopedic Foot and Ankle Society score (AOFAS) and the numeric rating

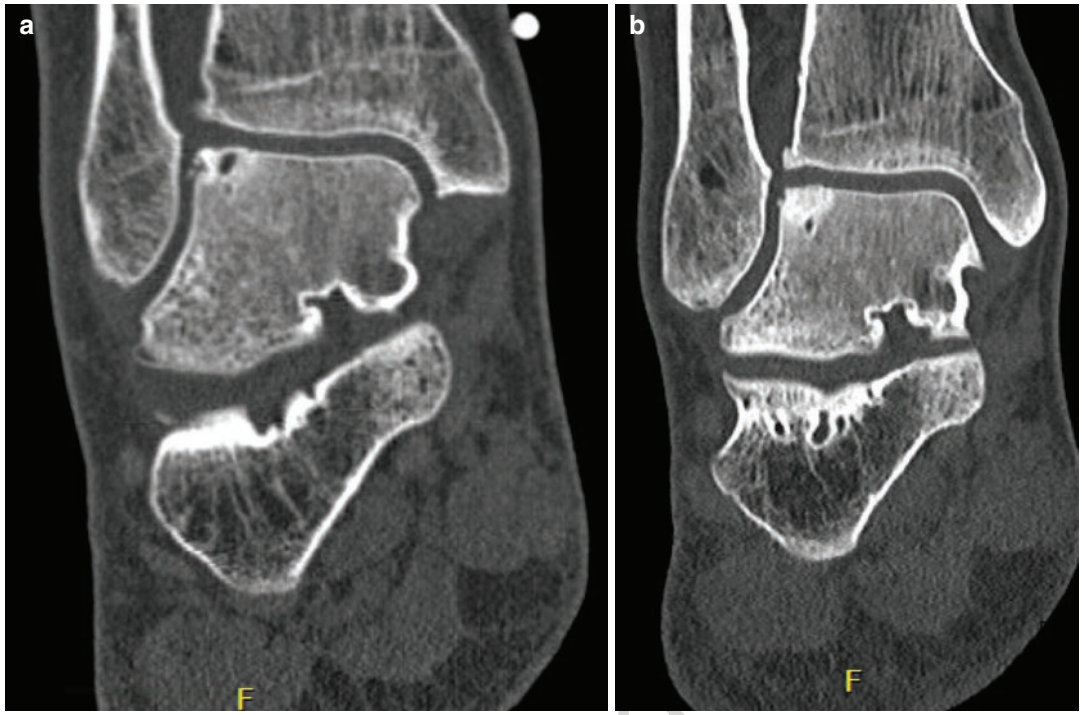

Fig. 13.2 Preoperative and 1-year postoperative computed tomography scans of a patient treated with an arthroscopic lift, drill, fill, and fix (LDFF) procedure. (**a**) Preoperative coronal CT scan. (**b**) 1-year postoperative coronal CT scan

scales (NRS) of pain at rest and during walking [34]. Additionally, all patients reported that they were satisfied about the procedure, and that they would be willing to undergo the surgery once again. Twelve months postoperatively, 71% of the patients showed remodeling and progressive bone ingrowth when assessed on CT scan (Fig. 13.2a, b).

More recently, in 2017, a prospective comparative case series was published by Reilingh et al. [35] and appeared in the KSSTA journal. This study evaluated the clinical and radiological results between arthroscopic LDFF and arthroscopic BMS in primary fixable talar OCDs at 1-year follow-up. Both the LDFF group and the BMS group consisted of 14 patients each. After the arthroscopic LDFF procedure, the AOFAS (preoperative score, 66 (SD 10.1), postoperative score 89 (SD 17.0), ($p = 0.004$)) and the NRS pain at rest (preoperative score 2.1 (SD 1.8), postoperative score 0.9 (SD 1.3), ($p = 0.043$)) as well as when running (preoperative score 7.4 (SD 1.9), postoperative score 2.5 (SD 3.1) ($p = 0.004$)) significantly improved. However, no significant differences were to be found between the arthroscopic LDFF procedure and the arthroscopic BMS procedure preoperatively as well as 1 year postoperatively concerning the functional results being measured by the AOFAS and the NRS. As opposed to the clinical results, there was a significant difference ($p = 0.02$) with regard to healing of the subchondral bone plate between both arthroscopic treatment groups. From the 14 patients that had undergone the arthroscopic BMS procedure, 11 patients were observed to have a depressed subchondral bone plate. Three of the fourteen actually contained a flush subchondral bone plate. On the contrary, 10 out of 14 patients in the arthroscopic LDFF group had a flush subchondral bone plate, and 4 had a depressed subchondral bone plate. Union of the osteochondral fragment was found in nine patients after arthroscopic LDFF.

In November 2017, 75 international experts in cartilage repair of the ankle representing 25 countries and 1 territory were convened and participated

in a process based on the Delphi method of achieving consensus at the International Consensus Meeting on Cartilage Repair of the Ankle in Pittsburgh. One of the working groups concerned the treatment of osteochondral defects of the ankle by internal fixation [28]. The statements derived from the whole process indicated that a bone marrow stimulation procedure can be performed in the case of surgical treatment failure by internal fixation in lesions smaller than 15 mm in diameter. However, the authors concluded that there is no indication to perform fixation after a prior bone marrow stimulation procedure. Furthermore, it was stated by the consensus group that fixation techniques for osteochondral defects of the ankle are likely to facilitate healing of the cartilage and/or subchondral bone. Therefore, satisfactory clinical results can be expected when the right type of lesion is chosen for arthroscopic fixation.

13.7 Conclusion

Despite these clinical and radiological results demonstrating that the arthroscopic "Lift, Drill, Fill, and Fix" procedure for primary large and fixable talar osteochondral defects is a highly clinically promising surgical intervention, longer follow-up times are certainly required. A greater cohort of patients needs to be included for a larger statistical power, and it is highly important to assess the outcomes of the arthroscopic LDFF procedure in a prospective comparative randomized manner. Furthermore, it is of clinical importance that in case of clinical failure after an arthroscopic LDFF procedure, alternative surgical interventions (i.e., BMS and osteo(chondral) transplantations) are still possible.

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14 One-Stage Treatment for Osteochondral Lesion of the Talus

Bogusław Sadlik, Alberto Gobbi, Karol Pałka, and Katarzyna Herman

14.1 Cartilage Restoration Considering Ankle Joint Congruency

In order for weight-bearing joints to remain functional and avoid premature failure, the articulation should meet several criteria that are consistent with mechanical laws. From a mechanical point of view, two weight-bearing surfaces that are moving relatively to each other should articulate over the functional range with the smallest frictional forces possible in order to minimize trauma to the opposing surfaces and avoid overheating. The second important criterion is the optimization of joint contact surface area. Although a smaller surface area of articulation may allow for a reduction in frictional forces, a larger surface area will decrease pressure and peak loads on the weight-bearing surfaces; these are important factors to minimize the destructive mechanical forces that lead to progressive degenerative joint injury. Given that the ankle joint is the most dynamic human weight-bearing joint, it is crucial that there is proper matching of adjacent articular surfaces over the full range of motion.

The ankle is characterized by higher congruence than the knee with thinner cartilage, and this is why it requires much more precision in chondral surface reconstruction. Surgical treatment of osteochondral lesions of the talar dome (OLT) aims to restore layers of the defect using biological material that undergoes further remodeling and integration with the surrounding tissue. The purpose of the reconstruction is to effectively recreate the shape of the talar dome in every location, especially on the medial edge, where the majority of traumatic lesions are located [1].

Considering the mechanical and geometric components of joint function, restoration of the articular cartilage surface after chondral injury is not complicated if subchondral bone remains intact and anatomically unaltered. In cases of subchondral osteophytes, restoration of anatomic surface geometry is accomplished with a shaver or burr that can be used to mill the subchondral bony protuberance. In cases of large osteochondral defects involving deep areas of subchondral bone deficiency, careful reconstitution of the bone deficit is needed. Special attention must be paid to restore the natural subchondral surface geometry, necessary for optimal adjacent cartilage regeneration.

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Until now the most commonly used single-stage treatment for large osteochondral lesions of the talar dome was OAT with a graft harvested from the knee [2–5]. Unfortunately, that technique may cause symptoms related to donor site morbidity in the knee after osteochondral autograft harvesting [6]. Moreover, the osteochondral graft harvested from the knee rarely restores the talar surface properly, especially in terms of its curvature and the joint congruence. Some authors have reported incomplete integration of the OAT graft with surrounding tissues as well as bone plug necrosis [7].

Biological scaffolds are frequently used to restore chondral tissue and can be implanted as cell-free or cell-embedded scaffolds. Second- or third-generation autologous chondrocyte implantation procedures have been developed to provide cartilage restoration in treatment of significant chondral injury. In cases of cartilage injury that are associated with significant subchondral bone loss, a dual-layer restoration procedure may be used, originally described by Peterson in 2003 as a "sandwich" technique.

Of the available techniques described, the dual-layer, cell-based technique has the greatest potential to restore articular congruity. This is achieved through the surgical contouring of the restored osteochondral surface to match the native radius of curvature and the postoperative plastic adjustments that inherently occur as result of the forces from the opposing articular surface. Additionally, progress in biomaterial engineering has allowed for development of three-dimensional scaffolds that are more malleable and therefore more stable within chondral defects, as opposed to periosteal tissue that was used by Petersen in the original method. Another important advancement in cell-based cartilage repair is the elimination of the two-stage ACI procedure. The use of autologous bone marrow aspirate concentrate in conjunction with biological scaffolds, as described by Gobbi [8, 9], has been introduced widely into clinical practice and is performed as a one-stage procedure at considerably reduced cost compared to autologous chondrocyte procedures. Bone layer of the defect is usually restored by a calcaneus bone plug taken from iliac crest or bone chips compacted into the defect before covering its surface with a chondrogenic matrix [10–12].

14.2 Single-Stage Surgical Treatment of the Osteochondral Lesion

Recent advances in arthroscopic instrumentation have enabled the provision of minimally invasive procedures to treat chondral and osteochondral injury by a one-stage, single- or dual-layer, cell-based reconstruction techniques [13]. These developments in instrumentation and biomaterials have greatly reduced the need for aggressive, open procedures in the treatment of chondral and osteochondral defects.

One of the examples of new one-stage procedures is the arthroscopically assisted approach in surgical repair of osteochondral lesions of the talus using biological inlay osteochondral reconstruction (BIOR) (Fig. 14.1) [14]. In our opinion, successful repair of the deeper osteochondral lesions of the talar dome requires a separate restoration of the bone layer and chondral layer. Filling of the lesion should be adapted to the shape of the curvature of the talar dome in the same way as a dentist molds a tooth filling. The bone plug filling of the defect should be formed and suitably concentrated, to carry the joint preload without the risk of subchondral layer collapse. Due to the limited accessibility to the articular surface of the talar bone, BIOR implantation can be made only through three minimally invasive portals. The classic approach through a medial malleolar osteotomy is advised in cases of larger lesions situated in the talar dome center. Due to a narrow joint space and deep location of the lesion, this arthroscopy assisted and minimally invasive approach may only be performed with the help of a skillful assistant surgeon.

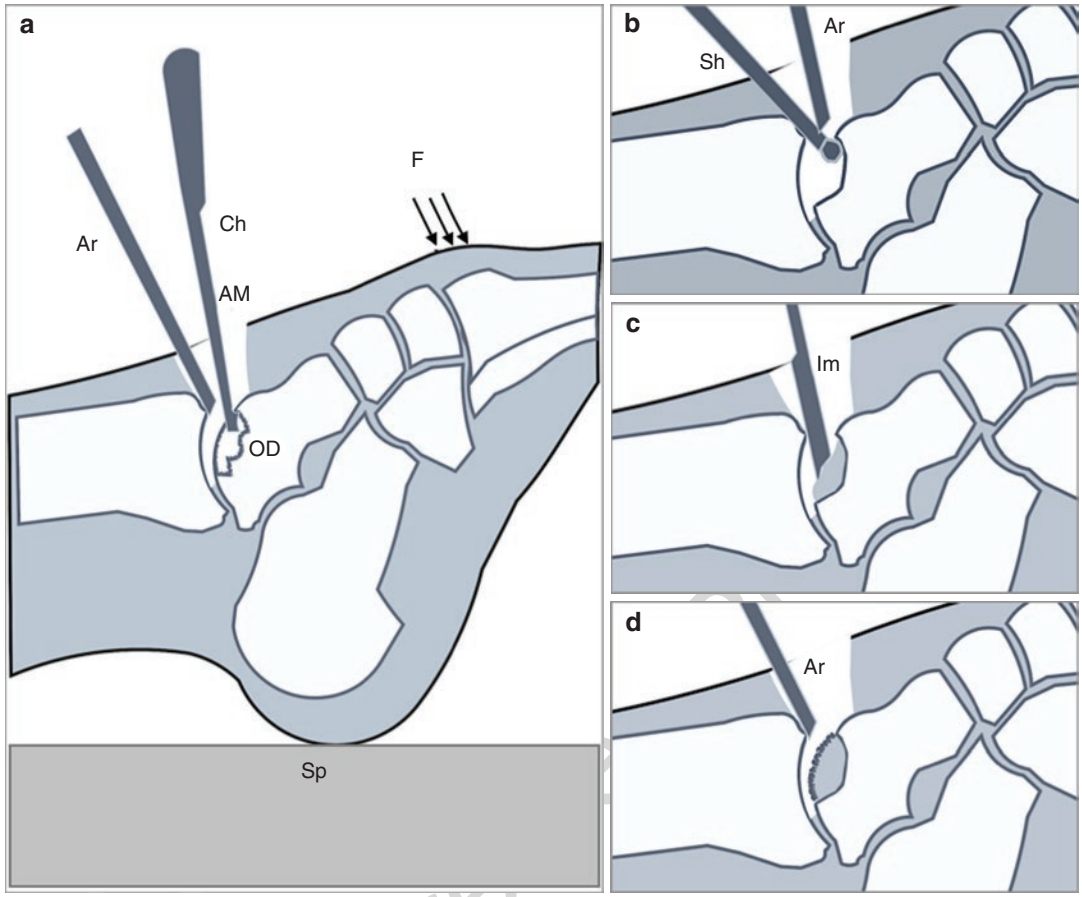

Fig. 14.1 Minimally invasive anteromedial approach to the talar dome with an arthroscope visual assistance for osteochondral defect biological inlay reconstruction (BIOR): AM, anteromedial approach; *Ar* arthroscope, *Ch* chondrectome, *F* force manually applied by an assistant, *OD* osteochondral defect, *Sp* heel support point, *Sh* "burr"-type shaver, *Im* barrel implantation device for bone chips. Biological Inlay Osteochondral Reconstruction step by step: (**a**) osteochondral defect removal using chondrectome or curette; (**b**) refreshing sclerotic bone plate on the bottom and walls of the defect; (**c**) bone chips inlay implantation using barrel implantation device; (**d**) bone chips inlay covered with matrix immersed with BMAC

14.3 Approaches to the Talar Osteochondral Lesion

An arthroscopic approach is useful in the cases of shallow osteochondral or chondral lesions treated by defect debridement and bone marrow stimulation technique. In favorable conditions, an arthroscopic matrix implantation might be achievable, although a minimally invasive open technique is much easier for that purpose. Osteochondral reconstruction of a deep lesion needs to be performed with an open approach.

14.3.1 Anteromedial Approach

The anteromedial approach that requires skin incision from 3 to 4 cm long directly above the joint line, medially to the tibialis anterior tendon, is the most often used technique for addressing lesions localized on the anterior and central surface of the medial talar dome. The patient is positioned supine, as for standard ankle arthroscopy. Up to 50% of the medial talar dome surface can be visualized arthroscopically after synovium removal and excessive plantar flexion of the foot.

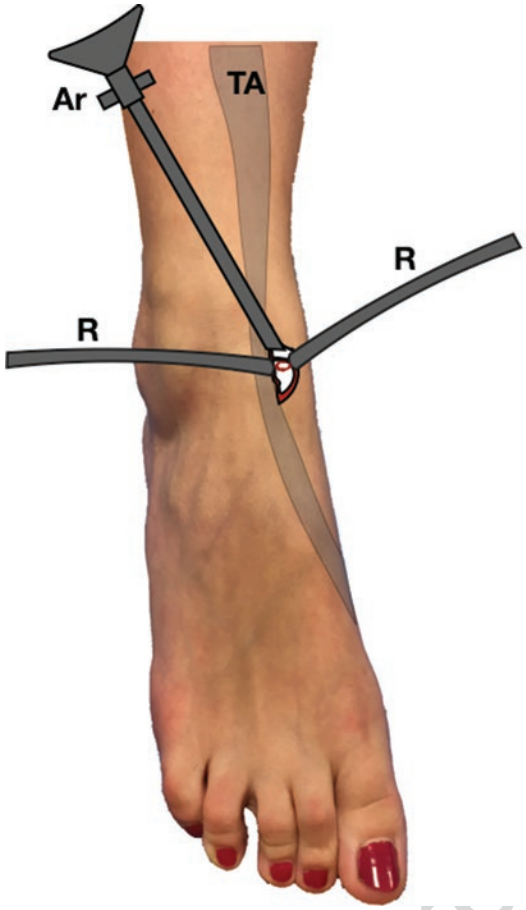

Fig. 14.2 Anteromedial arthroscopic approach to the ankle joint. *Ar* arthroscope, *R* retractor, *Ta* tibialis anterior tendon

Treatment of the lesion and implantation in such a narrow space is possible with the assistance of an arthroscope, controlled by an assistant, while the work of the second assistant should be focused on maneuvering the foot (Fig. 14.2).

14.3.2 Anterolateral Approach

In the case of a lesion localized in the anterolateral part of the talus, an analogous technique may be applied from the anterolateral approach. It is performed on the anterior edge of the lateral malleolus with the retraction of the third tendon of the sagittal muscle in the medial direction. Also in this situation, two assistants are required to perform the implantation. Special attention must be paid to not damage the intermediate dorsal cutaneous nerve, which should remain intact either on the lateral or medial side of the incision (Fig. 14.3).

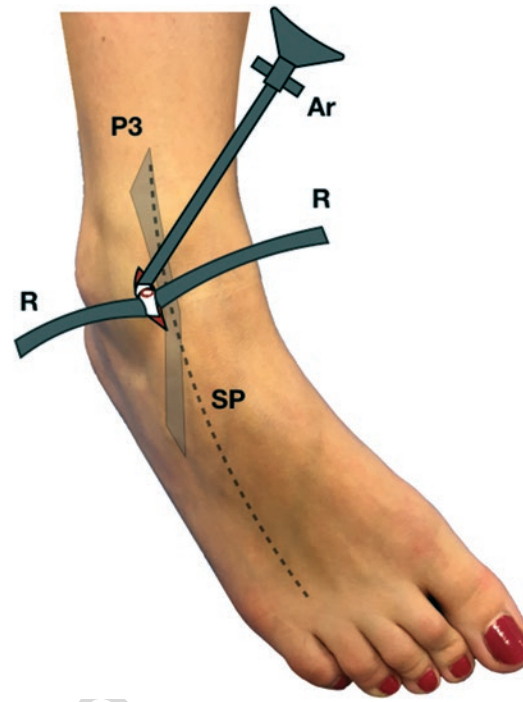

Fig. 14.3 Anterolateral approach to the ankle joint. *Ar* arthroscope, *R* retractor, *P3* peroneus tertius tendon, *SP* superficial peroneal nerve

14.3.3 Posterolateral Approach

If the defect is located in the lateral part, it is accessible from an incision located on the posterolateral side of the joint, near a lateral border of the Achilles tendon. The incision is made at the level of a standard posterolateral portal; the cut should be extended by 1.5 cm proximally and 1.5 cm distally. The patient is placed on their side, and the operated limb is turned upward with knee bent to 90°. This position relaxes the tension of the calf muscles and allows the Achilles tendon to be pulled toward the medial side. The dorsal flexion of the foot held by the assistant makes it possible to visualize 20% of the surface of posterolateral edge of the talus. Also here, the use of

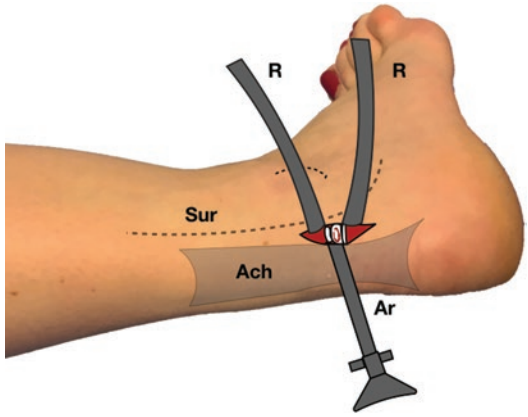

Fig. 14.4 Posterolateral approach to the ankle joint. *Ach* Achilles tendon, *Ar* arthroscope, *R* retractor, *Sur* Sural nerve

an arthroscope to visualize the operating field is necessary (Fig. 14.4).

14.3.4 Standard Approach Through the Medial Malleolus Osteotomy

Approach to the lesion located in the posteromedial-central part is possible only through the osteotomy of the medial malleolus, which is the most common and well-known approach, used and described by many authors [1, 3, 5].

14.4 Postoperative Management and MRI Monitoring

Deep osteochondral defects of the talus are more often seen at the outpatient clinic than chondral ones, which require only bone marrow stimulation technique or chondral scaffold implantation. Deep osteochondral defects are much more demanding and should be treated with surgical techniques considering reconstruction of both bone and chondral layers of the defect. Expected time of graft remodeling and healing is longer when compared with treatment of a chondral defect. Thus the type of surgical reconstruction method implies specific postoperative treatment and what is more important the rehabilitation protocol should be individually modified. In our opinion, the best way to properly control the osteochondral graft maturation is periodically checking of the graft status. In our center, after undergoing osteochondral regeneration procedures, patients are followed up with a monitoring MRI protocol, 6 weeks then 6 and 12 months postoperatively. Depending on the bone and subchondral lamina quality, patients are allowed more or less physical activity. Slow maturation process of the graft indicates a modified pharmacotherapy or/and physiotherapy.

The rehabilitation protocol after biologic surgical treatment of osteochondral injury is based on the size and location of the osteochondral defect and the contact angle (CA). CA is the angle of the reconstructed articular surface that stays in contact with the opposite articular surface during ankle movement. This crucial information allows the physiotherapist to determine a safe ROM in exercise progression.

In the authors' experience, the individual rehabilitation strategy should be planned carefully, taking into consideration these three key issues:

- Restricted joint motion in the initial phase of graft integration (first 7–10 days), in order to allow graft integration and the formation of a fibrous hematoma on its interface, and then progressively increasing joint motion up to full range, applying passive mobilization with the joint distraction.
- MRI graft maturation monitoring at 3 or 6 weeks and then 6 and 12 months after the surgery.
- Orthopedic equipment should be individualized, depending on the size, location, and CA of the osteochondral reconstruction.

In all cases, the rehabilitation process should be modified depending on the joint status as swelling, adhesion, additional procedures or injures, as well as MRI assessment.

In the first 7–10 days, we recommend limited joint motion, in order to encourage successful

integration of the repair tissue and the formation of a fibrous hematoma. After this period, range of motion exercises should be undertaken in conjunction with joint distraction. Partial weight-bearing should begin 4 or 5 weeks after surgery, with expected unrestricted weight-bearing by weeks 6, 7, or 8, depending on MRI assessment at week 6. It is important for a physiotherapist to understand that a predefined ROM is necessary to restore the anatomic curvature of the talus. To optimize postoperative monitoring of the healing process and formation of repair tissue, it is recommended that patients undergo MRI at 6 and 12 weeks after surgery. At 3 months, patients progress to straight-line running, with an emphasis on strength, endurance, and aerobic training. Sport-specific training typically begins at 8 months, with expected return to competition by 10 months postoperatively.

Most of the rehabilitation centers use standard postoperative rehabilitation protocols after ankle osteochondral lesion surgical treatment. Management can be various, depending on lesion size and localization, comorbidities, and patient age. The late postoperative management, considering various physical activities of the patients, should be administered with functional tests and graft maturation rate in MRI. Various graft maturation dynamics in MRI assessment can be seen (Figs. 14.5, 14.6, 14.7). There is noticeably slower graft rebuilding rate in older patients. The biological osteochondral reconstructions of the talar dome seem to be slower in maturation comparing to the knee. There is no universal postoperative protocol after osteochondral reconstruction, due to the fact that biological healing of the graft is not well defined and uncontrolled in vivo.

14.5 Summary

The treatment of cartilage injury associated with significant subchondral bone loss with the arthroscopic BIOR technique enables reconstruction of damaged osteochondral tissue and restoration of the natural anatomic contour of the articular surface, in a minimally invasive fashion. The one-step cell-based cartilage technique of HA-BMAC has been used at our institutions with success using both open and arthroscopic methods to treat cartilage defects of various dimensions and also multi-compartmental knee cartilage injury. The arthroscopic BIOR technique combines HA-BMAC cartilage repair with a malleable bony inlay to provide a bilayer autologous reconstruction of the osteochondral unit, with minimal morbidity.

The biological inlay osteochondral reconstruction technique of osteochondral repair has the capability to treat a wide range of lesion sizes, with various depths of subchondral bone loss. In addition, lesions of irregular shape may be repaired without sacrificing healthy adjacent tissue, as opposed to reconstruction procedures that involve circular-shaped osteochondral grafting. Furthermore, while osteochondral autograft or allograft procedures require graft implantation from a near-90° approach, the BIOR technique may be used to restore the natural anatomic radius of articulating surface curvature, from a wide variety of angles. This single-stage, dual-layer, cell-based cartilage repair procedure with bony inlay is a versatile technique that has an attractive cost profile and may be used in minimally invasive fashion for a variety of joint cartilage injuries that involve subchondral bone deficiency.

Biological materials, such as bone autograft, bone marrow concentrate, fibrin glue, and collagen matrix have been used in orthopedic surgery for many years. The presented modified surgical "sandwich" technique allows the talar convexity to be precisely recreated to match the anatomic radius of curvature of the articular surface. Furthermore, the reconstruction is performed as a one-step procedure. In the 4-year follow-up of our 22 patients, none of the cohort required revision surgery. Except for one patient, all were satisfied with the outcome. Postoperative MRI examinations typically demonstrated good quality repair tissue. A notable drawback of this surgical technique was a need to perform a medial malleolar osteotomy in a substantial number of cases (10 of 22 patients), which theoretically may increase procedure morbidity.

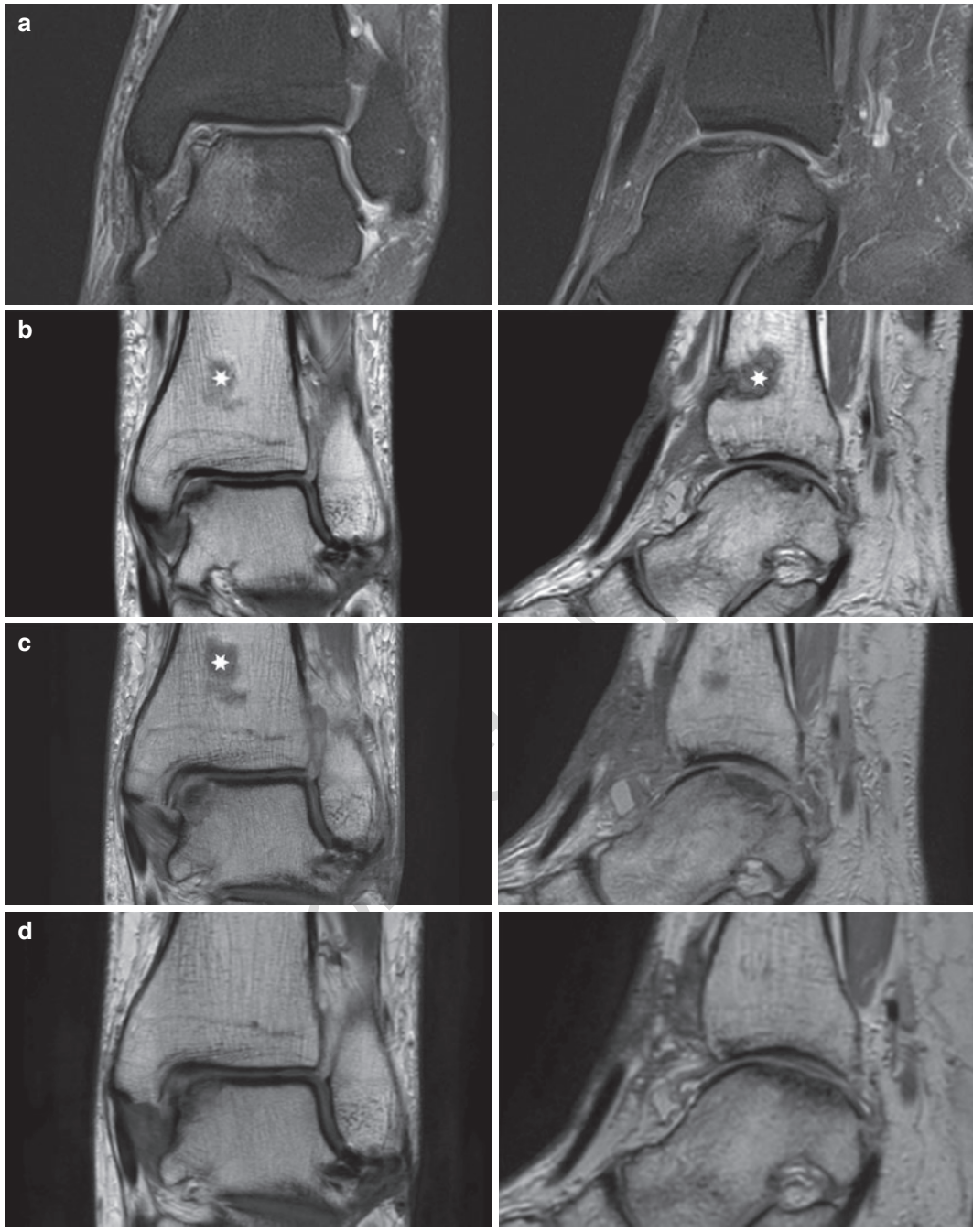

Fig. 14.5 An example of slow remodeling of the biological inlay of medial talus. MRI evaluation of the left ankle of a 48-year-old female regarding the stepwise remodeling of the subchondral lamina and chondral surface: (**a**) osteochondral defect grade III of the medial aspect of the talar dome, preoperatively; (**b**) biological osteochondral inlay (asterisk indicates donor site of a spongiosa bone graft), 3 months postoperatively; (**c**) 6 months postoperatively, a border of subchondral lamina and chondral surface are clearly visible, bone edema slightly decreased; proton density (PD) with or without fat saturation (FS) (m-SPIRE, 3.0 Tesla digital scanner) and sagittal and coronal scans shape of the talar dome properly formed (3 months postoperatively); (**c**) still proper shape of the talar dome, subchondral lamina not visible yet (12 months postoperatively); (**d**) subchondral lamina and chondral layer visible (24 months postoperatively); PD (proton density) with or without fat saturation (m-SPIRE, 3.0 Tesla digital scanner); sagittal and coronal scans

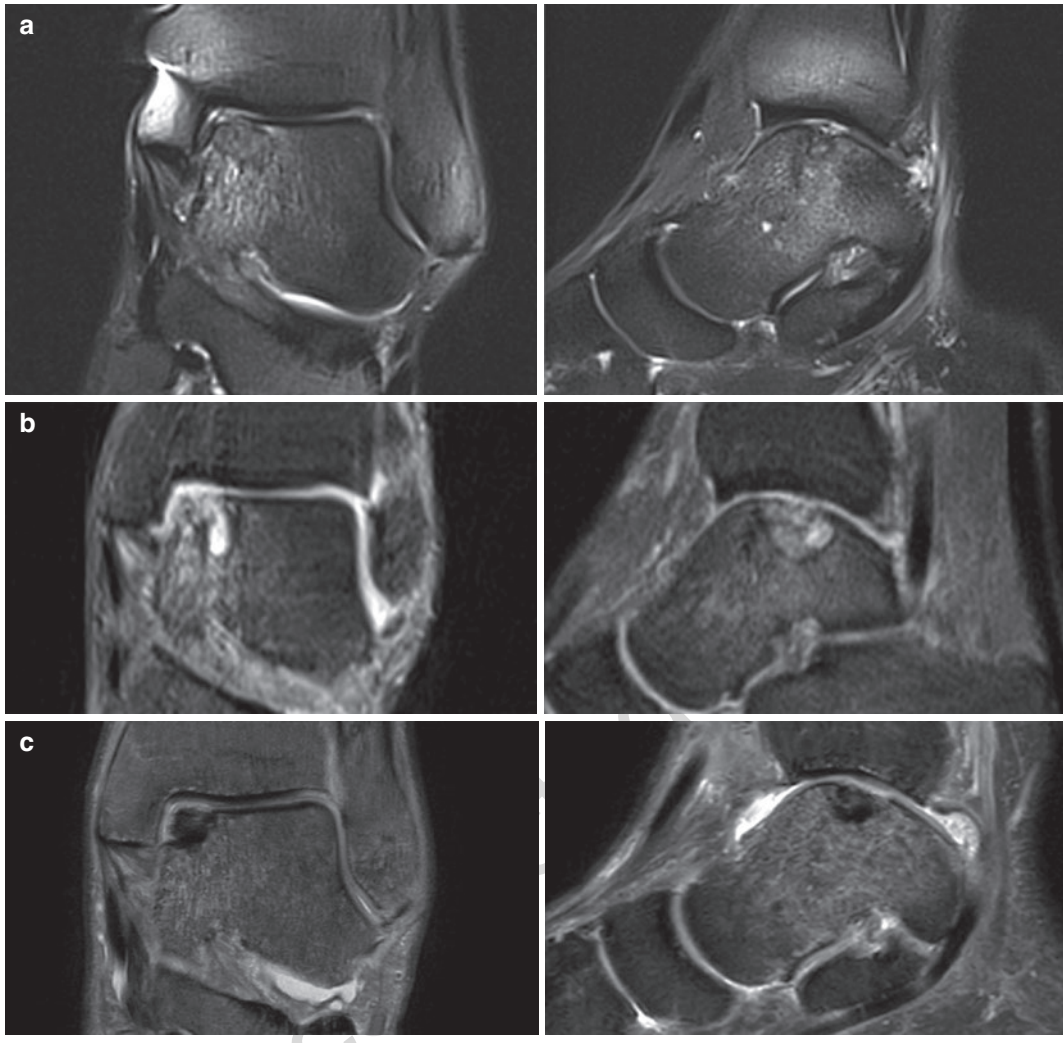

Fig. 14.6 Natural history of OLT: (**a**) the first MRI at the beginning of the ankle pain (2 years before surgery), only chondral lesion and subchondral bone edema can be seen on the medial boulder of the talus; (**b**) MRI scans 2 months before surgery, chondral lesion and edema extended, and several pseudocysts appeared in the region of talar edema. MRI 2 months after OLT reconstruction with BIOR technique; (**c**) talar dome curvature and structure were restored; PD (proton density) with or without fat saturation (m-SPIRE, 3.0 Tesla digital scanner) and sagittal and coronal scans

Currently, all surgical techniques for reconstruction of large osteochondral lesions of the talus require an approach that provides perpendicular access to the articular surface, thereby allowing the implantation of bone blocks, osteochondral grafts, or synthetic scaffolds. Moreover, there is less tolerance of articular incongruity in the ankle joint compared to the knee, and so surgical techniques to treat articular injury are more demanding. In our opinion, the focus of future treatments of osteochondral lesions should be to develop minimally invasive, or even arthroscopic, techniques that are appropriate for routine use. Such techniques would enable the restoration of anatomic articular congruence within the ankle joint, while minimizing postoperative morbidity. It should be specifically focused on the development of a technique that avoids the malleolar osteotomy, which remains a disadvantage of current regenerative surgical methods.

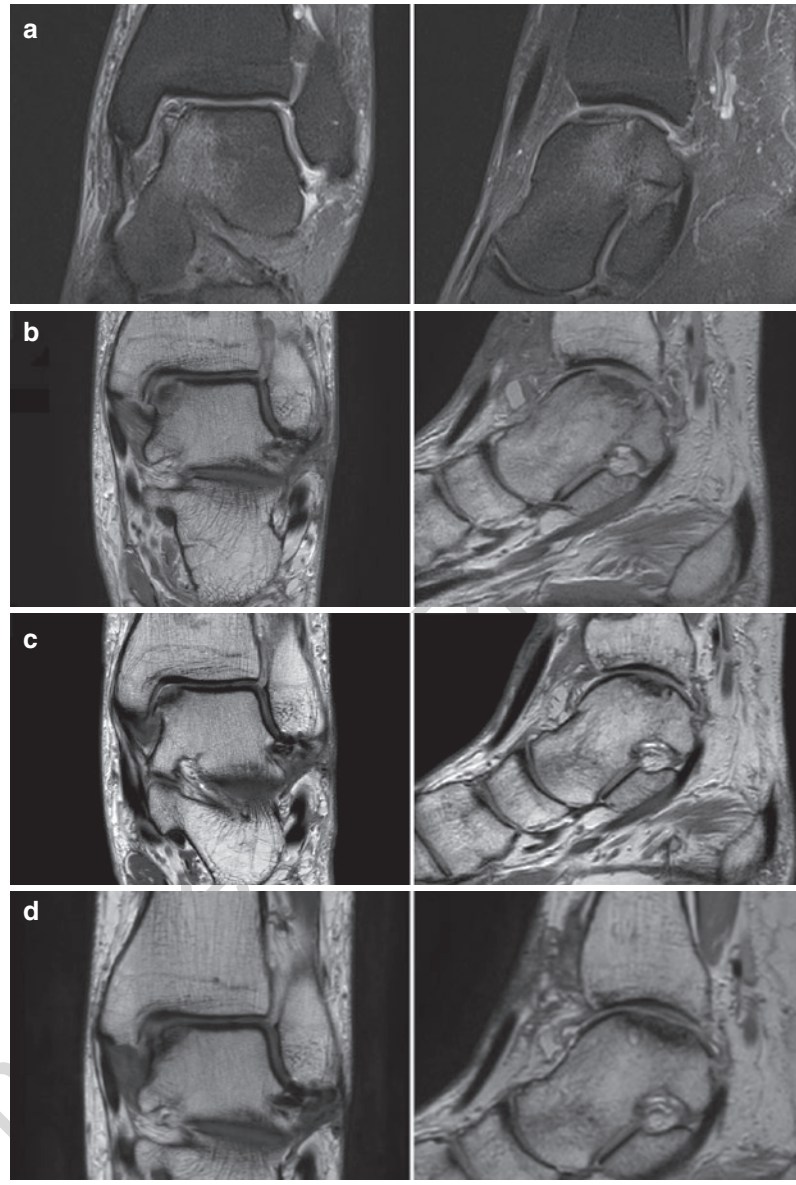

Fig. 14.7 A 45 year old female treated for OCD of the talus - BIOR procedure was performed through miniarthrotomy from antero-medial approach, additionally ATFL and CFL reconstruction was done. MRI scans show the lesion preoperatively (**a**), and a slow maturation process of the graft: 1 month postoperative (**b**), 3 months postoperative (**c**) and 18 months postoperative (**d**)

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Part III

Bone and Joint Injuries

15 Ankle Fractures

Shinji Isomoto, Kazuya Sugimoto, and Yasuhito Tanaka

15.1 Diagnosis

Dislocation fractures are easily diagnosed by plain radiographs. After reduction, medial clear space and widening of the distal tibiofibular joint should be checked. These findings indicate rupture of the deltoid ligament and the distal tibiofibular ligaments (Fig. 15.1). For this evaluation, comparison with the opposite ankle is recommended. Tibiofibular injuries are often concomitant with proximal fibular fractures. If a fibular fracture is not shown on the ankle radiographs, the radiographs should be checked for a proximal fibular fracture.

Computed tomography provides more information about sagittal dislocation of the tibiofibular joint, rotation of the distal fibula, dislocation of intra-articular fragments, and free bodies in the joint, etc. (Fig. 15.2).

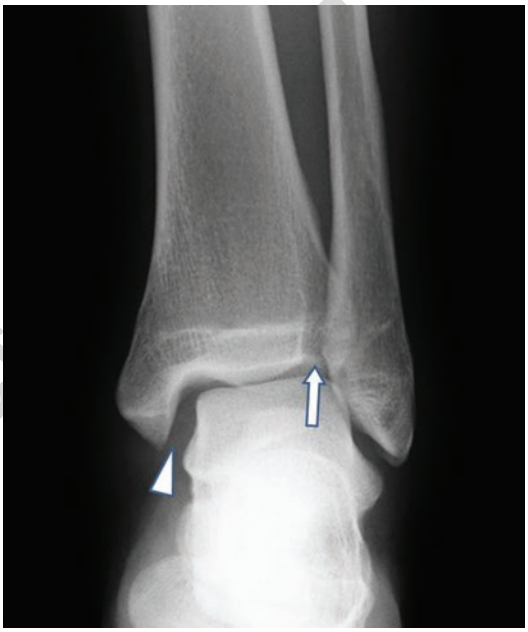

Fig. 15.1 Plain radiograph of the ankle with a fibular fracture, syndesmosis, and deltoid ligament injury. The medial clear space (arrow head) and widening of the distal tibiofibular joint (arrow) are shown

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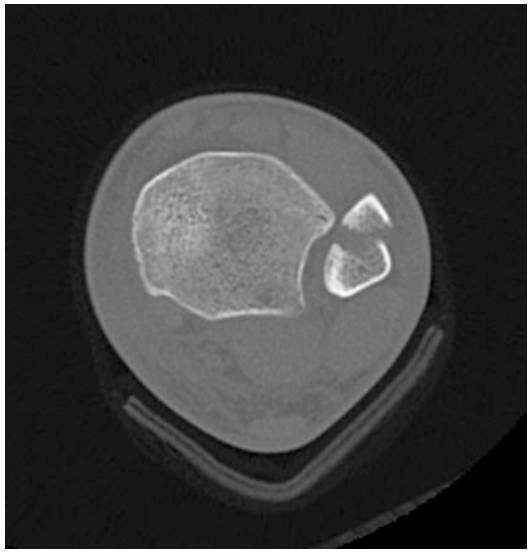

Fig. 15.2 Computed tomography of the distal tibiofibular joint. Sagittal dislocation of the tibiofibular joint is shown

15.2 Primary Care

After manual reduction, the ankle is fixed by a cast or splint. Open fractures or unstable fractures are fixed by external fixation, and skin damage, neurovascular injuries, and compartment syndrome are evaluated. Because such damage worsens over time, care must be taken over the following days. If skin damage or swelling is severe, early surgery has a high risk of complications. Surgery should be performed after swelling is decreased.

15.3 Surgical Procedure

15.3.1 Patient Position

The lateral decubitus position is used. If treatment for the medial malleolus or deltoid ligament is needed, the patient's position is changed to the supine position.

15.3.2 Arthroscopy

Distraction is not used. Anteromedial and anterolateral portals are used. Ligament injuries, fracture, dislocation, and instability are evaluated [1, 2]. Chondral damage, osteochondral damage, and free bodies are identified and evaluated. Free bodies are removed. Chondral defects without bleeding are treated with bone marrow stimulation by the microfracture technique (Fig. 15.3) [3].

15.3.3 Open Reduction and Internal Fixation of the Fibula

The lateral approach is commonly used. If access to the posterior malleolus is required, the posterolateral approach is used. In the lateral approach, a longitudinal lateral incision is made. At the proximal part of the incision, the superficial peroneal nerve lies anteriorly. It should be identified and protected. Fractures are reduced and fixed with a plate and screws (Fig. 15.4). In a comminuted fracture, care is taken to avoid malreduction with shortening and external rotation.

15.3.4 Open Reduction and Internal Fixation of the Posterior Malleolus

Open reduction and internal fixation of the posterior malleolus are needed if the gap or step off of the articular surface is large [4]. The posterolateral approach is used [5]. A skin incision is made between the posterolateral border of the fibula and the medial border of the Achilles tendon. The distal part of the incision is curved to the tip of the fibula to expose the fibular fracture. The sural nerve is identified and protected. The peroneal tendons are retracted laterally. In most cases, the posterolateral fragment of the tibia is exposed after the flexor hallucis longus is retracted medi-

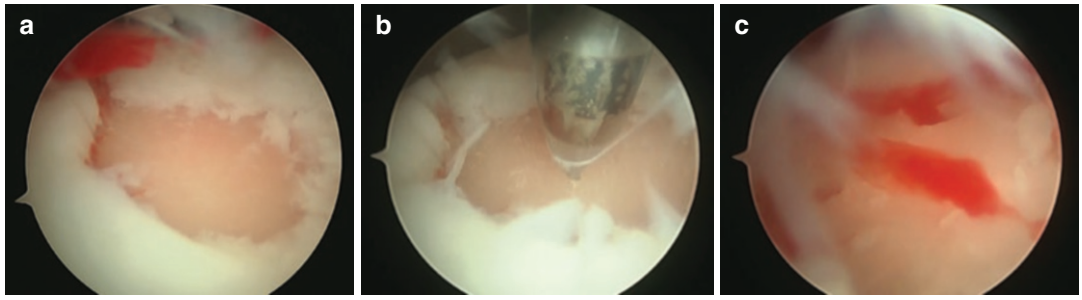

Fig. 15.3 Chondral lesion of the talus. The chondral lesion without bleeding (**a**) is penetrated by a pick (**b**). The bone marrow from the subchondral bone is shown (**c**)

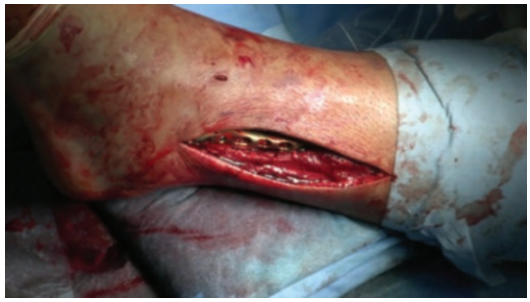

Fig. 15.4 Plate fixation of the fibular fracture

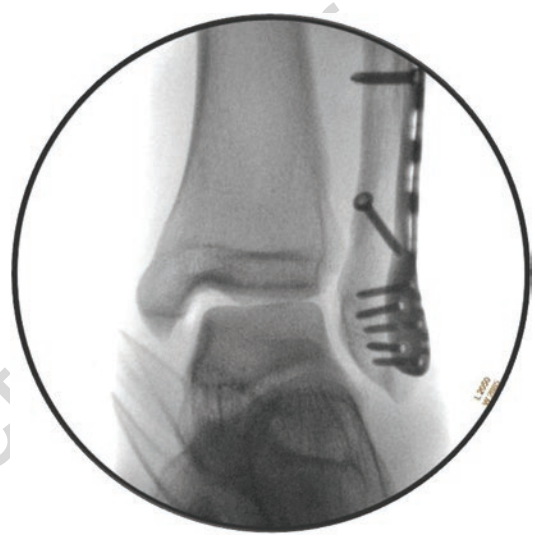

Fig. 15.5 Fluoroscopy of the ankle after fixation of the fibular fracture. Syndesmosis widening and the medial clear space remain, and instability of the tibiofibular joint is evident on the external rotation stress test

ally. The fracture line is identified and reduced directly. The periosteum and the attachment of the posterior tibiofibular ligament are preserved. The fragment is fixed with cannulated cancellous screws. The first screw is inserted beneath the tibial plafond. One or two screws are added according to the size of the fragment. A plate is used if the fragment of the posterior malleolus is large enough.

15.3.5 Syndesmosis Fixation

Syndesmosis instability is checked by fluoroscopy after fixation of the fibular fracture (Fig. 15.5). To identify instability, adequate external rotation stress is applied. If instability is evident, syndesmosis fixation is needed. Syndesmosis screws have been commonly used to fix syndesmosis injuries. Syndesmosis screws should be removed before weight-bearing or training to avoid breakage of the screws. Widening of the syndesmosis sometimes happens after removal of the screws. Recently, suture buttons have been used for syndesmosis injuries with good results [6]. Suture buttons have a lower rate of hardware breakage than syndesmosis screws, so athletes can return to sports activities with suture buttons. In this chapter, fixation with a suture button is shown.

If the tibiofibular joint is severely dislocated, it is reduced with a clamp. A guide wire is inserted from the posterolateral fibula to the anterolateral tibia. In a distal fibular fracture, a guide wire is inserted through one of the holes of the distal fibular plate (Fig. 15.6a). When there is no fracture of the fibula, or the proximal fibular fracture is not fixed, two sets of suture buttons are used.

Drilling is performed by a cannulated drill bit from the fibula to the tibia (Fig. 15.6b). The suture button is inserted, and the medial button is seated on the medial cortex of the tibia. The wire is tightened, and the lateral button is seated on the lateral cortex of the fibula or plate. The wire is tightened until there is sufficient tension (Fig. 15.6c).

15.3.6 Suture of the Deltoid Ligament or Fixation of a Medial Malleolar Fracture

Surgery for the deltoid ligament or a medial malleolar fracture is performed with the patient in the supine position. An ankle dislocation fracture in athletes is commonly accompanied by deltoid ligament rupture. Suture for the deltoid ligament rupture is controversial [7, 8]. The medial clear space of the medial gutter is closed after the syndesmosis is fixed appropriately. In cases where a medial clear space or talar tilt remains, the deltoid ligament is sutured [9, 10].

A medial longitudinal incision through the tip of the medial malleolus is used. The superficial deltoid ligament is exposed. A longitudinal incision of the superficial deltoid ligament is made to expose the deep deltoid ligament. The rupture of the deep deltoid ligament is identified and sutured (Fig. 15.7). In avulsion of the deltoid ligament from the medial malleolus, a suture anchor is used. The superficial layer is sutured after the deep layer is repaired.

15.3.6.1 Final Assessment

After suture of the deltoid ligament, reduction and instability are checked by fluoroscopy. The syndesmosis suture wire is tightened again if needed (Fig. 15.8).

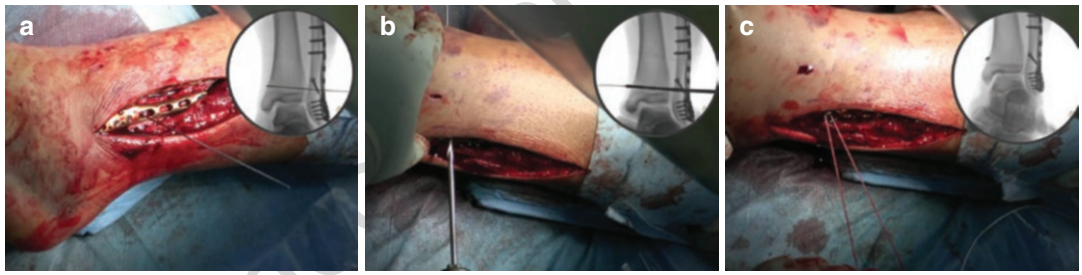

Fig. 15.6 Syndesmosis fixation with a suture button. (**a**) The guide wire is inserted in one of the holes of the fibular plate. (**b**) Drilling is performed by a cannulated drill bit from the fibula to the tibia. (**c**) The suture button is inserted, and the medial button is seated on the medial cortex of the tibia. The wire is tightened, and the lateral button is seated on the fibular plate

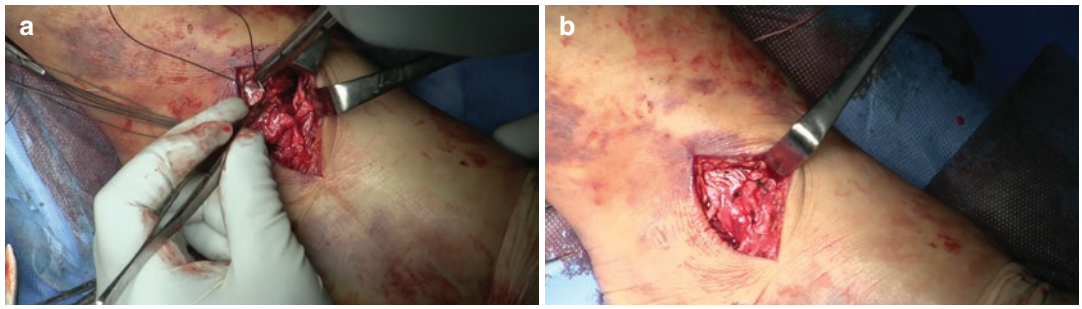

Fig. 15.7 Suture of the deltoid ligament. (**a**) Suture of the deep layer. (**b**) After suture of the deltoid ligament

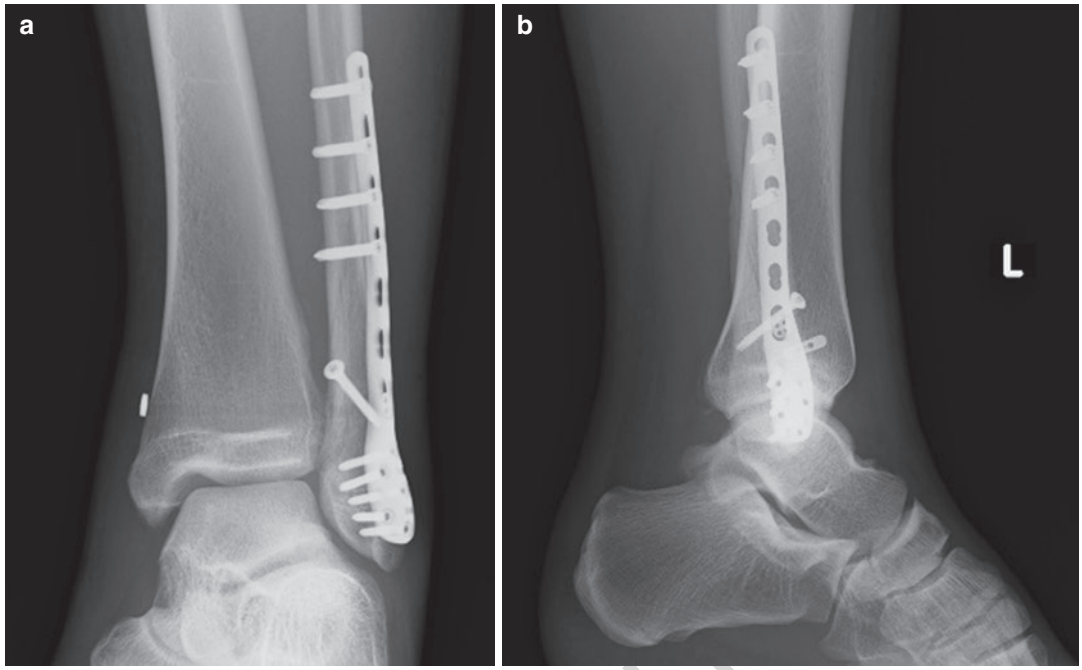

Fig. 15.8 Postoperative anteroposterior radiograph (**a**) and lateral radiograph (**b**)

15.4 Postoperative Treatment

The ankle is fixed in a cast for 3 weeks. Dorsiplantar flexion exercise and partial weight bearing are started 3 weeks after the operation with a semirigid brace. Inversion-eversion exercise and full weight-bearing are started 6 weeks after the operation, and physical training is started after bone union.

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16 Ankle Fractures and Return to Sports in Athletes: "Does Arthroscopy Add Value to the Treatment?"

Pieter D'Hooghe, Fadi Bouri, Akis Eleftheriou, Thomas P. A. Baltes, and Khalid Alkhelaifi

16.1 Introduction

The ankle is one of the most commonly injured joints in sports—ankle injuries constitute 12–23% of all injuries recorded during FIFA competitions. Although the incidence of ankle fractures in athletes is low, accounting for less than 3% of all ankle injuries [1, 2], the severity of this injury warrants meticulous treatment [1]. Throughout the past decades open reduction and internal fixation (ORIF) has established itself as standard of care for unstable ankle fractures. However, ORIF is associated with substantial surgical exposure and inherent complications, such as infection and skin necrosis. With the aim to minimize complications and further improve outcomes, arthroscopic reduction and internal fixation (ARIF) and arthroscopy-assisted open reduction internal fixation (AORIF) were introduced [3].

Potential advantages of arthroscopic treatment of ankle fractures include [4]:

- Limited surgical exposure and soft-tissue trauma
- Video-assisted fracture reduction
- Direct visualization of the joint articulation
- Evaluation of ligamentous injuries and associated intra-articular pathology (e.g., osteochondral injuries)

As ankle fractures constitute a major time-loss injury in athletes, treatment should address the demand for early and safe return to sports. Due to the minimal soft-tissue trauma associated with arthroscopic fracture treatment, it can facilitate early rehabilitation and may lead to improved return to sport [2]. In addition, the use of arthroscopy can aid in the diagnosis and treatment of concomitant pathology that is often found in acute ankle fractures.

Potential benefits of using arthroscopy for ankle fractures in athletes include the following:

- Concomitant treatment of cartilage lesions, which are observed in up to 63% of ankle fractures [2].
- Stability of the syndesmosis can be assessed (e.g., drive-through sign).
- Accurate tibial plafond reduction for complex intra-articular ankle fractures can best be achieved through arthroscopy.
- The minimally invasive nature of arthroscopy can facilitate early rehabilitation.

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Although arthroscopic surgery for posttraumatic pathology has been shown to have significant benefits, the evidence on its use in the treatment of ankle fractures is scarce. The aim of this chapter is to offer an evidence-based overview of the current literature regarding the indications for using arthroscopy in the treatment of acute ankle fractures and its associated injuries in athletes.

16.2 Materials

A Medline search using the keywords "ankle fracture, arthroscopy, and athlete" yielded a total of 55 articles, describing the surgical technique or the outcomes of arthroscopic reduction and internal fixation (ARIF) or arthroscopy-assisted open reduction internal fixation (AORIF) of various types of ankle fractures. Six of these papers focused on ARIF in elite athletes [2, 5–9]. Ligamentous injuries, except for syndesmosis injury, are not discussed in this chapter.

Current indications for ARIF/AORIF in sport-related ankle fracture management include:

- Malleolar fracture
- Intra-articular fracture
- (Osteo-)chondral injury
- Syndesmosis injury
- Talar body/neck fracture
- Talar process fractures

16.3 ARIF (Arthroscopic-Assisted Reduction and Internal Fixation)

Arthroscopic-assisted reduction and internal fixation of ankle fractures was first introduced in 1989 and has since gained acceptance [2]. The use of arthroscopy in the treatment of ankle fractures presents surgeons with the ability to directly visualize the articular surface and assess the presence of associated pathology (e.g., osteochondral lesions), all with minimal surgical exposure. The increased understanding of the pathophysiology of ankle fractures and its associated injuries, combined with a demand for rapid return to sport among athletes, has caused a surge in arthroscopic techniques for the treatment of various indications (Figs. 16.1 and 16.2).

A recent review on the indications of ARIF in ankle fractures concluded that the use of

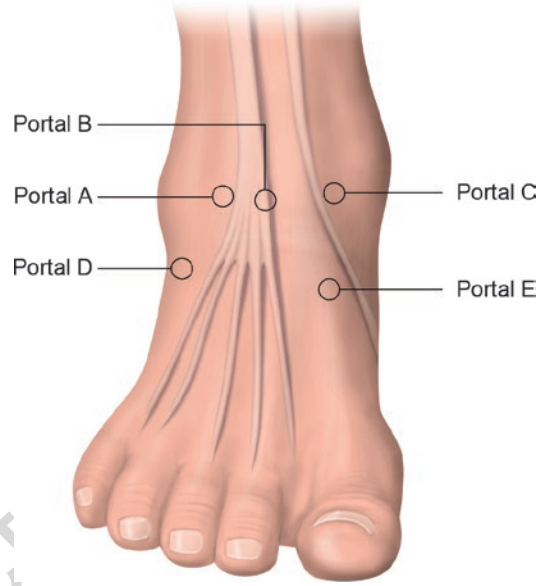

Fig. 16.1 The surge in arthroscopic techniques for ankle pathology has led to the development of different arthroscopic portals that can be chosen to treat the various (described) indications (image copyright: Pieter D'Hooghe)

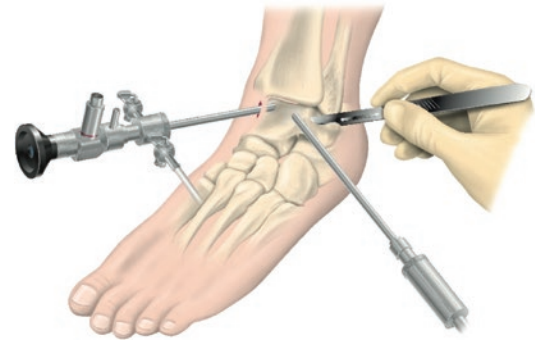

Fig. 16.2 The use of arthroscopy in the treatment of ankle fractures presents surgeons with the ability to directly visualize the articular surface and assess the presence of associated pathology (e.g., osteochondral lesions), all with minimal surgical exposure (image copyright: Pieter D'Hooghe)

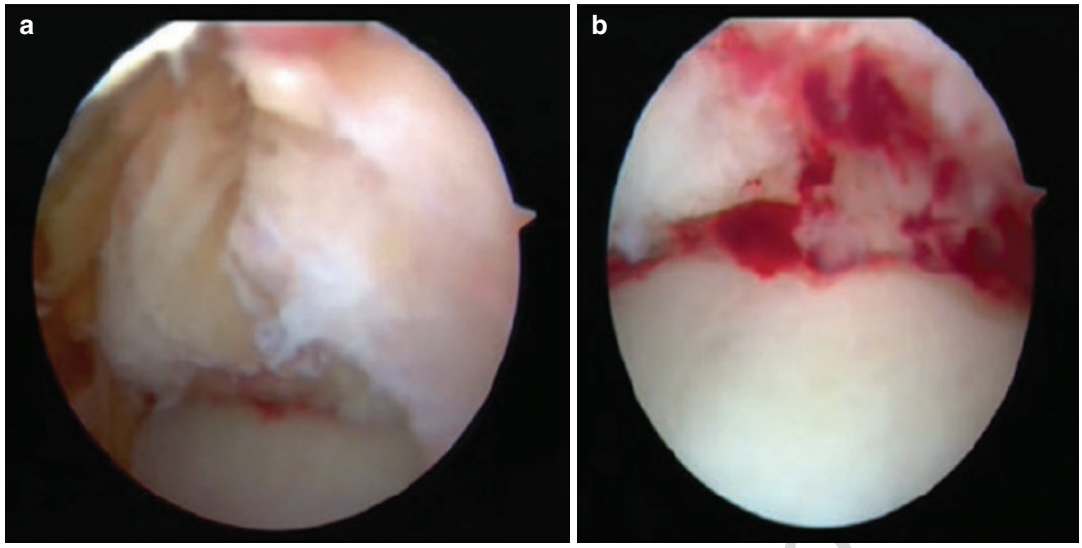

Fig. 16.3 (**a**) Intraoperative anterior arthroscopic view of a distal tibial fracture with intra-articular extension. (**b**) Intraoperative anterior arthroscopic view of a distal tibial fracture after intra-articular reduction (image copyright: Pieter D'Hooghe)

arthroscopy can be advantageous in the treatment of [4]:

- Acute ankle fracture dislocations
- High-energy ankle fractures requiring reduction
- Suspected loose bodies and chondral lesions

The use of arthroscopic reduction and internal fixation (ARIF) has been described for a wide variety of fractures, including fractures of the talus and talar processes, the distal tibia, and fractures of the medial and lateral malleolus [10–13]. Furthermore, using arthroscopic techniques, symptomatic fractures of the medial and lateral posterior process of the talus can be fixed or excised [14]. For most of these indications a classic two-portal anterior/posterior arthroscopic technique is utilized (Fig. 16.3a, b) [2, 5, 6].

In addition to fracture fixation, arthroscopy may facilitate immediate treatment of concomitant ligamentous injuries, tendon pathology, and osteochondral lesions, potentially enabling early rehabilitation and faster return to sports [5].

No absolute contraindications for using arthroscopy in the treatment of acute ankle fractures and its associated injuries have been formulated. However, concerns regarding increased surgical time, soft-tissue swelling, and surgeon-dependent ability to successfully utilize arthroscopic techniques have been stated [2]. Despite these concerns, only one case report describing an acute anterior compartment syndrome following ankle arthroscopy in the treatment of a Maisonneuve fracture in a football player has been published [15].

Relative contraindications for arthroscopy in the treatment of ankle fractures include [4]:

- Low-energy fracture mechanism
- Open fractures
- Degloving injuries with severe soft-tissue compromise

16.4 Indications for Combined Ankle Arthroscopy in Acute Athlete Ankle Fractures

16.4.1 Malleolar Fractures

Malleolar fractures are generally evaluated by physical examination and radiographs—they are then classified according to either the AO or the Weber classification systems. In case of dislocation, immediate reduction is mandatory to prevent

skin necrosis and possible nerve damage. The treatment strategy is chosen based on:

- Mechanism of injury
- Classification/injury severity
- Associated soft-tissue damage

Weber A fractures are usually treated conservatively, while Weber B and C fractures frequently require surgery. Specific attention should be given to the intraoperative evaluation of syndesmotic joint stability, as up to 66% of Weber B and C ankle fractures have some degree of syndesmotic ligamentous injury [5, 16–22]. A recent retrospective review by Chan et al. on a series of 254 ankle fracture patients showed associated syndesmosis disruption in 52% of Weber B fractures, 92% of Weber C fractures, and 20% of isolated medial malleolus fractures [23]. The most frequently encountered complications of open reduction and internal fixation of these fractures are formation of wound hematoma and wound necrosis with a postoperative infection rate of around 2%.

Stufkens et al. analyzed the long-term outcome after surgical treatment of malleolar fractures and noted that over 10% of patients eventually go on to develop ankle arthrosis [16]. The evidence regarding optimal treatment strategies, and in particular regarding the return to sports, for these types of fracture is scarce.

ARIF is shown to be effective in discovering undetected osteochondral defects in the ankle and enabling the surgeon to evaluate the quality of anatomical reduction [3, 5, 17, 22–26]. Up to 60–75% of ankle fractures (that require surgical fixation) have demonstrated evidence of articular cartilage damage—previously undiagnosed prior to surgery [16]. Such injuries are mostly cartilaginous in nature and therefore not radiographically visible (Fig. 16.4a–c).

These lesions usually occur at locations not accessible through traditional fracture surgery incisions. Therefore, simultaneous arthroscopic assessment and management of these lesions are required to improve the rate and quality of recovery after fracture surgery. Since radiographs are

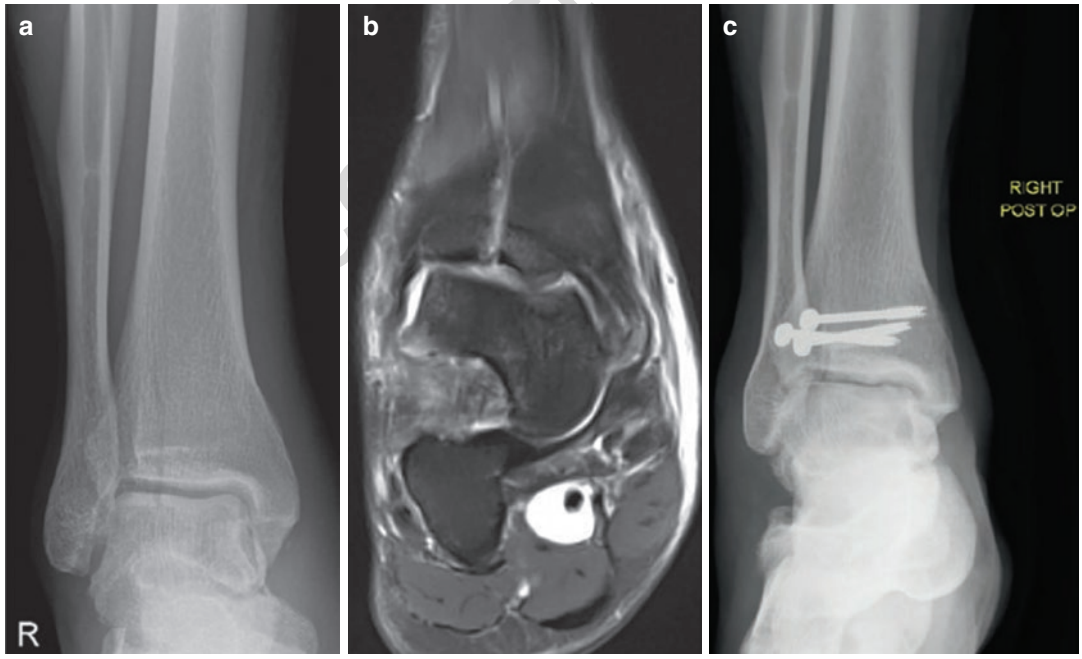

Fig. 16.4 (**a**) Anteroposterior (AP) X-ray of an elite athlete with a centro-lateral distal tibial stress fracture with intra-articular excursion. (**b**) Coronal T2 MRI image of the centro-lateral distal tibial stress fracture with intra-articular excursion. (**c**) After arthroscopic-assisted percutaneous reduction and fixation with control over the anatomical reduction and articular cartilage status (image copyright: Pieter D'Hooghe)

commonly used as the preferred diagnostic tool in acute ankle fractures, the very low sensitivity of plain radiography leads to underdiagnosis of osteochondral lesions [5, 16, 17, 27–29]. In a prospective randomized trial comparing arthroscopic-assisted with traditional non-assisted lateral malleolar fracture fixation, Takao et al. showed a very high rate of secondary pathology. This was mostly chondral damage and syndesmotic injury [17]. At an average follow-up of 40 months, there was a small but significantly greater AOFAS outcome score in the arthroscopically assisted group compared to the traditional group [17].

16.4.2 Intra-articular Fractures

Intra-articular fractures like triplane and Chaput-Tillaux fractures clearly benefit from an arthroscopic-assisted approach as fracture site clearance and intra-articular realignment can be visualized intraoperatively with minimal surgical exposure. Some authors claim that the treatment of triplane fracture should be performed in two steps. The first step is closed reduction under fluoroscopic view. If the displacement is less than 2 mm after closed reduction, it is regarded acceptable and conservative treatment with a short-leg cast is recommended. If the displacement is more than 2 mm after closed reduction, open reduction and internal fixation should be performed [30]. However, a long-term follow-up study of triplane fractures found that in patients treated conservatively, despite there being less than 2 mm of displacement after closed reduction, complications such as decreased ankle mobility, early osteoarthritis, and pain were present at 5-year follow-up [30].

In a case report by *Imade* et al. they applied ankle arthroscopy for the treatment of an ankle triplane fracture for the first time [15]. The use of arthroscopy allowed for a minimally invasive treatment strategy and accurate anatomical reduction. The patient was able to walk without discomfort 2 months after surgery and was able to fully participate in athletic activities with no pain at 3 months postoperatively. A second-look arthroscopy at 1-year follow up showed an articular surface over the previous fracture area that was smooth and congruous. They noted that the fracture line was filled with fibrocartilage-like tissue and concluded that this technique had provided satisfactory results [15]. Various other case reports reporting similar outcomes have been published since [2].

In a recent study by Feng et al. [31], a series of 19 patients with a Chaput-Tillaux fracture (treated with ARIF) were retrospectively followed up after a mean of 19.0 months [2]. Good to excellent results were reported in all patients. The Visual Analogue Scores for pain scores improved from a mean preoperative 8.1 (±0.8 SD) to a postoperative 0.1 (±0.3 SD), at 6-month follow-up. Furthermore, the AOFAS score improved from a mean 52.8 (±6.4) preoperatively to a mean 91.7 (±4.3) at final follow-up.

The use of arthroscopy for isolated malleolar or distal tibial stress fractures with an intra-articular fracture line extension can be equally beneficial, as in Chaput-Tillaux fractures complete cartilage assessment can be performed with arthroscopy without the need for large exposures. Any step-off into the joint line, comminution, or depressed fragment can be recognized and realigned (Fig. 16.5a–d).

Percutaneous temporary K-wires can be used to manipulate and aid in fracture reduction before definitive osteosynthesis is performed [32, 33] (Fig. 16.6a–d).

However, the technique can be technically demanding and no quality comparative studies are available [5, 25].

16.4.3 Osteochondral Lesions

Although open reduction and internal fixation of ankle fractures leads to good result in most patients, poor functional outcome is observed in a subset of patients. It has been hypothesized that these lesser results can be attributed to undiagnosed osteochondral lesions, present in up to 63% of the patients [18, 26].

Acute osteochondral defects associated with ankle fractures are commonly amenable to arthroscopic treatment. Arthroscopic diagnosis of the defect location, defect size, and condition of

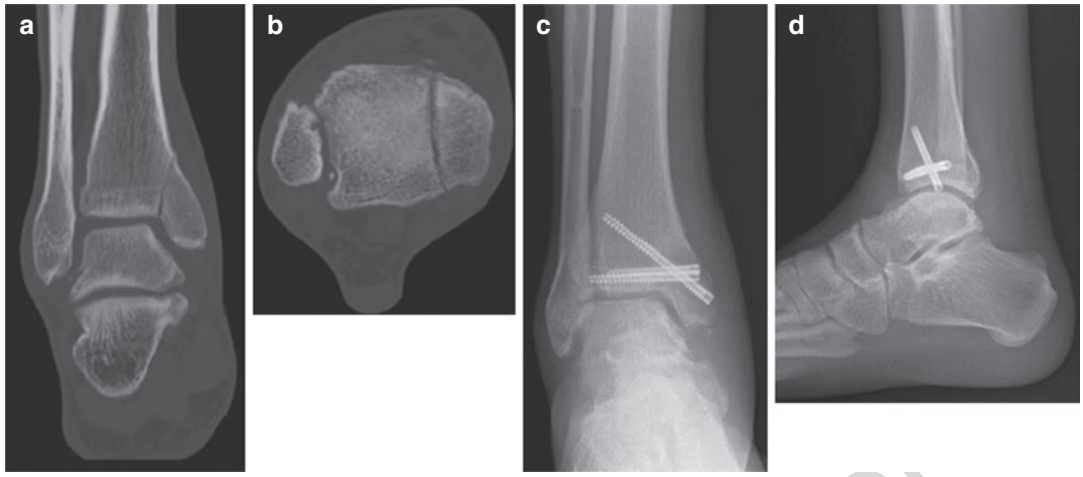

Fig. 16.5 (**a**) Coronal CT image of a medial malleolar stress fracture in the ankle of an elite athlete. Note the talar varus deformity alignment. (**b**) Axial CT image of a medial malleolar stress fracture in the ankle of an elite athlete. Note the anterior small fragment. (**c**) Postoperative AP X-ray after arthroscopic-assisted percutaneous fracture reduction and fixation. (**d**) Postoperative lateral X-ray after arthroscopic-assisted percutaneous fracture reduction and fixation (image copyright: Pieter D'Hooghe)

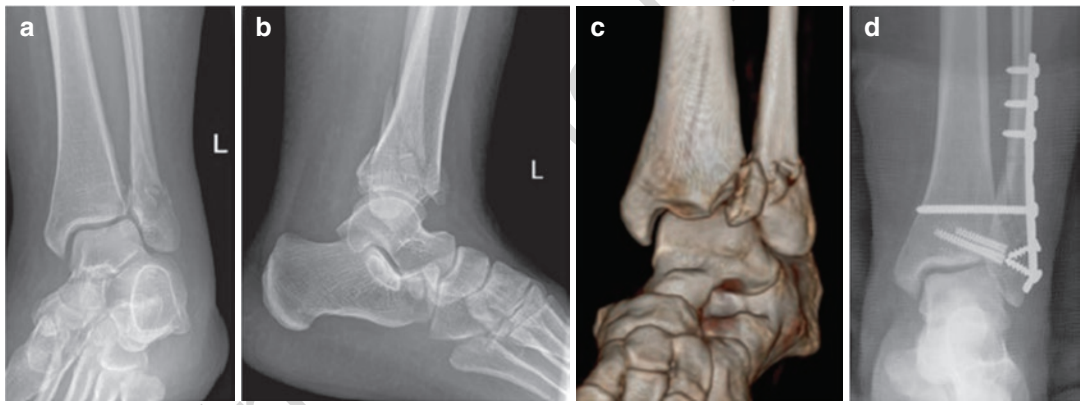

Fig. 16.6 (**a**) AP X-ray of a Weber B distal fibular fracture in an athlete. (**b**) Lateral X-ray reveals the combined bony anterior syndesmotic fracture. (**c**) Coronal 3D CT image of the intra-articular ankle fracture. (**d**) AP X-ray image after arthroscopic-assisted fracture reduction and fixation (image copyright: Pieter D'Hooghe)

the osteochondral fragment can guide the selection of appropriate treatment [2, 17, 18, 28]. Based upon the talar dome/tibial plafond osteochondral defect size, bone marrow stimulation techniques (e.g., drilling, abrasion, or microfracture) or transplantation techniques (autograft/allograft) can be used instantaneously [34–38].

Furthermore, as cartilage-regenerative procedures (autologous chondrocyte implantation [ACI], matrix-induced autologous chondrocyte implantation [MACI]) gain popularity in the treatment of athletes with a chronic osteochondral defect of the talus [39, 40], ARIF in the acute setting can provide cartilage biopsies for cell culture and cartilage implantation in a later stage (ACI). The same treatment strategy is applicable for the less common tibial plafond osteochondral lesions [32].

Currently there is sufficient evidence that arthroscopy can be successfully employed in the treatment of fracture-associated intra-articular injuries. However, despite the obvious potential

of arthroscopy, evidence comparing functional outcome and complication rates of ARIF to ORIF is lacking [41].

16.4.4 Syndesmosis

Fracture-related injury to the syndesmosis is observed in 47–66% of patients and is associated with the development of chronic ankle complaints [19]. Intraoperative stress views are more reliable—when compared to plain radiographs—at detecting definitive instability [20]. Nevertheless, borderline instability or partial injury to the syndesmotic complex without instability is difficult to detect. Magnetic resonance imaging (MRI) has been shown to provide accurate information when documenting a syndesmotic injury, but has a significant false-positive rate, whereas arthroscopic assessment has been shown to be more sensitive and specific and an accurate guide for anatomical reduction of the syndesmosis because it provides 3-dimensional assessment and reduces the chance of having malreduction [2, 5, 20, 21, 23, 42].

In addition, arthroscopy can debride the extra-syndesmotic fibers of the most commonly ruptured anteroinferior tibiofibular ligament that may otherwise produce chronic pain due to anterior impingement [43–45]. Good to excellent results have been reported in a few studies where arthroscopic assessment (with fixation) and/or debridement were used to manage such injuries [17, 18, 27, 29]. Arthroscopic evaluation may also detect sagittal and rotational ankle instability, which may not always be visualized on intraoperative stress radiography [2, 46].

Arthroscopy is also useful in detecting the relationship between malleolar fractures and syndesmotic injury [2, 23] where they found a statistical significance association between Weber B fractures and syndesmotic injuries but no statistical significant association between posterior malleolus fracture and syndesmotic injury [23]. Another important role of arthroscopy can be to monitor residual syndesmosis instability after removal of the syndesmotic screw where they found a low number of residual syndesmosis instability of 3% after screw removal [23]. Finally, damage to the medial area of the talocrural joint, which is an indirect finding commonly associated with syndesmotic injury, can be visualized using the arthroscope.

16.4.5 Talar Body and Neck Fractures

Fractures of the talar neck and body (Fig. 16.7a–e) are rare injuries that can cause significant morbidity and complications.

For the athlete, these injuries can have a deleterious effect on their long-term functional outcome. Treatment efforts are aimed at the quality of fracture reduction and the preservation of talar blood supply. Arthroscopic-assisted surgery has been shown to be of value in both aspects but the technique is demanding, prolongs operative time, and increases soft-tissue swelling. However, case reports and small case series provide some evidence to recommend this technique [16, 47–49]. The underlying principle in managing a talar fracture is to achieve an anatomical reduction and stable fixation with minimal disturbance to the soft tissue—for the abovementioned reasons [47, 48]. Skin necrosis, infection, malunion, and posttraumatic arthritis are well-recognized complications of talar fractures, and management should be designed to minimize these. *Subairy* et al. have shown that arthroscopic-assisted surgical stabilization of these fractures is advantageous and reduces the time to union [48]. Stress fractures are the most common overuse bony injuries in sports but stress fractures of the talar body are extremely rare and have only rarely been reported [6, 10, 50]. More common—but still rare—are stress fractures of the talar neck or lateral talar process [6, 11, 12]. Due to their minor displacement, most stress fractures of the talar body are treated nonsurgically [6, 10, 13]. Stress fractures in sports are the result of excessive, repetitive cyclic loads traumatizing bones with normal form and structure [51]. Predisposing factors may be both intrinsic and extrinsic and include malalignment, lack of flexibility, increase in training, training of excessive volume and intensity, hard or soft activity

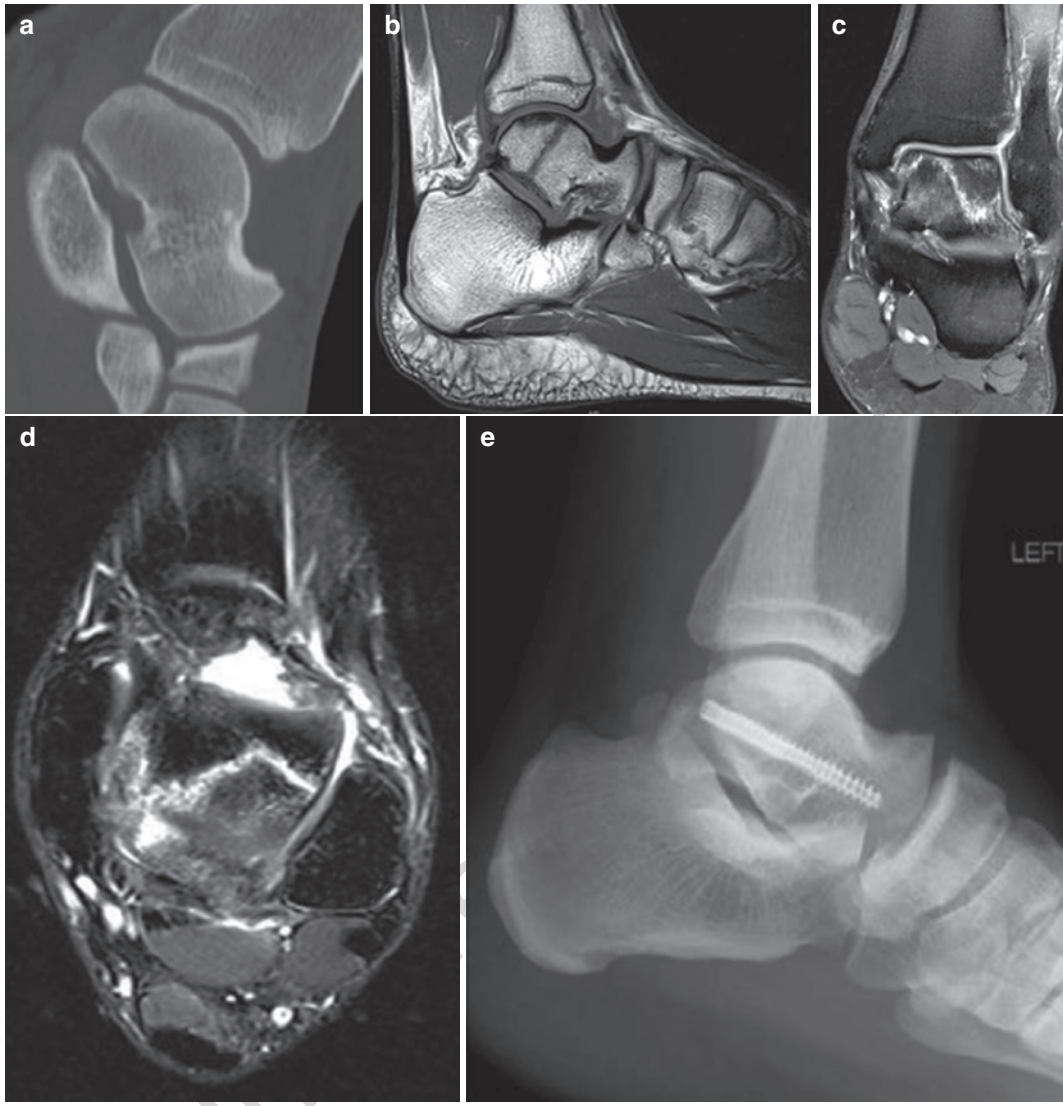

Fig. 16.7 (**a**) Sagittal CT image of an athlete with sudden ankle pain after a preseason training camp. (**b**) Sagittal T1 MRI image of a talar body stress fracture. Note the Hawkins sign. (**c**) Coronal T2 MRI image of the progressive diastasis of the talar body stress fracture during conservative treatment. (**d**) Axial T2 MRI image of the progressive diastasis of the talar body stress fracture during conservative treatment. (**e**) Lateral X-ray of the arthroscopic-assisted talar body fracture compression screw fixation (image copyright: Pieter D'Hooghe)

surfaces, inappropriate shoes, and inadequate coaching [6, 10]. Additional factors to be considered include age, ethnicity, gender, fitness, skill level, and menstrual history [6, 52]. Mechanical factors that may lead to a stress fatigue fracture remain unclear but may result from repeated loading or repetitive prolonged muscular action on bone not yet conditioned to such heavy and novel action. In athletes, significant pathogenetic movements predisposing to a talar stress fracture can be identified in repetitive, restricted axial loading while sprinting, kicking a ball, or landing after heading. The load that has to be absorbed during these actions (the extremes in plantar/dorsiflexion of the foot while kicking the ball and other traumatic actions) should be

considered as an important pathogenetic factor in repetitive strain injuries. Moreover, when playing toward the end of a match or tournament, coordination is less precise as athletes are often fatigued [6, 52].

The diagnosis of a stress fracture is based on clinical suspicion, a detailed history, and a physical examination, followed by appropriate imaging investigations. The role of conventional radiography is important, although initial findings are often minimal or absent (Fig. 16.7a). The earliest sign—often delayed until after the onset of symptoms—may be a lucent linear image (more often a sclerotic band, periosteal reaction, or callus formation) seen on X-ray [6, 10, 13]. MRI has a high sensitivity for the detection of stress fractures (Fig. 16.7b). In addition, MRI signs are evident several weeks before radiographic signs appear.

Conservative treatment is preferred if there is no (or only minor) displacement at the fracture site. There is only limited literature on adequate healing times for stress fractures of the talar body but overall stress fractures are known for their prolonged time to heal [6, 53]. Generally, treatment of stress fractures is immobilization for 4–8 weeks [10, 50, 52, 53]. Avascular necrosis remains a relatively high risk—given the suboptimal talar vascular status—even after an adequate immobilization period [53, 54]. Hawkins classified (non-stress) fractures of the talus in an attempt to predict the risk of avascular necrosis [55]. A Hawkins type 1 fracture has a good prognosis as the risk of avascular necrosis is less than 15% [56]. If significant diastasis/displacement (Hawkins type 2) occurs, the risk of avascular necrosis rises to 50%, and surgical repositioning and fixation is indicated [56] (Fig. 16.7c–e). If adequate measures—with rapid intervention to reposition the displaced fracture—are taken, it is possible to achieve a positive outcome without ongoing problems [6] (Fig. 16.7e). *d'Hooghe* et al. described the management of progressive talar body stress fractures in professional football players through posterior arthroscopy-assisted compression screw fixation with excellent healing results [6] (Fig. 16.7a–e). No other articles were found that combine arthroscopy with talar stress fracture fixation management.

16.4.6 Talar Process Fractures

16.4.6.1 Lateral Tubercle Fractures and Os Trigonum Complex

Posterior impingement in the ankle refers to a mechanical conflict on the posterior side of the ankle. In athletes, it accounts for about 4% of all ankle injuries and can present either acutely or chronically [2]. Posterior ankle impingement syndrome is a clinical pain syndrome reflecting the most common cause of posterior ankle pain. It can be provoked by a forced hyperplantar flexion movement of the ankle [14, 17, 57, 58]. In the event of bony posterior impingement of the ankle, plantar flexion induces a conflict between the posterior malleolus of the distal tibia and the posterosuperior calcaneal bone. A hypertrophic posterior talar process or an os trigonum is present in almost 7% of the sports population [2]. Not every apparent posterior bone—caused by acute or repetitive overload (micro)trauma—induces posterior ankle pain and is not necessarily associated with the posterior ankle impingement syndrome.

Acute forced hyperplantar flexion movement of the ankle can induce a bony conflict in the posterior ankle joint as is frequently seen in sports like football and ballet. The mechanism of injury is a repetitive forced plantar flexion or an acute blocked kicking action. Compression of the os trigonum between the distal tibia and calcaneal bone can also cause this lesion, thus potentially leading to displacement of an os trigonum or fracture of the processus posterior tali or distal tibia (Fig. 16.8).

Over the last three decades, posterior arthroscopy of the ankle joint has become a standardized procedure, with numerous indications for treating posterior (intra-articular) ankle pathology. Lack of direct access and nature and deep location of its hindfoot structures are reasons why posterior ankle problems still pose a diagnostic and therapeutic challenge today.

The two-portal endoscopic technique by *van Dijk* et al. allows for excellent access to the posterior ankle compartment and also to the surrounding extra-articular posterior ankle structures [57]. This technique, using modified classic arthroscopic tools and skills, has introduced a broad spectrum of new indications in posterior ankle pathology [57–59]. The most influential indication to perform posterior ankle arthroscopy remains the treatment of os trigonum. This is an attractive alternative to open surgery for experienced arthroscopic surgeons. Improved functional outcomes after surgery, lower morbidity, and more rapid rehabilitation time make this technique a beneficial technique in athletes [56–59].

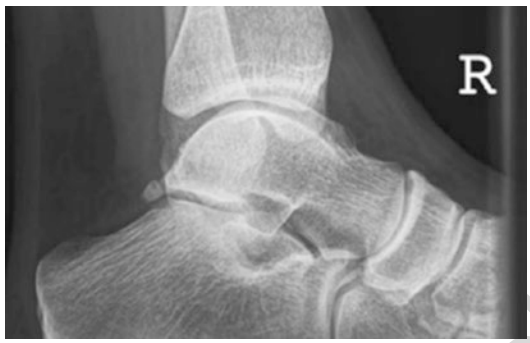

Fig. 16.8 Lateral X-ray of an os trigonum in an athlete's ankle (image copyright: Pieter D'Hooghe)

16.4.6.2 Medial Tubercle Fractures

Fractures of the medial tubercle are rare but can occur due to [2]:

- Avulsion of the posterior tibiotalar ligament
- Dorsiflexion and eversion (Cedell fracture)
- Direct compression of the process as above
- Impingement of the sustentaculum tali in supination

In contrast to lateral tubercle injuries, pain and swelling are usually present between the Achilles tendon and the medial malleolus. However, there may be limited pain on walking or movement of the ankle. It is difficult to visualize fractures of the medial tubercle on plain AP and lateral radiographs, and it has been suggested that the addition of two oblique views at 45° and 70° of external rotation may significantly aid in the detection prior to resorting to a CT or MRI [2] (Fig. 16.9a, b).

These fractures can be approached through the posterior arthroscopic technique—their extent can be visualized and the necessary treatment can be performed in a one-stage procedure.

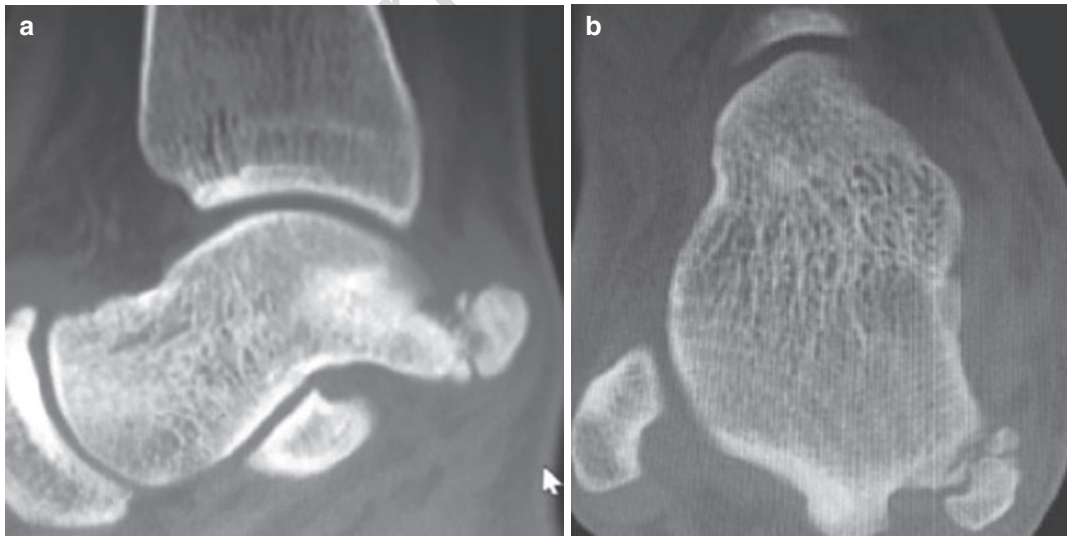

Fig. 16.9 (**a**) Sagittal CT image of a Cedell fracture in an athlete's ankle. (**b**) Axial CT image of a Cedell fracture in an athlete's ankle (image copyright: Pieter D'Hooghe)

16.4.6.3 Entire Posterior Process Fractures

These injuries are usually fractures of the lateral or posterior process and comprise some of the most commonly missed fractures in acute ankle injuries. Routine AP and lateral radiographs do not often show acute fractures and may be incorrectly interpreted. CT scan remains the mainstay of diagnosis, but there also needs to be a high index of suspicion by the assessing physician [2, 5]. Lateral process fractures in sports often present with signs and symptoms of a simple ankle sprain. Undiagnosed and untreated fractures often lead to persistent lateral ankle pain and late subtalar joint arthritis. Outcomes are suboptimal when diagnosis and treatment are delayed for more than 2 weeks [5, 60]. Type 1 fractures benefit from stable fixation usually via an open surgical technique. Type 3 fractures respond well to conservative treatment. Type 2 fractures, however, appear to respond best to early removal of the fracture fragments as opposed to delayed surgery. Removal of these fracture fragments by arthroscopy would reduce the surrounding soft-tissue dissection and potentially accelerate return to normal activity. However, at present, there is no study available that supports this theory. Further studies are therefore necessary in this area. Posterior process fractures usually occur as a result of forced plantar flexion injuries and are even less common than lateral process fractures. Most of these injuries are initially treated with conservative management, but a small number of cases with significant comminution may be appropriately treated by early arthroscopic debridement [5].

16.5 Rehabilitation

Rehabilitation is an essential aspect in the management of the athlete ankle fracture. The aim of arthroscopy is to improve functional outcome and reduce morbidity and shorten rehabilitation time. Therefore, it is commonly used as a valuable tool in sports-related ankle injuries. Initial elevation after injury or operation, as well as early range of motion exercises as soon as safely possible, is encouraged in the early postoperative phase [2] (Table 16.1).

During the healing process of operatively treated ankle fractures, adequate follow-up is advised, as chronic ankle pain may occur. Chronic pain after fracture consolidation may arise as a result of soft-tissue impingement, bony impingement, or loose bodies. Arthroscopy has been shown to improve the outcome of chronic pain after fracture surgery. As demonstrated by *Kim* et al., pain scores improved when hardware removal after ORIF of ankle fractures was combined with arthroscopy, compared to hardware removal alone [61].

16.6 General Outcomes and Time to Return to Competition (Table 16.1)

Outcomes from the general population cannot be directly extrapolated to athletes, who usually receive better and more intense rehabilitation. Their safe and prompt return to a highly demanding level of activity is paramount. Evidence on outcomes on the rare fractures around the ankle (i.e., process and talar fractures) in sports is scarce as discussed earlier. Some evidence on the more common malleolar type fractures has been

Table 16.1 Time (in weeks) athletes required the use of rehabilitative devices and time when athletes were able to resume activities [2]

Classification	N	Crutches	Boot	Brace	Daily living	Practice	Competition
Lateral malleolus fracture	6	1.3 ± 0.5	3.0 ± 0.9	4.3 ± 3.8	1.2 ± 0.8	5.0 ± 0.9	6.8 ± 2.4
Medial malleolus fracture	2	2.0 ± 1.4	2.0 ± 1.4	7.0 ± 1.4	2.0 ± 0.0	12.0 ± 5.7	17.0 ± 9.9
Bimalleolar fracture	10	3.7 ± 1.6	3.7 ± 2.0	4.2. ± 2.2	1.0 ± 0.5	10.9 ± 4.0	12.7 ± 4.0
Syndesmosis disruption injury	4	3.3 ± 1.0	2.3 ± 1.3	6.8 ± 6.1	0.8 ± 0.5	13.5 ± 2.5	15.8 ± 1.7
Salter-Harris-type fracture	4	2.0 ± 0.8	3.5 ± 1.7	9.0 ± 1.2	1.0 ± 0.0	6.3 ± 1.3	8.5 ± 1.0
Pilon fracture	1	4.0	2.0	2.0	1.0	8.0	16.0

documented and allows for conclusions to be made [2]. It has to be noted that a number of studies reporting time-loss ankle injuries provide limited information. These studies often group ankle injuries together, with the severity of injury often being defined by the time to return to sport (rather than the type of injury) [2].

Surgical treatment may allow a more rapid recovery, with earlier weight bearing and functional rehabilitation providing a speedier return to normal daily living and work. However, a recent systematic review by *Donken* et al. looked at surgical versus conservative intervention for treating ankle fractures in adults [62]. They concluded that there is insufficient evidence to determine which type of treatment provided better long-term outcomes. The review only identified four controlled trials (292 adults with displaced ankle fractures) from the general population. Also, there were significant variations and limitations in the types of patients, the surgical and rehabilitation protocols applied, the outcomes reported, and the duration of follow-up. Another study by *Colvin* et al. looked at the functional ability of 243 patients who underwent operative fixation of unstable ankle fractures to return to "vigorous activity" and sport [7].

In their study, young and healthy male patients were more likely to return to sport. At 1-year follow-up—although 88% of recreational athletes were able to return to sport—only 11.6% of competitive athletes were able to do so. Specifically, those with bimalleolar fractures were more likely to return to sport, compared with those with unimalleolar fractures. However, this retrospective study analyzed self-reported outcomes from a general trauma population only [7]. Nevertheless, it has been suggested that surgical management (by open reduction and internal fixation of unstable ankle fractures) in athletes may provide a number of advantages. Firstly, it would avoid the issues of secondary fracture displacement which delay recovery. Secondly, it would ensure anatomic fracture reduction and articular surface restoration. Finally, it allows for early range-of-movement exercises and early weight bearing (within 1–2 weeks of fixation) and a more rapid recovery and return to sport [8].

Studies specifically looking at ankle fractures in elite athletes are limited [2, 8, 9, 63], but appear to demonstrate that a successful return to high-level competition can be expected. A study by *Dunley* et al. on three professional American football players showed that all three returned to their pre-injury level [9]. Walsh et al. reported similar findings in a study on the surgical treatment of ankle fractures in three American football players and one soccer player [63]. Another study by *Oztekin* et al. looked at the time-loss from play in ankle injuries of Turkish professional football players. In this study, all patients that were surgically treated for their ankle fracture were able to return to their previous level of play [64]. A layoff of 150 days in this study was reported for two football players (one with a Maisonneuve fracture and one with a lateral malleolar fracture with deltoid rupture), while a patient that was treated for a lateral malleolus pseudarthrosis took 200 days. Another study by *Porter* documented the management, rehabilitation, and outcomes in 27 athletes with ankle fractures that underwent ORIF (including repair of any injured ligaments). The indication for surgery was either displacement of ≥3 mm or if the athlete was "especially enthusiastic" for an early return to sports [8]. The most common sport injuries were in American football (ten athletes) and baseball (three athletes), but two athletes involved in soccer were also included. At an average follow-up of 2.4 years (12 months to 3.7 years), all athletes reported an average 96.4% functional rating compared to their pre-injury level, with 12 athletes rating their ankle as 100%. Early rehabilitation and ambulation were encouraged, which included the use of an ankle Cryo/Cuff™, with athletes encouraged to weight bear in a walking boot within a week postoperatively.

The ability of athletes to be weaned off their rehabilitative devices and the time required to reach activity goals are shown in Table 16.1 [8]. Those athletes with isolated Weber A and B lateral malleolar fractures were able to return to sport within the shortest time. In this study, return to full activity was seen as early as 4 weeks. Two out of the six athletes did not rate their ankle 100% in either flexibility or decreased stability

issues. Two athletes in this study (with isolated medial malleolar fractures) required deltoid ligament repair at the same time. These athletes took longer to return to competition, with one patient taking 24 weeks to return to motocross racing.

Athletes with bimalleolar fractures required 12.7 ± 4.0 weeks to return to competition, while athletes with syndesmotic injury and pilon fractures took slightly longer. The authors did not document the recovery of patients with stable and undisplaced ankle fractures that underwent non-operative treatment. There is a lack of evidence with regard to outcomes and return to competition in athletes with such injuries but they felt that early rehabilitation and ambulation would be possible in such cases, and a similar return to sport should be expected [2]. No study was found that documents arthroscopic-assisted ankle fracture fixation and its value in return to elite sports resumption, compared to a control group (without arthroscopy). Further work is required to objectively describe the potentially added value of arthroscopy in this return-to-sport perspective.

16.7 Conclusion

The incidence of ankle fractures is low, making up less than 3% of all ankle injuries in athletes. Optimal management for the elite athlete has to address the demand for early and safe return to a high level of activity. The evidence for current best practice in athlete-related ankle fractures remains limited. A thorough history, examination, and adequate imaging are essential to correctly diagnose injuries and decide upon the optimal treatment plan. Early rehabilitation allows for an early return to sport within 2–4 months depending on the fracture severity. Surgical reduction (when indicated) and provision of stability by fixation optimize both outcomes and return to competition in the athlete ankle fracture. Arthroscopy may be helpful in diagnosing (and treating) intra-articular pathology (up to 60% of ankle fractures may have a cartilage injury). Furthermore, arthroscopy may also have a role in the assessment of syndesmosis stability and can assist in the accurate reduction of displaced (tibial plafond, malleolar, and talar) fractures. Arthroscopic techniques allow for a more rapid rehabilitation, with fewer complications, than conventional techniques in athletes.

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17 Arthroscopic Treatment of Anterior Ankle Impingement

Thomas Bauer

17.1 Introduction

Ankle impingements are painful syndromes due to hyperplasic synovitis and fibrotic soft tissues being caught between the ankle and hindfoot bony surfaces during ankle motion. Basically anterior ankle impingements occur after injuries or supination trauma and can be localized either on the anteromedial or the antero-lateral part of the ankle joint. Diagnosis of ankle impingement is clinical: palpation associated to passive mobilization of the ankle reproduces the localized pain recognized by the patient. Local injection is an important step not only to confirm the diagnosis but also to definitely remove painful symptoms in some cases.

17.2 Distinction Between Anteromedial and Antero-lateral Ankle Impingement Syndrome (ALAIS)

Antero-lateral ankle impingement syndrome (ALAIS) manifests as anterior ankle pain at the talo-fibular groove. A distinction is classically made based on whether the impingement is due to bone or soft tissue [1–9]. Bony impingement is caused by osteophytes originating at the anterior tibial margin and talar neck [10]. However, whereas anteromedial ankle impingement syndrome usually involves tibial and talar osteophytes, ALAIS is usually due only to soft tissue interposition. The first report of ALAIS, written in 1950 by Wollin, describes joint invasion by a mass of connective tissue originating from the anterior talo-fibular ligament (ATFL) [11]. In 1991, Ferkel and Scranton provided further details on the pathophysiology of ALAIS [1]. The inciting event is an ankle sprain with injury to the ATFL. If ligament healing is incomplete, repeated ankle movements result in synovitis, followed by fibrosis with the development of a soft tissue mass, whose interposition in the joint space causes pain at the talo-fibular groove. Thus, pain due to ALAIS is extremely common and perhaps even inevitable after an ankle sprain, as the ATFL healing process is accompanied with local inflammation. However, the pain is expected to resolve within a few weeks after complete ATFL healing.

ALAIS is closely linked to ATFL injury and, in some patients, to chronic ankle instability. Rotational micro-instability of the ankle is challenging to document. Pain may be the only manifestation, with no objective evidence of laxity, and the presentation is then identical to that of ALAIS.

17.3 Diagnostic Strategy

Diagnosis of an anterior ankle impingement is clinical, and distinction is made with the localization of the pain at palpation: an anteromedial pain

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with osteophytes is an anteromedial ankle impingement and a bony impingement, whereas an antero-lateral pain without osteophyte at palpation is an antero-lateral soft tissue ankle impingement. The diagnosis of ALAIS rests on clinical findings. ALAIS should be considered in patients with persistent pain 6 months after appropriate treatment of an inversion ankle injury [12]. The reported frequency of ALAIS after ankle sprains is 1–2% but is no doubt considerably underestimated [12–14].

The clinical manifestations of ALAIS [1, 5, 15, 16] include range-of-motion limitation, a swelling in the antero-lateral groove, and a locking sensation or snapping during dorsiflexion and eversion of the foot. The best diagnostic test is the Molloy test, which is 94.8% sensitive and 88% specific for ALAIS [17]. The examiner places the foot in forced dorsiflexion while applying pressure to the antero-lateral groove (Fig. 17.1). The test is positive if this maneuver replicates the usual pain.

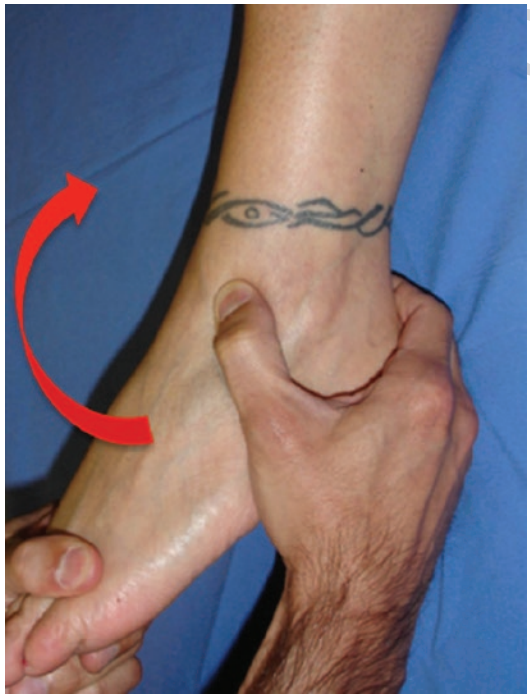

Fig. 17.1 Molloy test: the examiner applies pressure to the antero-lateral groove while moving the ankle into forced dorsiflexion

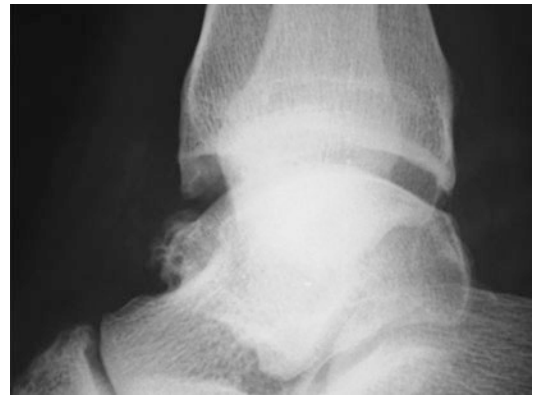

Fig. 17.2 X-rays lateral ankle view: anterior bony impingement

Anteroposterior and lateral radiographs of the ankle rule out bony impingement (Fig. 17.2) and osteochondroma and may provide suggestive evidence of an osteochondral lesion [18]. Ultrasonography documents the soft tissue impingement. A heterogeneous mass larger than 7 mm in diameter is visible at the antero-lateral corner of the ankle [19, 20]. The mass is hypervascular by Doppler ultrasonography. Performing the Molloy test during ultrasonography confirms the soft tissue impingement, with a mass bulging in the antero-lateral groove during ankle dorsiflexion, but fails to add to the physical examination (77% sensitivity and 55% specificity) [19]. Importantly, ultrasonography serves to guide the corticosteroid injection, which is crucial to both the diagnosis and the treatment of ALAIS [20, 21]. Computed tomography (CT) arthrography has 97% sensitivity and 71% specificity for ALAIS. Nodules may be visible in the antero-lateral groove, and the joint capsule contour may appear uneven. However, CT arthrography has little impact on therapeutic decision-making [22]. Magnetic resonance imaging (MRI) contributes little to the diagnosis of ALAIS. Sensitivity has ranged from 39 to 100% and specificity from 50 to 100% [23–28]. MR arthrography performs better, however, with 96% sensitivity and 97% specificity [29].

Liu et al. defined six clinical criteria for the diagnosis of ALAIS [4]: persistent antero-lateral pain after a sprain of the lateral collateral ligaments, antero-lateral effusion and swelling,

recurrent tibio-talar pain after exercising, antero-lateral pain during dorsi- flexion with eversion, pain during single-leg squats, and absence of lateral laxity. Patients with at least five of these criteria were diagnosed with ALAIS [4]. These criteria require the elimination of ankle instability based on the absence of objective lateral laxity. They do not consider rotational micro-instability, which is difficult to establish clinically. The six criteria may be met in patients with true rotational micro-instability who have no symptoms other than those of ALAIS. The physical examination alone has 94% sensitivity and 75% specificity for the diagnosis of ALAIS [4, 30].

17.4 Arthroscopic Treatment

Anterior ankle impingement surgical treatment is performed as an arthroscopic procedure. The standard patient installation for anterior ankle arthroscopy is used, without joint distraction. Two portals are created, one anteromedial and the other antero-lateral. The arthroscope is 4.0 mm in diameter. The instruments (hook probe, 4.0-mm power shaver, power scalpel) are introduced through an antero-lateral portal created under direct visual guidance after insertion of a needle. The anterior part of the joint is cleared with the ankle in forced dorsiflexion until the anterior tibial margin, talar neck, and both malleoli are visible. The fibrous and inflammatory tissue is removed completely, to make the bony landmarks and any osteophytes clearly visible.

In patients with anteromedial bony impingement, an anterior synovectomy is first performed and then a complete resection of the tibial and talar osteophytes after complete visualization. Osteophyte resection is begun at the level of the origin of the bone spur (anterior tibial margin or talar neck) with a progression from its insertion to the articular surface: thus for a tibial osteophyte the resection is performed from proximal to distal, and for a talar osteophyte, the resection is performed from distal to proximal (Fig. 17.3a–c). With this technique a complete and flat resection of the osteophyte can be achieved without residual bone spur that can lead to a recurrent anterior ankle impingement syndrome (Fig. 17.4a, b). In case of malleolar osteophytes (at the tip and anterior margin of the medial malleolus), after resection of the osteophyte, a large resection of the anterior surface and tip of the medial malleolus is made in order to decrease the volume of the medial malleolus and avoid anteromedial remnant impingement in dorsiflexion and inversion.

In patients with ALAIS, arthroscopy may show several abnormalities, which are often present in combination: focal or extensive inflammation of the synovial membrane, which has a pinkish-purple hue; one or more bands of scar tissue, in some cases with a meniscoid appearance at the level of the distal band of the antero-inferior tibio-fibular ligament; osteophytes arising from the anterior margin of the distal tibia and neck of the talus, best seen with the ankle in forced dorsiflexion; ossifications at the anterior edge and tip of the lateral malleolus; and osteochondral loose bodies in the anterior talo-fibular groove.

The resection is started at the distal band of the anteroinferior tibio-fibular ligament in order to visualize this major anatomical landmark. The synovectomy is then extended to the antero-lateral corner of the ankle and, subsequently, to the anterior tibio-talar compartment and antero-lateral groove.

At the antero-lateral groove, the resection of synovial membrane and fibrous tissue should be stopped at the upper edge of the ATFL, which should be identified routinely. At this point, the risk is excessive extension of the synovectomy, with partial or complete resection of the ATFL, which would worsen any pre-existing instability and, even more importantly, result in persistent pain from ALAIS.

After starting the synovectomy, the crucial step in the arthroscopy procedure is a visual assessment of the antero-lateral groove with detection of any ATFL lesions. Following the anteroinferior tibio-fibular ligament in the medial-to-lateral direction leads to the ATFL, where any lesions can be assessed visually and with the probe [31, 32]. Distension of the ligament plane should be sought, as well as detachment from the malleolus (by inserting the hook between the anterior mal-

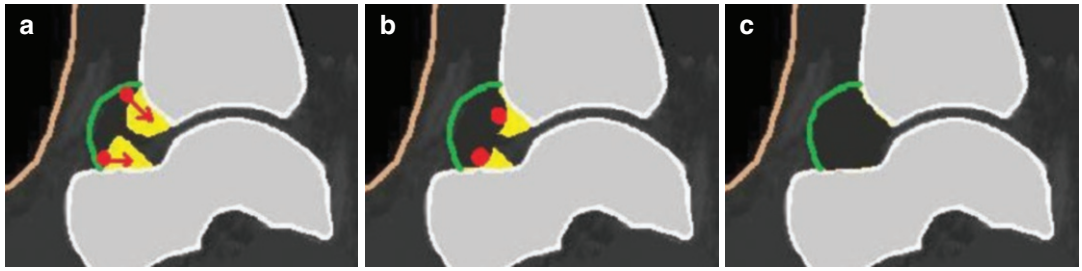

Fig. 17.3 (**a**–**c**) Arthroscopic technique of anterior ankle osteophyte resection from its implantation in the direction of the articular surface to achieve a complete resection

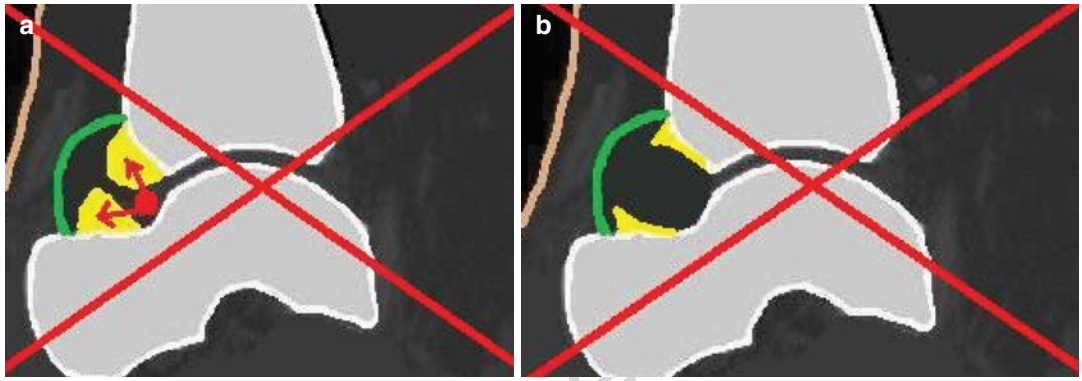

Fig. 17.4 (**a**, **b**) Bad technique of osteophyte resection (beginning at the joint line) with risk of residual osteophyte

leolar edge and the ATFL), talar avulsion, and a tear in the body of the ligament (which is less common). The quality of the residual ATFL should be assessed as thinned, discontinuous and irregular, or thick and strong [32]. Appropriate repair of any ATFL lesions seems reasonable [33].

17.5 Outcomes of Surgical Treatment: Literature Review

In early studies of arthroscopic methods for treating ALAIS, outcomes were good or excellent in over 60% of cases, with a complication rate ranging from 10 to 15% (nerve injury, superficial surgical-site infection) [4]. In more recent studies, the rate of good or excellent outcomes was 67–100%, and complications were considerably less common than with open surgery and in early studies of arthroscopic treatment [3, 5, 8, 34, 35]. Anterior bony impingement involving osteophytes had the best prognosis, with over 80% of good or excellent outcomes [3, 5, 9, 35–37]. Compared to open surgical treatment of ALAIS, the time to recovery is halved with arthroscopic treatment, and the time to sports resumption is decreased by about 1 month [35]. An important distinction is between isolated anterior impingement, in which a good outcome can be expected, and impingement due to osteophytes occurring as an early manifestation of tibio-talar cartilage degeneration, which has a more reserved prognosis. Tol et al. and van Dijk [27, 35] reported that the proportion of patients with good or excellent outcomes after arthroscopic treatment for anterior osteophytes was 82% when the joint space was intact compared to only 50% in the event of joint space narrowing. In the medium or long term, however, no progression of the cartilage lesions occurs after arthroscopic treatment for ALAIS, and about two-thirds of patients remain satisfied or very satisfied for many years despite experiencing functional impairments [35]. Furthermore, although the osteophytes recur

within a few years after the arthroscopic procedure, most patients remain free of symptoms, indicating that the ankle pain is not caused by the osteophytes but, instead, by pinching of the synovial membrane and synovitis [35]. A multicenter study reported in 2007 identified three predictors of arthroscopic treatment failure in patients with ALAIS [36]: older age (mean age at surgery was 46 years in patients with poor outcomes and 34 years in those with good or excellent outcomes), longer trauma-to-surgery time (mean was 33 months in the group with poor outcomes and 20 months in the group with good or excellent outcomes), and cartilage damage (grade 2 lesions were present in 50% of patients with poor outcomes compared to only 18% of those with good or excellent outcomes).

Arthroscopic treatment of ALAIS is extremely effective in relieving the anterior ankle pain, allowing a return to previous activities, providing a good subjective outcome, and improving range of motion. Mobility can be maximized by extensive capsule and ligament release combined with extensive resection of any anterior osteophytes [37]. The low complication rate is among the main advantages of arthroscopic treatment. Proper arthroscopic technique must be followed to avoid injury to nerves and tendons.

In a recent systematic review of arthroscopic treatment for anterior ankle impingement syndrome, outcomes did not differ significantly between antero-lateral and anteromedial impingement, bony and soft tissue impingement, or impingement with versus without concomitant lesions [38]. The main published studies pooled all types of anterior ankle impingement and thus provided no specific data on ALAIS.

17.6 Concept of Rotational Ankle Micro-instability

Rotational ankle micro-instability is defined as any combination of chronic ankle instability symptoms with no objective evidence of forced varus or anterior-drawer laxity. The symptoms may consist of recurrent ankle sprains, weakness of the ankle, ankle pain and instability, and manifestations of ALAIS. No anterior or lateral laxity is found upon physical examination or imaging studies. Use of the term "functional instability" to designate this presentation, as opposed to "mechanical instability" (with objective laxity), in the English-language literature adds to the confusion. In a study by Takao et al. of 14 patients with functional instability, arthroscopy consistently showed lesions of the ATFL (partial fibrosis, $n = 9$; total fibrosis, $n = 3$; and detachment, $n = 2$) [39]. More recently, Vega et al. reported findings in 38 patients with ALAIS and functional instability who underwent arthroscopic surgery [40]. Only half the patients had evidence of synovitis. However, proximal detachment and fibrosis of the ATFL were noted in 60% and 50% of patients, respectively. These recent data confirm the very high prevalence of ATFL lesions in patients with ALAIS. Most of the studies reporting outcomes in patients treated for ALAIS did not consider micro-instability, which is a recent concept. Thus, for many years, ALAIS was described under the assumption that the absence of objective laxity ruled out ankle instability. Although outcomes of anterior ankle impingement overall are generally described as good, the data are less clear for ALAIS. Most importantly, although the symptoms of ALAIS originate in ATFL lesions, the treatment and outcome of these are only very rarely discussed in the literature [1]. This underestimation of the close intertwining between ATFL lesions and ALAIS is probably ascribable to the definition of ALAIS, which excludes ankle instability, and to the techniques used early in the development of anterior ankle arthroscopy (traction, 2.7-mm arthroscope).

Advances in ankle arthroscopy have improved the ability to explore the talo-fibular groove and lateral ligament complex, thus providing new insight into the pathophysiology of ALAIS by demonstrating the key role for ATFL lesions and shedding light on the concept of rotational micro-instability. A new arthroscopic classification of chronic lesions of ATFL in chronic ankle instability has recently been published showing that for early stages of lesions (stage 1 = ATFL distension, stage 2 = ATFL avulsion) it creates a rotational ankle micro-instability with symptoms

of ALAIS [41, 42]. This new knowledge has directly affected the therapeutic strategy by supporting the addition of ATFL repair procedures (as appropriate for the observed lesions) in addition to antero-lateral synovectomy. Prospective multicenter studies are under way with the goal of gaining further knowledge about ALAIS and rotational ankle micro-instability and of obtaining details on outcomes.

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18 Posterior Impingement and Os Trigonum

Hélder Pereira, Jorge Batista, Duarte Sousa, Sérgio Gomes, J. P. Pereira, and Pedro L. Ripoll

18.1 Introduction

The posterior ankle impingement (PAI) is a clinical syndrome which involves a bony structure or soft tissue that becomes entrapped between the calcaneus and the posteroinferior aspect of the tibia (Fig. 18.1). Repetitive microtrauma in plantar flexion is the most frequent mechanism leading to PAI syndrome [1, 2].

Recurrent loading in plantar flexion might lead to inflammatory response, soft tissue and/or bony edema, and spur formation, ultimately causing impingement syndromes [3, 4]. However, major acute trauma can also be the source of bony or soft-tissue symptomatic PAI [5].

One must understand the involved mechanism of each sports/activity, and always perform a global assessment of the joint and comprehend patient's symptoms.

According to Ribbans et al. [6], the first description of surgical treatment of posterior ankle impingement syndrome was related to pathology of the flexor hallucis longus (FHL) in dancers [7]. Given the hindfoot anatomy, FHL pathology can be either a differential diagnosis or the cause of symptomatic posterior ankle impingement (Fig. 18.2).

The description of *os trigonum* is attributed to both Cloquet and Shepherd who have independently performed anatomic descriptions of this structure [8]. Moreover, both initially considered that this structure derived from a fracture (for this reason it was called Shepherd's fracture). The former finally recognized that this structure was in fact a secondary ossification center of the

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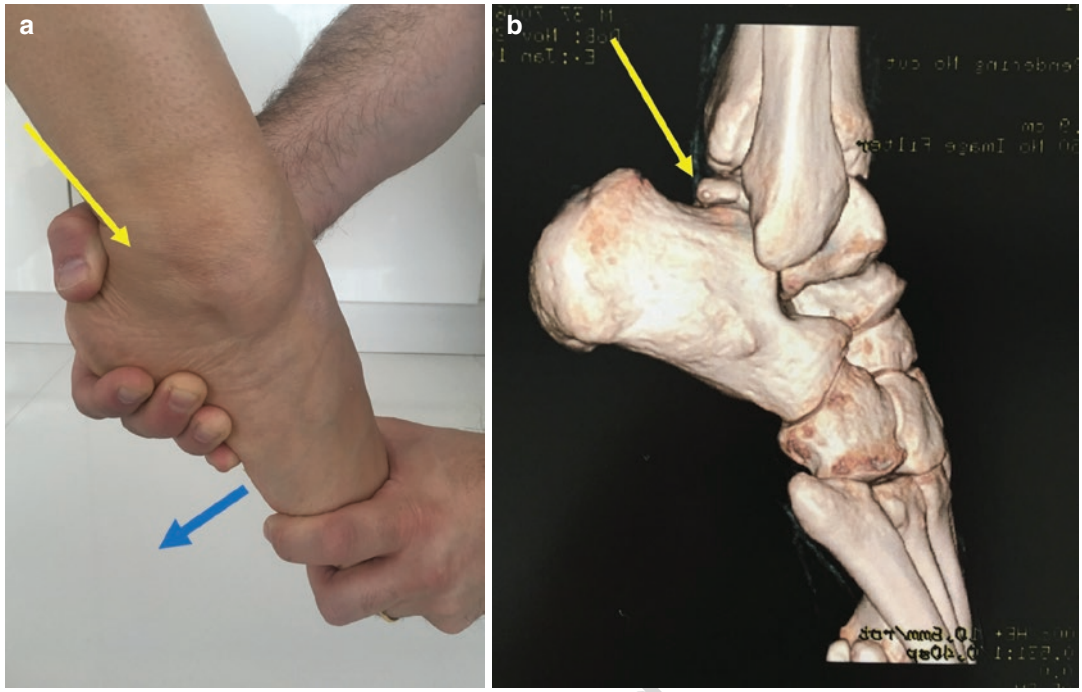

Fig. 18.1 (**a**) Posterior impingement test, in which the examiner induces stress in hindfoot combining some plantar flexion (blue arrow) and posterior manual gridding of the hindfoot aiming to reproduce a recognizable posterior pain from the patient (yellow arrow); (**b**) 3D CT image in plantar flexion demonstrating the impingement of an *os trigonum* between the posterior distal tibia and calcaneus—posterior ankle impingement

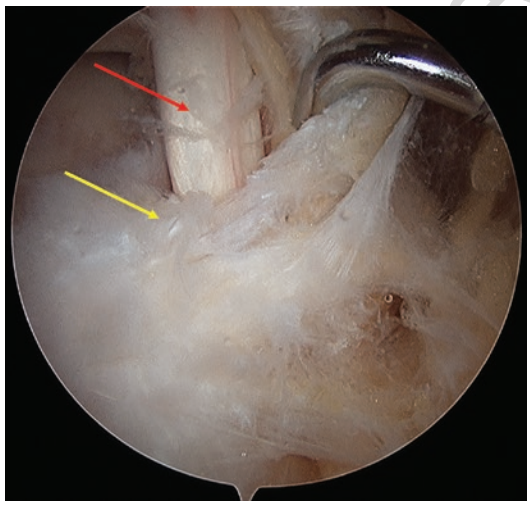

Fig. 18.2 Entrapment of the *flexor hallucis longus* (FHL—red arrow) inside its sheath with hypertrophic soft tissue (fibrosis—yellow arrow) causing soft-tissue posterior ankle impingement. The hook probe is used to find the opening of FHL tunnel

posterior talar process [8]. Often this structure is asymptomatic, but after a trauma or repeated loading it might cause pain and functional limitation (Figs. 18.3 and 18.4). Together with hypertrophic posterior talar process, these are probably the most frequent causes of symptomatic PAI [9].

History and clinical examination are the main focuses for the diagnosis of this syndrome. However, imaging might be useful for differential diagnosis or preoperative planning.

Conservative treatment (physiotherapy, injections, shoe wear) is usually the first option when dealing with symptomatic PAI [3, 5, 10, 11].

Upon failure of conservative management surgical treatment is recommended [3, 5, 10, 11].

Surgery evolved from open to endoscopic procedures [6]. Since Vand Dijk's description of the two-portal endoscopic approach of the hindfoot in the year 2000, most cases of PAI are treated by minimally invasive endoscopic approach [2, 9, 12].

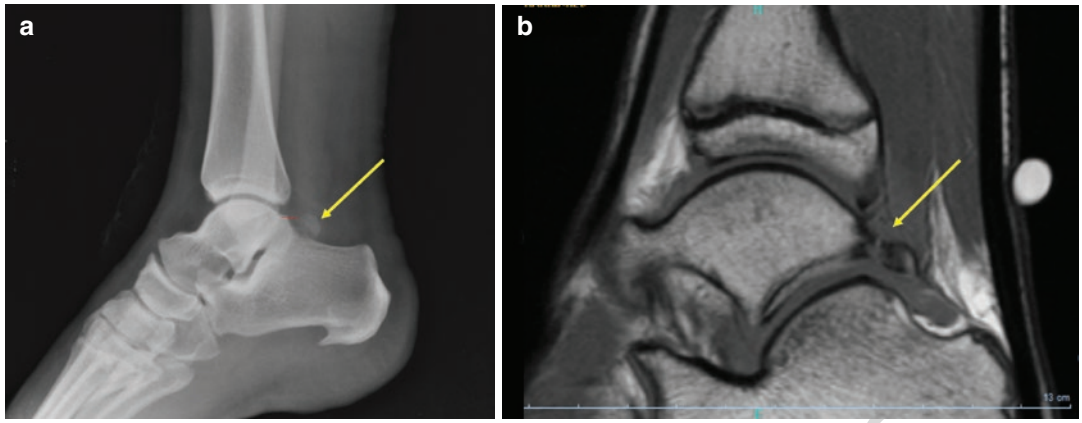

Fig. 18.3 (**a**) Lateral X-ray demonstrating a fracture of the fibrous connection of an *os trigonum* (yellow arrow) which became dislocated (red line shows the difference between its original and final position); (**b**) MRI showing *os trigonum* fracture with focal edema in a child (yellow arrow)

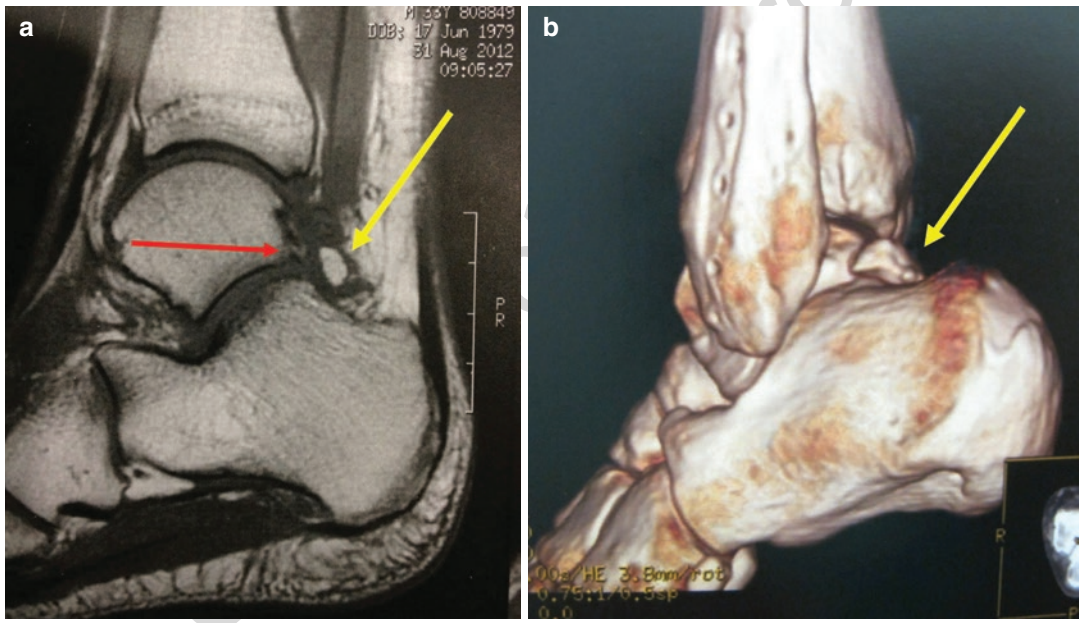

Fig. 18.4 (**a**) MRI with synovitis and inflammatory tissue surrounding an *os trigonum* (yellow arrow); (**b**) 3D CT showing posterior entrapment of *os trigonum* (yellow arrow)

18.2 Epidemiology, Diagnostic, and Mechanisms of Injury for Posterior Ankle Impingement (PAI)

Posterior impingement syndrome is related to a mechanical conflict of the hindfoot aggravated by hyperplantarflexion [11]. It can be either considered as an acute entity following a traumatic event (*os trigonum* or Stieda process fracture or dislocation) [10, 13–15] or chronic, linked to repetitive microtrauma (which might also be combined/aggravated by CAI). Chronic cases can be linked to hypertrophic *os trigonum* or posterior talar process as well as related fractures or soft-tissue impingement (e.g., cysts,

labral injuries, hypertrophic intermalleolar ligaments). It is often observed in ballet dancers, footballers, cyclists, swimmers, acrobatic gymnasts, and downhill runners (Fig. 18.5) [5, 8, 10, 16].

18.2.1 Epidemiology

When considering literature, most descriptions of PAI are connected to dance (around 60% of reported studies) [6, 8] followed by increasing interest and research in football [6, 8].

Somewhat surprisingly, PAI is one of the most frequent causes for players considered unable to train/play related to foot and ankle problems [6, 8].

However, the true incidence and prevalence of this condition are still unknown or at least debatable, particularly if all possible causes are considered [5, 6]. One study followed 186 ballet dancers during 1-year follow-up and identified a prevalence of PAI of 6.5% [17].

PAI was the cause for 31% of all days lost for sports activity due to foot and ankle conditions, which was higher than lateral ankle ligament injuries and Achilles disorders combined [6].

Bony-related PAI is apparently two times more frequent than soft-tissue etiology [6, 8].

Amongst bony reasons, os trigonum or hypertrophic Stieda process are the most usual causes [6].

An os trigonum is an accessory bone which follows a developmental variation of the secondary ossification center of the posterolateral talus. In 7–14% of adults it remains as a separate accessory bone, which is bilateral in 1.4% of cases [8]. This structure is usually asymptomatic, but it may become painful in individuals participating in sports involving repeated plantar flexion [18].

On the other hand, the incidence of *os trigonum* syndrome in athletes is highly variable, ranging from 1.7% to 50%. Moreover, it has also been stated that between 33% and 50% of athletes present it bilaterally [19, 20]. It appears that there are no major differences concerning gender or age [20].

It is less frequent to find PAI in the nonathletic population or athletes from sports which require less frequently ankle plantar flexion. In patients who present with chronic hindfoot pain and do not participate in activities with repetitive plantar flexion, it is more likely to find anatomic variations as cause of PAI and these should be ruled out [5].

18.2.2 Diagnosis

Once being considered a syndrome, clinical diagnosis based on a complete history and careful clinical examination is of major relevance. The clinical presentation of PAI usually includes deep posterior ankle pain caused/aggravated by plantar flexion of the ankle joint, descending stairs or uneven ground, or high heels [21]. Patients tend

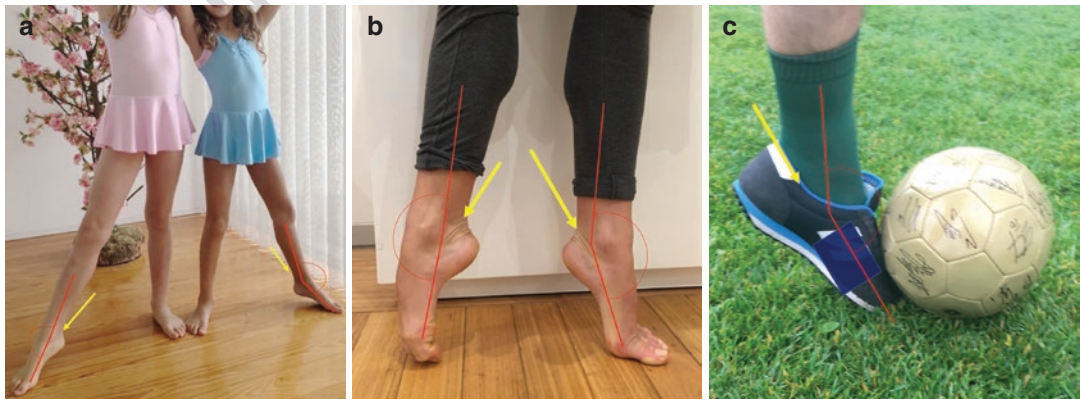

Fig. 18.5 Mechanisms of repeated microtrauma and hindfoot overload in plantarflexion (yellow arrows) in gymnasts (**a**). Ballet dancer en-pointe (right foot) and demi-pointe (left foot) (**b**) and football player while kicking a ball (**c**). In red is shown the angles of plantar flexion required for these technical gestures

to describe consistent, sharp, dull, and radiating hindfoot pain. Nevertheless, often they cannot indicate the exact painful location/spot. The posterior impingement test, in which the examiner induces stress in hindfoot combining some plantar flexion and posterior manual gridding aiming to cause a pain that the patient recognizes as his/her major complaint (Fig. 18.1), is the most important maneuver [1, 2]. A significant amount of false positive for hyperplantarflexion test has been empirically reported [1, 2].

Physical examination should include evaluation of gait and alignment. A complete neurovascular examination as well as assessment of strength and range of motion (active and passive) are also required. Hindfoot pain aggravated by plantar flexion of the ankle reinforced by a positive posterior impingement test (Fig. 18.1) provides the diagnosis of PAI. A negative test makes the diagnosis of PAI much more unlikely; however, there are no studies reporting on the specificity or sensitivity of the plantar flexion test in the diagnosis of PAI. The examiner must carefully try to assess the precise location of tenderness. As example, posteromedial pain over the posterior tibial tendon suggests posterior tibial tendon pathology and not PAI.

It is suggested to flex and extend the great toe (passive and active examination) while palpating the course of the FHL once this might help in the identification of FHL pathology.

A neurologic examination should be performed to exclude tarsal tunnel syndrome, once the pain might also be caused by Valleix's sign (proximal tingling and plantar paraesthesia when tibial nerve is percussed posterior to medial malleolus).

In most cases, imaging is used for differential diagnosis or preoperative planning [3, 22].

In standard X-rays, standing anteroposterior (AP), mortise, and lateral views of ankle joint are routinely used. The lateral view is the most helpful to assess the hindfoot (e.g., Stieda process, *os trigonum*, osteophytes, loose bodies, chondromatosis, subtalar coalition). However, more recently, the posterior impingement (PIM) view has shown to be more effective [23]. The PIM view is a lateral, 25° exo-rotation, oblique view of the ankle, which has shown significant superior diagnostic accuracy compared with the lateral view in the detection of *os trigonum* or other bony causes of PAI.

In a study from the Amsterdam School, the mean sensitivity and specificity of the lateral view were 50% and 81%, respectively. For the PIM view, these were 78% and 89%, respectively [21].

CT is considered gold standard having higher sensitivity for bony impingement [10, 14, 15, 22, 24–26] (particularly small ossicles, loose bodies, painful broken osteophytes. or missed fractures (e.g., Cedell's fracture) (Fig. 18.6). It is also a valid resource for preoperative planning concerning joint's bone morphology.

Ultrasound provides dynamic assessment of the hindfoot, besides being operator dependent, and is often more useful in soft-tissue pathologies [22].

MRI (Fig. 18.7) for PAI will show edema of bone or surrounding soft tissue (T2 images are the most valuable in this setting) [22]. Moreover, it enables assessment of several anatomic variations including accessory muscles, labral pathology, synovitis, cysts, capsule, or ligament's changes [16, 27, 28].

However, in some cases, imaging might fail in providing definitive diagnostic. Currently, the arthro-endoscopic approach enables minimally invasive assessment of the hindfoot while providing a tool for final treatment [2, 9].

18.2.3 Mechanisms of Injury

Mechanisms of injury in PAI can basically be divided as overuse or blunt trauma.

It must be considered that, in some movements/technical gestures (e.g., kicking a ball), both the anterior (by direct trauma) and posterior (indirect forces with repeated strain on the hindfoot) ankle compartments are affected [29, 30]. Indirect recurrent loading of both compartments can occur in cycling. On the other hand, ballet dancing in *pointe/demi-point* or gymnastics might load more frequently the hindfoot through indirect forces (Fig. 18.5).

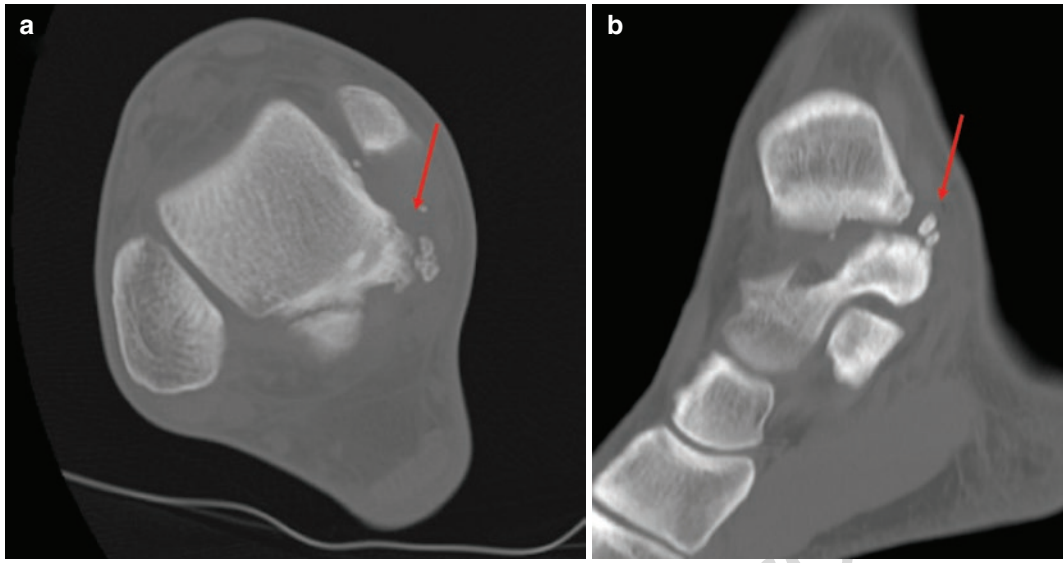

Fig. 18.6 (**a**) Axial and (**b**) sagittal CT views of a Cedell's fracture (fracture of the medial tubercle of the posterior process of the talus with risk for FHL entrapment)

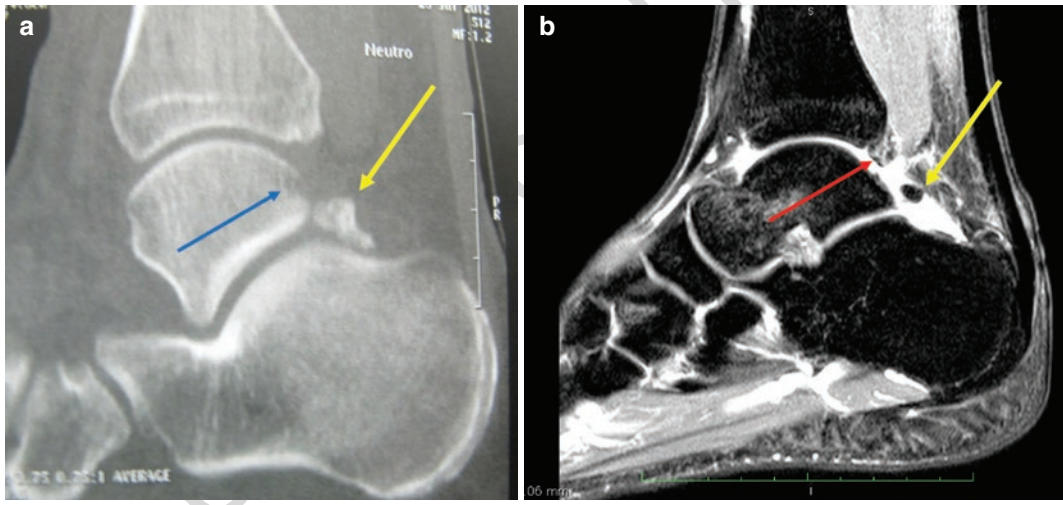

Fig. 18.7 (**a**) CT sagittal view in which is visible the talar impression (blue arrow) caused by posterior impingement by an *os trigonum* (yellow arrow); (**b**) MRI showing hypertrophic intermalleolar ligament (red arrow) and effusion surrounding *os trigonum* (yellow arrow)

PAI can sometimes be found combined with anterior impingement syndrome, ankle instability, or other joint pathologies. So, differential diagnosis must be considered. Other causes of posterior ankle pain include Achilles tendon or *tibialis posterior* pathologies, peroneal sheath contents, tibial or sural nerve lesions, as well as ankle or subtalar primary joint lesions (from osteochondral defects to arthritis) [2, 3, 5, 27].

Repetitive microtrauma can be due to specific activities and gestures but might also be linked or aggravated by combined chronic ankle instability [5] which facilitates repetitive strains in the posterior ankle compartment. As previously referred, examples of sports-related and gesture-related PAI include kicking the ball in football, ballet dancing (mainly due to *pointe* or *demi-pointe* positions), cycling, swimming,

acrobatic gymnasts, amongst others [3, 5, 6, 13, 18, 27, 30, 31].

The rapid direction and step's changes in addition to landings from falls, collisions, and jumps present players with high injury risk during sports. These maneuvers, which are key elements of the sport at the top level, produce high loads to the hindfoot, frequently exceeding the mechanical resistance of the ankle joint [32, 33].

D'Hooghe et al. have shown an increased likelihood for surgical treatment amongst high-level athletes with combined chronic ankle instability and posterior impingement related to *os trigonum* syndrome [19]. This might be due to the demands of this specific sports, combined with the consequences of joint instability which is known to affect globally the biomechanics of the ankle [28, 29, 31, 34, 35].

Another possible cause of PAI is major traumatic events including higher energy single trauma (e.g., hindfoot fractures or dislocated *os trigonum*) [2, 13, 14].

18.3 Types of Posterior Ankle Impingement (PAI)

Several conditions have been identified as possible sources of PAI.

Two major groups of pathologies were identified as symptomatic PAI requiring surgical treatment (according to Ribbans et al.): bony PAI (81% of surgeries) and soft-tissue PAI (42%) [6]. However, causes related to joint changes in the ankle and subtalar joints should also be considered [6]. The most frequent cause enrolling soft-tissue etiology is related to flexor hallucis longus (FHL) pathology [6, 15, 27]. A summary is provided in Table 18.1, based on Ribbans et al. [6].

18.3.1 Bony Posterior Impingement

The bony structures possibly involved in PAI are located in the tibio-calcaneal interval. Such structures include the posterior malleolus, the posterior talar process (Stieda process), an *os trigonum*, the posterior subtalar joint structure, and the posterior calcaneal tuberosity [3, 5, 10, 26]. Shepherd's fracture is still considered whenever fracture occurs of the posterolateral talar process.

Bony impingement seems to be twice more frequent than any causes related to soft tissue [6].

18.3.2 Soft-Tissue Posterior Impingement

Soft-tissue posterior impingement enrolls cysts (Fig. 18.8), hypertrophy of posterior intermalleolar ligament, "labral injuries" (Fig. 18.9), *flexor hallucis longus* pathology, and anomalous muscles (anatomical variation inducing hindfoot overload/overstuffing) [5, 6, 16]. Amongst the causes of soft-tissue-related, hypertrophic or damaged posterior ligaments including the posterior intermalleolar ligament (Fig. 18.10) or tibiotalar component of the deltoid are sometimes difficult to assess by preoperative imaging but must be kept in mind [36].

18.3.3 Ankle and Subtalar Joint-Related Posterior Impingement

The bone morphology of the posterior ankle and/or subtalar joints can cause symptomatic PAI. Despite not very frequent, the posterior tibial plafond slope can be implicated [6]. Osteophytes or loose bodies, possibly connected to joint degeneration (Fig. 18.11), are another source of PAI. Golano et al. have described particularly the possible entrapment of the posterior intermalleolar ligament (besides being a soft-tissue impingement, it can also be considered as related to joint pathology—for this reason this and other causes might appear intentionally repeated in Table 18.1) [36].

18.4 Principles of Treatment of Posterior Ankle Impingement (PAI)

Either enrolling, bony, or soft-tissue causes overuse or direct trauma the principle of treatment is reducing mechanical impingement and recurrent inflammation. The clinical prognosis appears to be better in those presenting with overuse injuries rather than trauma [1, 19, 20].

AU3

Table 18.1 Classification of possible causes of posterior ankle impingement

Causes of posterior ankle impingement		
Bony causes	Soft tissue	Ankle and subtalar joints
• Hypertrophic posterior talar process • Os trigonum • Shepherd's or Cedell's fractures • Accessory ossicles • Ossification of FHL tunnel • Sequelae of posterior malleolus fractures (malunion, avulsions, periosteal thickening) • Syndesmotic lesions (including avulsions) • Chondromatosis	• FHL-related pathologies: – Tendinopathy/synovitis – Stenosing tenosynovitis – Low ridding belly – Scars/adhesions – Tears – Nodules – Ossicles • Hypertrophic or damaged posterior ligaments • Synovitis/posttraumatic thickened capsule • Accessory muscles – Peroneocalcaneus internus – Tibiocalcaneus – Peroneus quartus • Cysts/ganglions	• Increased slope posterior tibial plafond • Loose bodies • Osteophytes • Pseudomeniscus syndrome • Synovitis/posttraumatic thickened capsule • Hypertrophic or damaged posterior ligaments

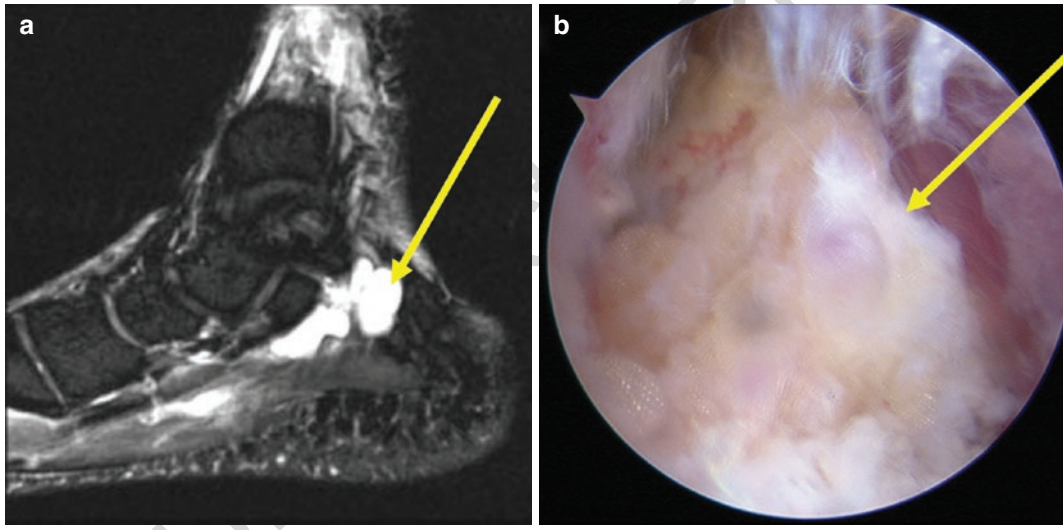

Fig. 18.8 (**a**) MRI showing cyst with fluid causing impingement (yellow arrow); (**b**) arthroscopic view of the same cyst (yellow arrow) causing soft-tissue impingement

18.4.1 Conservative Treatment

Conservative treatment of PAI includes rest, modification of shoe wear, change of activity, orthoses, physiotherapy, anti-inflammatory drugs, and ultrasound-guided injections [6]. Biologics including hydrogels (hyaluronic acid), growth factors (e.g., platelet-rich plasma), and stem cells (e.g., concentrated bone marrow aspirate) may be used even without any evidence supporting it [37]. However, these biologic agents are becoming recognized as promising adjuvants that may have a positive impact on tissues and decrease the inflammatory responses [15, 37, 38].

Ultrasound-guided injections may be useful in high-level athletes for transient symptom relief, possibly enabling them to finish the competitive season [39].

Although there is lack of published evidence concerning the success rate with conservative treatment [6], a small cohort study reported around 60% of success rates following conservative treatment in PAI [40].

18.4.2 Surgical Treatment

Upon failure of conservative measure, surgical resolution of the mechanical conflict with/without removal of inflammatory tissue is required.

According to Ribbons et al., 81% of patients required surgical excision when osseous pathology was involved and 42% when soft-tissue problems were implicated [6].

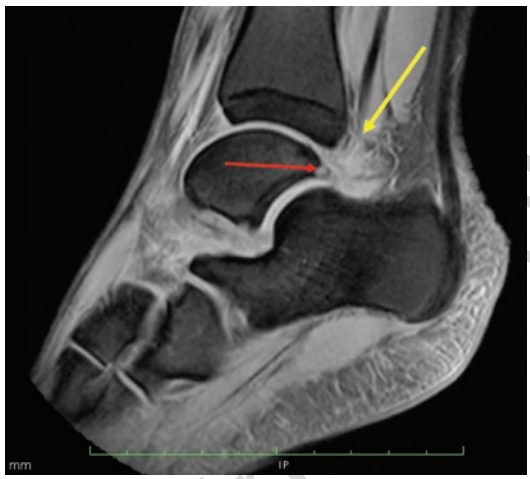

Fig. 18.9 MRI showing labral lesion (yellow arrow) and posterior talar process edema (red arrow)

There is a poor standardization of outcome reported in literature concerning the different treatment options which impairs definitive conclusions. However, the complication rates [41] are quite low for both open medial and arthro-endoscopic surgery [42, 43] (around 4% or less for endoscopy).

However, the chance for complications is three times higher (12%) for open lateral approaches [6]. So, this option must be carefully considered and limited in its indications.

Earlier return to activities, including all levels of sports, has been reported in the arthro-endoscopic group [11, 13, 16, 18, 19, 25, 27, 28, 35, 44, 45]. However, there is insufficient evidence supporting differences on the long-term outcome of one approach over the other [6, 11, 13, 16, 18, 19, 25, 27, 28, 35, 44, 45]. Football players apparently return faster to same level of previous activities when compared to dancers [6, 8, 11, 13, 16, 18, 19, 25, 27, 28, 35, 44, 45]. As previously stated, there seems to be an increased likelihood for surgical treatment amongst high-level athletes with combined chronic ankle instability and posterior impingement related to *os trigonum* syndrome [19].

The two-portal endoscopic approach for the hindfoot described by Van Dijk et al. represented a huge step forward in the surgical treatment of PAI [12]. It enables addressing bony or soft-tissue impingement in a reproducible, safe, and adaptable method [1, 3, 6, 14, 25, 44–46]. The arthroscope is placed first in the lateral portal aiming for the first interdigital space. Afterwards, the shaver is introduced in the medial portal at 90° with the arthroscope and it slides along its course until hit-

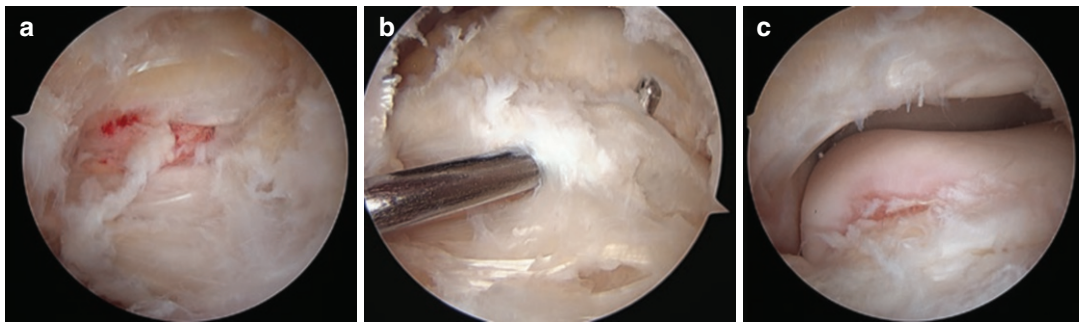

Fig. 18.10 (**a**) Arthroscopic view of fibrosis and synovitis; (**b**) hypertrophic intermalleolar ligament; (**c**) final look after endoscopic removal of soft-tissue impingement

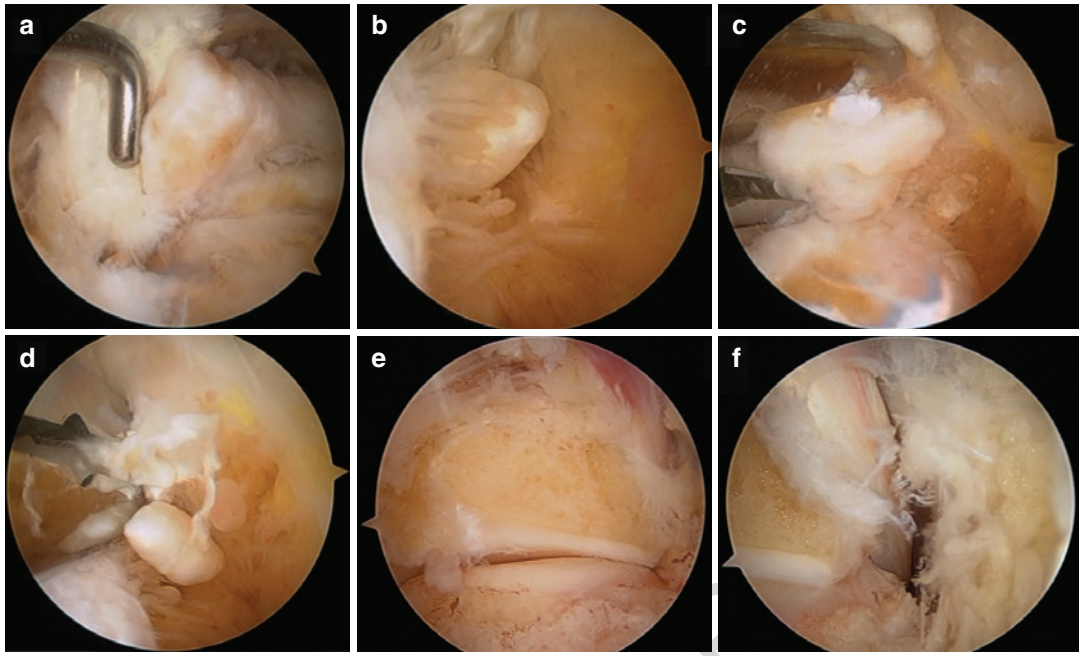

Fig. 18.11 Ankle and subtalar joint degeneration causing *flexor hallucis longus* (FHL) entrapment (**a**), several osteophytes and loose bodies (**b**–**d**) and final look of the cleaned subtalar joint (**e**), and cleaning and release of FHL sheath (**f**)

ting on solid bone. The scope is gently and slightly pulled back keeping its orientation and the shaver slightly advanced. The shaver is now visible. A few turns of the shaver blade will create an opening in Rouviére and Canela ligament. The arthroscope is afterwards introduced through this opening and the hindfoot and subtalar joint become visible. The next step will be to find the FHL which represents the medial border of the safe working area (lateral to it). Keep in mind that the neurovascular bundle is medial to the FHL. The hindfoot is now available for assessment and treatment [1].

The arthro-endoscopic approach lowered the surgical aggression enabling an outpatient procedure for most cases with early weight bearing (from day 1 if tolerated) and active range of motion [1, 3, 6, 14, 25, 44–46]. Several authors highlight the relevance to start active dorsiflexion-plantar flexion exercises as soon as possible (from day 1) [2, 5, 9, 43]. Stiches are removed around weeks and full return to activity is possible within 4–6 weeks for most isolated procedures [11].

The knowledge of hindfoot anatomy [36] is essential and the step-by-step technique has been described elsewhere [1]. Whenever it is the case, effort shall be made to remove the *os trigonum* in one piece aiming to avoid leaving small loose bodies behind (Fig. 18.12).

Sometimes, besides the clinical diagnosis, imaging fails to provide the etiology of the complains. This is the case of loose bodies which can move around the hindfoot. In such cases, the diagnosis can be made by endoscopic approach with immediate treatment. However, patients must receive information and agree with the approach (Fig. 18.13).

Taking a look on the future, the good results and low complication's rate of posterior endoscopic approach have provided a valid source of tissue (cells, autograft) from *os trigonum* or posterior talar process either for transplantation or advanced tissue engineering approaches [47].

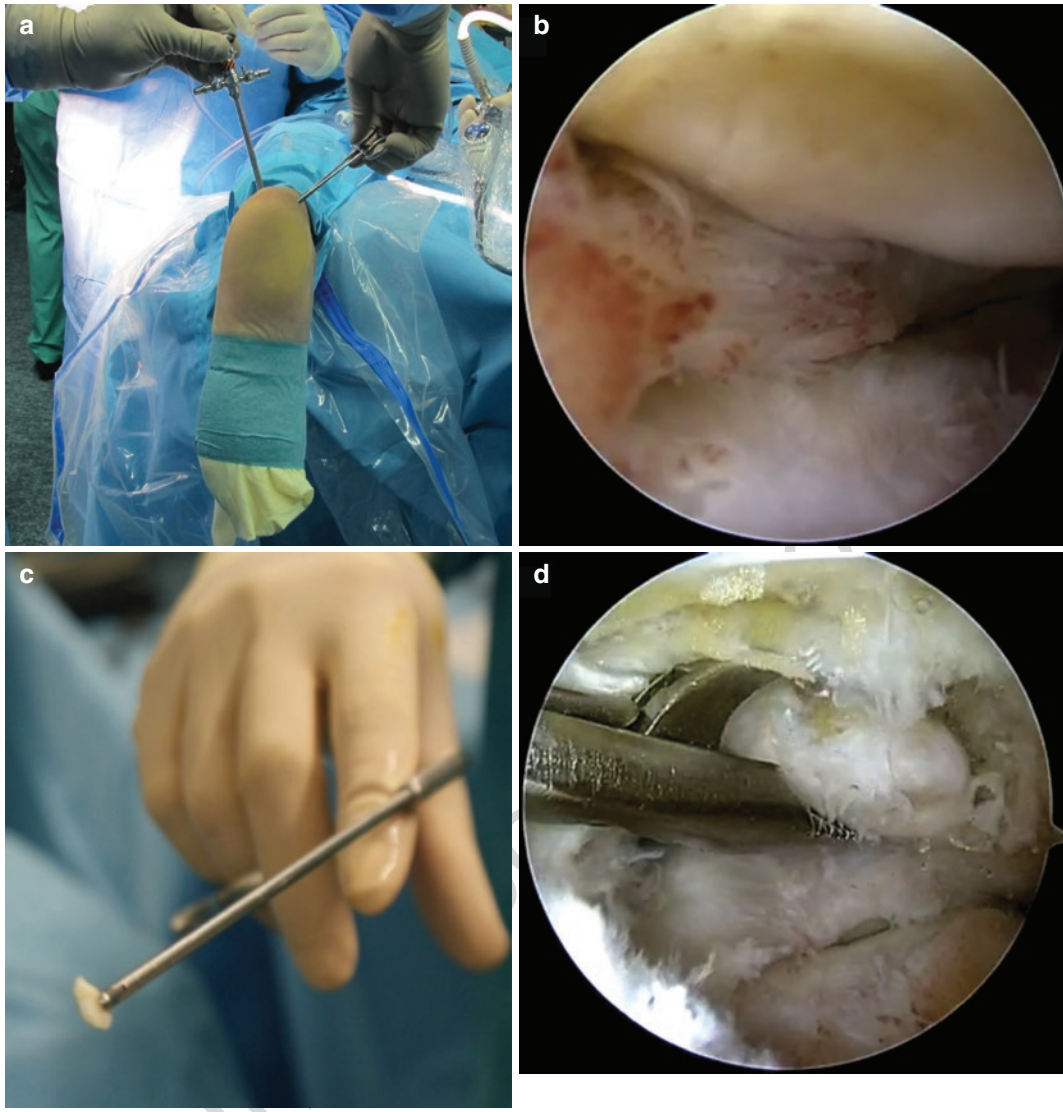

Fig. 18.12 (**a**) Standard two-portal endoscopic approach; (**b**) bony and soft-tissue impingement with synovitis; (**c**) *os trigonum* removal in one piece (external view); and (**d**) arthroscopic view showing the grasper holding the *os trigonum* after its liberation

18.5 Take-Home Messages

The posterior ankle impingement (PAI) is a clinical syndrome which involves entrapment of some structure between the calcaneus and the posteroinferior aspect of the tibia.

Posterior impingement syndromes are mostly based on clinical diagnosis, with posterior impingement test playing a role in clinical evaluation. History of hindfoot pain aggravated by plantar flexion of the ankle reinforced by a positive plantar flexion test provides the diagnosis of PAI. However, global assessment including alignment and biomechanics of foot and ankle is mandatory.

It is more frequent to find PAI in athletes (mainly sports which require frequent ankle plantar flexion) than in the general population.

PAI is one of the most frequent causes for absence of activity related to foot and ankle for

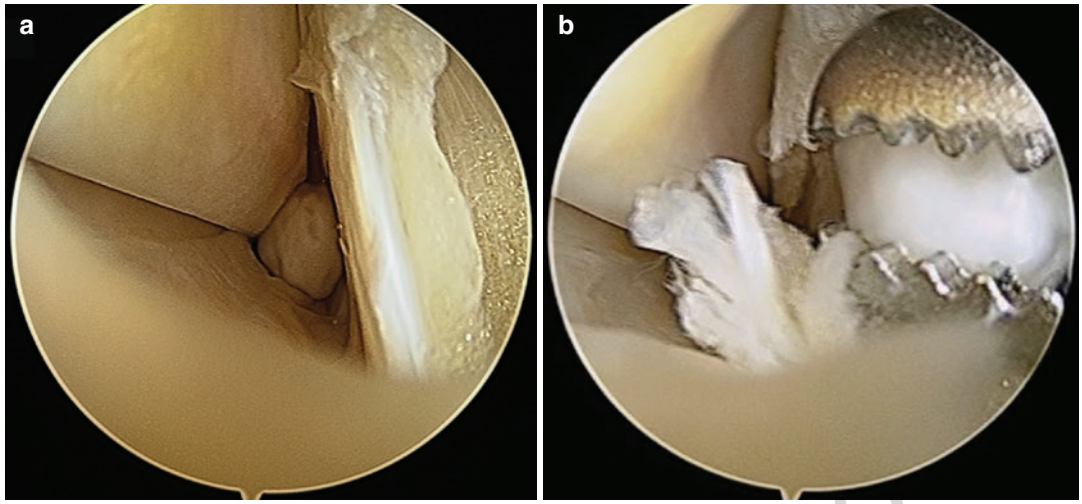

Fig. 18.13 A case of recurrent hindfoot pain in a football player with sudden onsets of pain in different spots. No relevant changes could be identified in preoperative planning. (**a**) Endoscopic view where a loose body is seen inside *flexor hallucis longus* tunnel; (**b**) removal of the loose body and cleaning of some tendon's damage. The patient became asymptomatic and returned to the pitch at 4 weeks

ballet dancers, footballers, cyclists, swimmers, acrobatic gymnasts, and downhill runners.

Bony impingement appears to be much more frequent than soft-tissue causes of PAI.

Imaging might be helpful in preoperative planning.

CT has higher sensitivity for bony impingement (particularly small ossicles or missed fractures). MRI and ultrasound can be more helpful for soft-tissue-related causes.

Etiology can be overload by repeated microtrauma or traumatic events. Combined chronic lateral ankle instability seems to increase the need for surgical treatment of PAI.

Usually, the first treatment approach is conservative treatment.

Arthroscopic/endoscopic approach enables high percentage of good results with minimal complications and fast return to activity for most causes of PAI.

The outcome seems to be better in those presenting overuse injuries when compared to traumatic causes.

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Fact Box 1: Posterior Impingement Epidemiology and Mechanisms of Injury (PAI)

- There is low evidence on incidence and prevalence of PAI.
- It is more frequent to find PAI in athletes than in the general population.
- PAI is often observed in ballet dancers, footballers, cyclists, swimmers, acrobatic gymnasts, and downhill runners.
- PAI was the cause for 31% of all days lost for sports activity due to foot and ankle conditions, which was higher than lateral ankle ligament injuries and Achilles disorders combined.
- Bony impingement appears to be two times more frequent when compared to soft-tissue causes of PAI.
- Usually repeated microtrauma in plantar flexion is the most frequent mechanism (e.g., dancers, footballers).
- Mechanisms include repetitive overload or blunt trauma.
- Anatomy seems to play a role.
- An asymptomatic *os trigonum* might became painful after an ankle sprain—"There is no such thing as a simple ankle sprain."
- Trauma or "hidden" hindfoot fractures must be ruled out.

Fact Box 2: Diagnosis of Posterior Ankle Impingement (PAI)

- PAI is, by definition, a clinical diagnosis.
- Imaging might be helpful in preoperative planning.
- CT has higher sensitivity for bony impingement (particularly small ossicles or missed fractures (e.g., Cedell's fracture).
- MRI will show edema, of bone or surrounding soft tissue (T2 images are the most valuable in this setting).
- Ultrasound provides dynamic assessment of the hindfoot, despite being operator dependent.

Fact Box 3: Types of Posterior Ankle Impingement (PAI)

- Bony or soft-tissue causes are possible.
- Os trigonum syndrome, hypertrophic posterior talar process, or flexor hallucis longus-related pathologies are the most frequent causes.
- Some cases combine both.
- Ankle and subtalar joint pathologies are also included.
- Anatomical variations might play a role.

Fact Box 4: Treatment Options for Posterior Ankle Impingement (PAI)

- Conservative treatment is usually the initial option for treatment of PAI.
- Arthroscopic approach of bony or soft-tissue impingement is the rule upon failure of conservative treatment.
- Arthroscopy/endoscopy enables outpatient clinical treatment, normally with immediate active range of motion and weight bearing. Full return to activity is usually achieved between 4 and 6 weeks.
- It is very important to start active dorsiflexion-plantar flexion as well as weight bearing in order to prevent stiffness and provide proprioceptive stimulus (prevention of complex regional pain syndrome).
- Open approach is also a possibility (depending on surgeon's experience or specific cases) and medial approach has lower morbidity when compared to lateral approach.

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AU1	Please check and confirm if the author names and affiliations are presented correctly.	
AU2	References have been sorted to maintain sequential order. Please confirm if correct.	
AU3	Please check the presentation of Table 18.1 and correct if ncessary.	
AU4	Please check the presentation of Box contents are fine or correct if necessary.	

19 Advanced Techniques in Arthroscopy of the Foot

Alastair Younger, Andrea Veljkovic, Michael Symes, and Wafa Al Baluki

19.1 Introduction

Less invasive techniques result in smaller wounds. This will reduce postoperative swelling and pain. This has been shown in ankle arthritis to potentially improve outcomes and reduce length of stay. Other benefits may include reduced narcotic utilization, less wound complications, and less hematoma formation. It may also improve the blood supply around the surgical site to bone. Better cartilage removal may occur in fusions. Cartilage has growth factors preventing new bone formation.

Prior to performing arthroscopic procedures surgeons should be familiar with and be able to perform the open procedures well. The surgical goals with minimally invasive and arthroscopic procedures still need to be achieved.

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19.2 Subtalar Arthroscopy

19.2.1 Indications

Subtalar arthroscopy is of use in removing cartilage to perform subtalar, pantalar, and triple fusions. Arthroscopy reduces the dissection involved. A symptomatic os trigonum can be removed through the subtalar joint to avoid the dissection of a posterior approach. Fractures extending into the subtalar joint involving either the talus or the calcaneus can be percutaneously reduced and transfixed, with arthroscopy being used to assist in understanding the fracture pattern and assessing the reduction once performed. Impingement on the anterior lateral side of the posterior facet can be addressed as part of subtalar arthroscopy. Subtalar arthroscopy can be performed in conjunction with sinus tarsi debridement and peroneal tendoscopy.

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19.2.2 Positioning

The patient is positioned either prone or supine depending on surgeon preference and other pathology needing to be addressed.

For supine positioning the patient is placed on a beanbag-positioning device. The hip needs to be internally rotated so that the lateral side of the Achilles tendon can be accessed as a portal. In some cases the patient may need to be positioned laterally. A calf or thigh tourniquet is used. Regional, spinal, or general anesthesia can be used and this may change the choice of tourniquet.

For prone positioning the same anesthetic and tourniquet alternatives exist. The posterior aspect of the subtalar joint is easily accessed through portals on each side of the Achilles tendon. However anterior ankle arthroscopy or sinus tarsi debridement is harder to perform in this position.

19.2.3 Technique

- Equipment: 2.9 scope, 3.5 shaver, 4.5 burr, osteotomes, curettes, thigh or calf tourniquet.
- Portals:
  - As many portals to access the joint as needed are performed.
  - The simplest way to start a subtalar arthroscopy is to start with a sinus tarsi approach. The arthroscope can be inserted dorsally in the sinus tarsi next to the talar neck aimed down towards the floor of the sinus tarsi. The shaver is inserted in from a direct lateral approach to the floor of the sinus tarsi. The shaver is in a safe position from the skin nerves, and will be visible from the scope. The shaver can then be used to debride the sinus tarsi and achieve visualization and the contents of the sinus tarsi as well as the anterior and lateral portion of the subtalar joint can be seen. The peroneal tendons can also often be seen from this portal.
  - The third portal lies just posterior to the subtalar joint in the recess behind the fibula, and above the calcaneus, behind the talus, and below the tibia. The lateral and posterior side of the joint can be seen from this portal.
  - The final portal for use in the supine position lies just lateral to the Achilles tendon. The posterior and medial side of the subtalar joint can be seen and visualized from this approach.

19.2.4 Procedure

- Impingement:
  - For impingement the sinus tarsi approach is first used. The shaver is used to remove the synovium and the interosseous ligament if need be. Once visualized a burr can be inserted in from the lateral sinus tarsi portal and used to remove the anterior lateral osteophytes. The direct lateral osteophytes can also be removed by aiming the shaver posterior instead of medial. Once the anterior and lateral side has been debrided the posterior and medial osteophytes can be removed by sequentially switching to the posterior portals. The scope is therefore moved from the dorsal sinus tarsi portal to the lateral sinus tarsi portal, and to the posterior lateral portals.
  - While the margins of the joint can be reached, it is rarely possible to reach an osteochondral injury within the joint to debride it unless it is small, well contained, the rest of the joint is in good condition and the lesion is towards the joint edge. Sometimes this type of cartilage defect exists after a lateral talar process fracture.
- ORIF:
  - Arthroscopy of the subtalar joint can be used to assist reduction in a lateral talar process fracture, a posterior talar process fracture, a medial talar fracture extending into the subtalar joint, or a calcaneal fracture. The benefit of this approach is that the soft-tissue attachments are left in place and the hardware can be correctly placed into the fracture fragment.
  - Anterior lateral process fracture:
  - For this fracture the fragments need to be large enough to transfix. The soft-tissue attachments are left intact and the fracture fragment reduced and held with a K wire.

The reduction is confirmed with arthroscopy and fluoroscopy. Sometimes a pelvic reduction forceps is beneficial if placed medially anterior to the FHL tendon to hold the fragment in place. 2 mm or 2.7 mm solid screws can be used to maintain fixation with an appropriate arch to prevent penetration of the joint.

- Posterior fractures and medial process fractures can be visualized in the prone or supine position. These can be reduced using either K wires or a pelvic reduction forceps. The screws are then placed with the assistance of the scope from the stable to the unstable segment.
- For calcaneal fractures the space to enter the subtalar joint exists with the joint depression. The fracture hematoma is removed and the fracture fragments are visualized. Once seen the fracture fragments can then be reduced using plantar medial and plantar lateral portals, as well as using traction on the tuberosity segment. Provisional fixation can be achieved using K wires. The fracture reduction can be assessed from the joint margin and by viewing the angle of Gissane.

- Arthroscopic fusion:
  - For arthroscopic fusion the above portals are used to visualize the joint. The cartilage is then sequentially removed. The anterior and lateral sinus tarsi approach can initially be used and once the joint is visualized using a shaver a burr can be used to then start removing cartilage from the posterior facet. Approximately 50% of the cartilage in the posterior facet can be removed from these two portals, and once cartilage has been removed the arthroscope can be advanced into the joint. All the cartilage should be removed as it may inhibit bone healing. The medial cartilage can only be removed using the posterior portal next to the Achilles tendon. The joint is then held in a neutral position and screw fixation achieved.

19.3 Calcaneocuboid Arthroscopy

19.3.1 Indications

Synovitis, dorsal chondral injury, acute fracture reduction, removal of fracture fragments either acutely or in symptomatic nonunion of the anterior calcaneal process, calcaneocuboid fusion in isolation or as part of a triple arthrodesis, arthroscopic resection of calcaneonavicular coalition or its variants, ganglion cysts, and CC impingement [1].

19.3.2 Positioning

The patient is placed either in the lateral position with the surgical side superior or in a supine position with the surgical leg internally rotated. A calf, sterile calf, or thigh tourniquet can be used depending on anesthetic choice.

19.3.3 Technique

- Nick spread portal technique is used.
- Instrumentation: 30° 2.7 mm, 2.4 mm, 1.9 mm arthroscope for visualization, 2 or 3 mm arthroscopic shaver, 4.5 mm burr.
- Portals:
- Three portals can be used to visualize the calcaneocuboid joint (Fig. 19.1):
  - The direct lateral portal is at the level of the joint line and half way from the dorsal to the plantar side. The sural nerve may be close so deep dissection should be blunt.
  - The direct dorsal portal is at the joint line and lies just next to the lateral side of the navicular and talar neck.
  - A dorsal lateral portal is considered the most important portal located between these two other portals. Fluoroscopy can be used to guide localizing this portal. It is directly over the space between the TN and CC joints. Lateral branch of superficial peroneal nerve and the lateral terminal branch of the deep peroneal nerve are at risk [1, 2].

19.3.4 Procedure

- Fracture reduction:
  - The arthroscope is placed in the lateral portal and the fracture site visualized. The fracture is reduced using K wires and the fracture reduction reviewed. If satisfactory then dorsal to plantar screws can be placed. Accessory portals may be required.
- Dorsal osteophyte removal:
  - On occasion a dorsal osteophyte can either overhang the calcaneocuboid joint or impinge. The osteophyte can first be visualized using a shaver from the dorsal portal with the arthroscope placed in the lateral portal. Once visualized the osteophyte is then removed under visualization with a burr. Once resected the resection can be checked using the arthroscope and imaging. A similar approach is used to remove fracture fragments that usually lie dorsal and proximal to the calcaneocuboid joint.
- Chondral injury:
  - Chondral injuries can occur on the dorsal side of the calcaneocuboid joint. A curette can be used to debride the cartilage back. The bone margin can also be removed using a burr with removal of the bone back to healthy cartilage. This is equivalent in principle to a dorsal cheilectomy of a first MTP joint.
- Fusion:
  - Arthroscopic fusion of the calcaneocuboid joint can be achieved with the same portals described above. A burr can be placed from the dorsal and lateral side to sequentially remove cartilage. The plantar and medial cartilage should be removed to ensure solid fusion. Screws can be placed percutaneously either from the anterior process of the calcaneus to the cuboid or from the cuboid back. Full thread headless screws are helpful and the trajectory of the screw needs to be correct to prevent medial penetration on the cuboid. For isolated calcaneocuboid joint fusion three screws are likely needed.

19.4 Talonavicular Arthroscopy

19.4.1 Indications

The talonavicular joint is very congruent and tight. It is therefore difficult to perform a procedure without cartilage removal. Some of the conditions where arthroscopy can be used are fusion, dorsal cheilectomy, and fracture reduction.

19.4.2 Positioning

Patient is positioned supine. The arthroscopy tower is placed on the nonsurgical side next to the head of the operating table. Tourniquet can be used according to the surgeon preference [1].

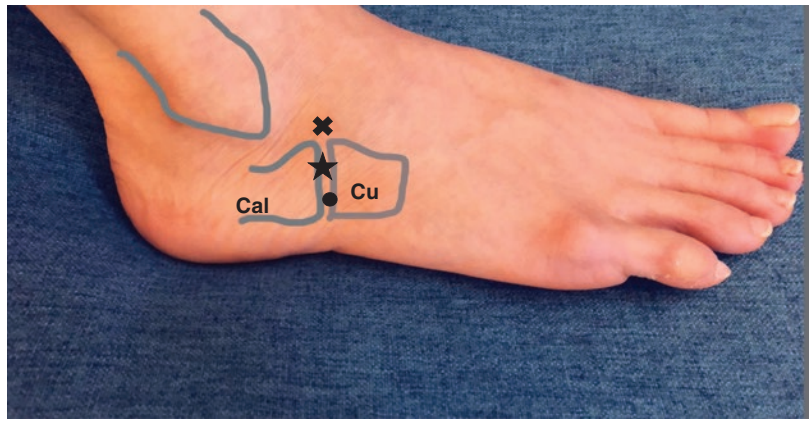

Fig. 19.1 Calcaneocuboid joint arthroscopy: • direct lateral portal, + direct dorsal portal, ★ dorsal lateral portal. *Calc* calcaneus, *Cu* cuboid bone

19.4.3 Technique

- Portals:
- Three portals are usually used for the TN joint (Figs. 19.2 and 19.3):
  - Dorsal portal to the calcaneocuboid joint and also acts as a lateral portal to the TN joint.
  - Plantar medial portal: located at the medial side of the TN joint, just dorsal to the posterior tibial tendon [1, 2].
  - Dorsal-medial: located midway between the medial and dorsolateral portals. This portal is in close proximity to intermediate cutaneous branch of superficial peroneal nerve and extensor hallucis longus tendon [1, 2].
  - Dorsal lateral portal can be placed at the level of the joint line on each side of the dorsal neurovascular bundle.

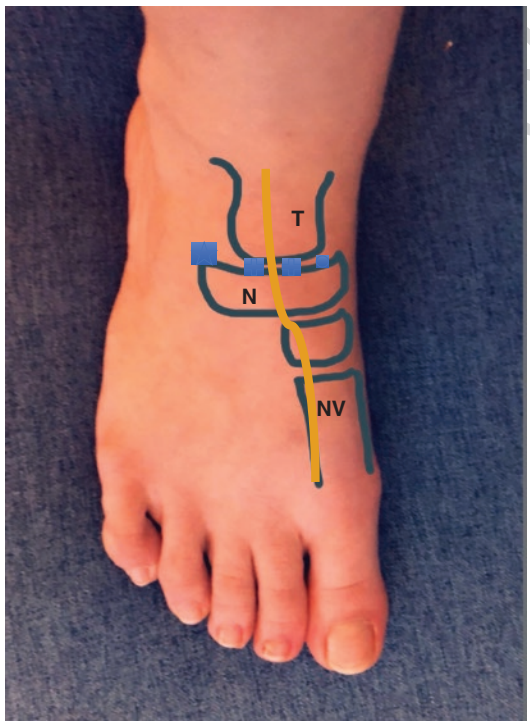

Fig. 19.2 Talonavicular arthroscopy portals: ★ lateral portal, + dorsal lateral portal, • dorsomedial portal. *T* talus, *N* navicular bone, *NV* dorsal neurovascular bundle

19.4.4 Procedure

- Fracture reduction:
  - On occasion a talar head or navicular fracture can benefit from an arthroscopic approach. The portals are used to visualize the fracture fragments to assist in reduction. The arthroscopic approach can assist the surgeon with reduction while maintaining the soft-tissue attachments that might otherwise be removed for visualization.
- Fusion:
  - Talonavicular fusion is a relatively easy arthroscopic procedure. Once the joint is entered the cartilage can be sequentially removed with a burr. Fixation is achieved percutaneously from the medial tubercle and from the dorsal lip distal to proximal into the talar head.
- Dorsal osteophyte removal:
  - Dorsal osteophytes can be removed using the dorsal medial and dorsal lateral portals. However extreme care must be taken to avoid damage to the deep peroneal nerve and dorsalis pedis artery that lie just superficial to the dorsal capsule.

19.5 Navicular Cuneiform Arthroscopy

19.5.1 Indications

Fusion and fracture reduction.

19.5.2 Positioning

Supine position. Tourniquet can be applied to either thigh or calf.

19.5.3 Technique

- Portals (Figs. 19.4 and 19.5):
  - The navicular cuneiform joint can be approached from the dorsal lateral, lat-

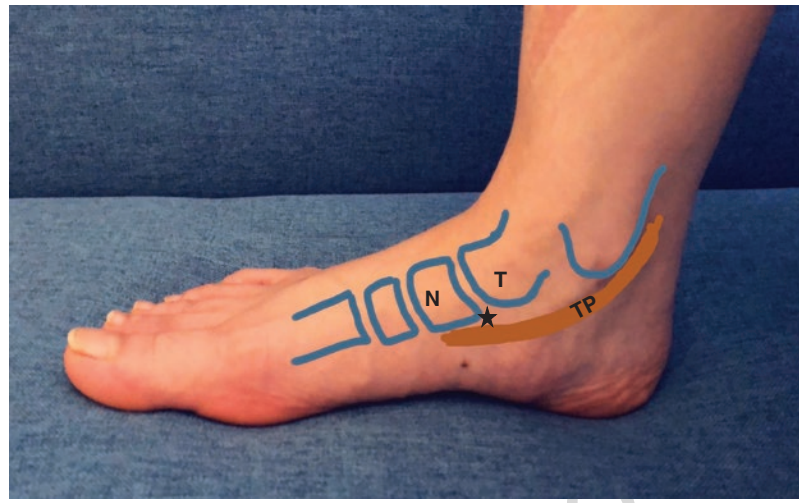

Fig. 19.3 Talonavicular arthroscopy: ★ plantar medial portal, *T* Talus, *N* navicular bone, *TP* tibialis posterior tendon

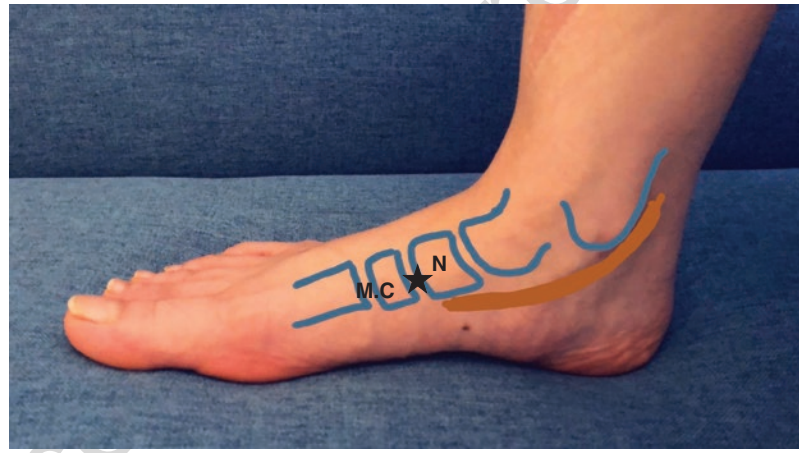

Fig. 19.4 Naviculo-cuneiform arthroscopy: ★ the medial portal, *N* navicular, *M.C* medial cuneiform

eral, dorsal medial, and medial sides. The medial portal is located over the plantar medial corner of the navicular-medial cuneiform joint. The dorsal-medial portal is placed at the junction between the medial and middle cuneiform. The dorsal-lateral portal is placed at the junction of middle and lateral cuneiform joint. The lateral portal is positioned at the lateral corner on the navicular-lateral cuneiform joint. The dorsalis pedis and deep peroneal nerve lie centrally dorsally and should be avoided.

19.5.4 Procedure

- Fusion:
  - Using the appropriate portals the joint margin is identified. This can be challenging and fluoroscopy images may be required. A curette can be advanced into the joint to assist in visualization. Once visualized the cartilage is sequentially removed from all three segments of the joint—the navicular to lateral, medial, and intermediate cuneiform joints. Once debrided the joint is held in a reduced position and cross screws are placed.

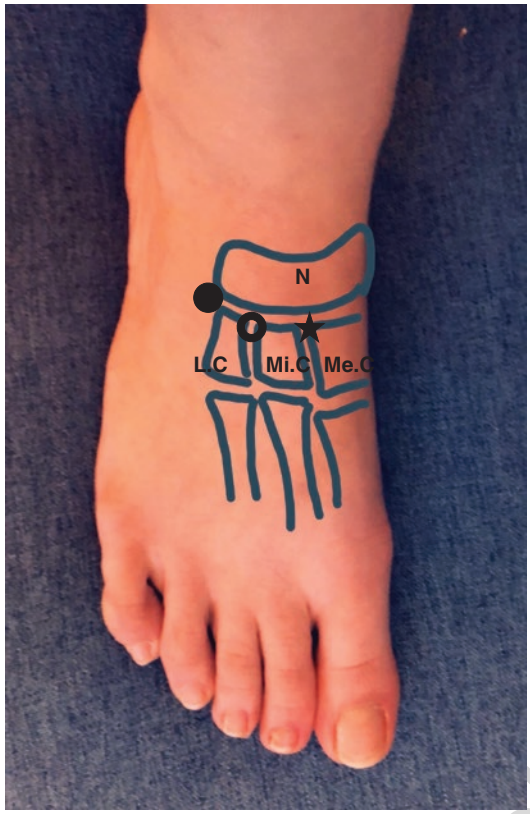

Fig. 19.5 Naviculocuneiform arthroscopy: ★ dorsal-medial portal, ○ dorsal-lateral portal, • lateral portal, *Me.C* medial cuneiform, *Mi.C* middle cuneiform, *L.C* lateral cuneiform, *N* navicular bone

Distal to proximal screws are more easily placed from each cuneiform. Proximal to distal screws are also placed from the tubercle of the navicular down to the cuneiforms.

- Fracture care:
  - On occasion a midfoot injury, subluxation, or dislocation involves the navicular cuneiform joint. The fracture or dislocation makes a space for arthroscope insertion. The injury pattern can be determined and reduction achieved using percutaneous techniques. The reduction can be maintained using either suture bridge constructs or percutaneous screws.
  - Once reduced and held the reduction can be checked using a periarticular technique. The joint lining is often stripped and this allows visualization of the reduction alongside the joint margin.

19.6 TMT Arthroscopy

19.6.1 Indications

Hypermobility of the first tarsometatarsal (TMT) joint associated with hallux valgus, transfer metatarsalgia, arthritis of the second TMTJ, and posttraumatic arthritis [1].

19.6.2 Positioning

Patient is positioned supine with application of thigh tourniquet [1].

19.6.3 Technique

First TMT portals: Identify the joint by moving the first metatarsal and locate the motion at the joint. If needed, fluoroscopy can be used to confirm the portal site. This is done by passing a 22-gauge needle to the proposed portal site and confirmed under fluoroscopy.

19.6.4 Portals (Figs. 19.6 and 19.7)

- Six TMT portals are described. The middle four portals (P1–4) are junction portals that can be used to approach the intercuneiform spaces and spaces between the proximal parts of metatarsal bones.
- Medial portal: located over the plantar medial aspect of the first TMTJ.
- P1–2 portal: located at the junction point between the medial cuneiform, first metatarsal, and second metatarsal bones. The first TMTJ can be approached using this portal.
- P2–3 portal: located at the junction point between the second metatarsal, intermediate cuneiform, and lateral cuneiform bones. This is used to approach the second TMT joint.
- P3–4 portal: located at the junction point between lateral cuneiform, cuboid, third, and fourth metatarsal bones. Third and fourth TMT joints can be approached using this portal.

- P4–5 portal: located between the proximal articular surfaces of the fourth and fifth metatarsal bones. This is used to visualize the fourth and fifth TMT joints.

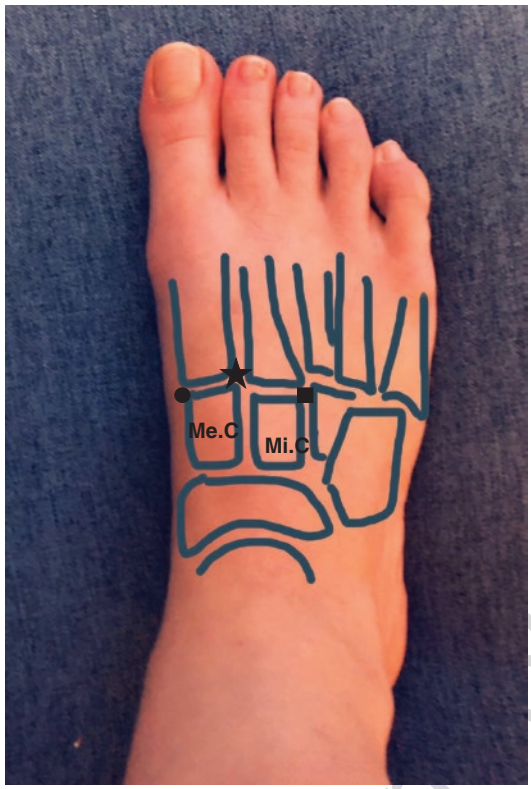

Fig. 19.6 TMT arthroscopy: • medial portal, ★ P1–2 portal, ■ P2–3 portal, *Me.C* medial cuneiform, *Mi.C* middle cuneiform

- Lateral portal: located at the lateral corner of the fifth metatarsal-cuboid articulation. This portal is used to approach fifth metatarsal-cuboid articulation, the insertion of the peroneus brevis tendon, and the peroneus tertius tendon [1, 2].

19.6.5 Procedure

- First TMT fusion (arthroscopic Lapidus procedure):
  - Two portals, plantar medial and dorsomedial, are utilized for this procedure. The cartilage is denuded using arthroscopic osteotomes, shaver, or a burr until the subchondral bone is exposed. Arthroscopic awl can be used to perform microfracture. Then, intermetatarsal angle is closed up manually and the first metatarsal is plantarflexed [1]. A temporary K wire can be used to hold the position and fluoroscopy is used to evaluate the alignment. Adjustment is made accordingly. For fixation, percutaneous screws are inserted. The usual construct is two antegrade screws inserted from proximal dorsal to distal plantar direction across the joint and one retrograde-directed screw inserted from the dorsal distal at the metatarsal base to proximal plantar. An additional positional screw can be used. It is inserted from the

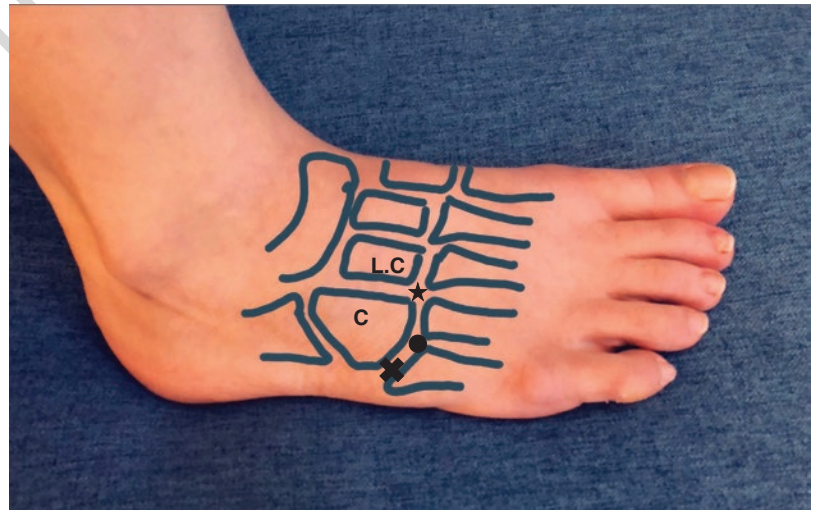

Fig. 19.7 TMT arthroscopy: ★ P3–4 portal, • P4–5 portal, + lateral portal, *L.C* lateral cuneiform, *C* cuboid

first metatarsal base to the second metatarsal base [1].

- TMT joint fusion:
  - Portals are chosen according to the joint being fused. Usually only the medial three TMTJs are needed to be fused. The corresponding joint spaces can be approached through the proper portal and joint preparation is done. Fusion surface preparation technique is similar to arthroscopic Lapidus procedure. After that, the joint reduction is performed in the desired position and percutaneous screw fixation is done according to the surgeon choice between cannulated and non-cannulated screws. If bone graft is to be used, it can be packed through the portals with the aid of a small drill sleeve [1].

19.7 First MTP Arthroscopy

19.7.1 Indications

MTP joint arthroscopy is a much easier procedure to perform than other midfoot or hindfoot arthroscopies [3]. Some of the conditions that can be treated arthroscopically are MTP joint fusion for MTP joint arthritis, synovectomy, removal of a painful sesamoid bone, partial cheilectomy for hallux rigidus, debridement of an osteochondritis dissecans defect, removal of loose bodies, arthroscopic drainage in septic arthritis, and assessment of the plantar plate in turf toe injury [2–6]. Contraindications: There are situations where arthroscopy may not be performed like in overlying cellulitis, in sever deformity preventing adequate visualization, or in joint malalignment that necessitates an osteotomy or other corrective procedures. Other contraindications are when plate is required for fixation or in Charcot arthropathy because normal anatomy is distorted [3, 6, 7].

19.7.2 Positioning

Supine position with a bump in the ipsilateral hemipelvis to hold the foot in neutral rotation [3, 6–8]. Traction is not required for this procedure [3, 6, 9] although some authors had reported applying traction to great toe. This can be in the form of attaching Chinese finger trap to the great toe [4, 7] with traction weight suspended from the pulley [4, 10]. Others have reported using moisten cotton bandage tied with tension loop over first web space. This is attached to distractor and tension adjusted accordingly [5]. Tourniquet is not necessary for this procedure [3, 4]. However, some authors prefer using calf tourniquet [2, 5, 6, 8]. Intraoperative fluoroscopy should be used to confirm the position of the portals in tight joint [3].

19.7.3 Technique

Skin incision is made using 11-blade scalpel. This is followed with blunt dissection of the subcutaneous tissue using a mosquito forceps that is used to penetrate the joint capsule. A blunt trocar is then introduced [3, 7]. Some authors reported injecting the MTP joint with 2–3 mL of normal saline prior to skin incision [2, 4, 5, 10].

19.7.4 Portals and Accessory Portals (Figs. 19.8 and 19.9)

- Different portal has been described to approach the MTP joint. The most common technique utilized is two-portal approach. In two-portal approach the portals are dorsomedial and dorsolateral portals located just medial and lateral to the EHL tendon at the level of the MTP joint, which is about 0.5 cm on both sides of EHL tendon [2–8, 10]. Both portals run close to the dorsomedial and dorsolateral cutaneous nerves of the great toe. In a cadaveric study by Vaseenon, these portals were about 3.4 mm and 4.0 mm from the dorsolateral and dorsomedial cutaneous nerves, respectively [7].
- Another described technique is three-portal approach. In this technique a third medial portal is added [4, 5, 7, 8]. This portal is centered over the medial side at the level of MTP join,

midway between the dorsal and plantar aspect of the joint. This technique was found to improve the amount of cartilage debridement on the distal metatarsal as shown by Vaseenon [7]. The medial portal lies about 10 mm medial to the dorsomedial cutaneous nerve and about 13 mm from the plantar medial nerve [7].

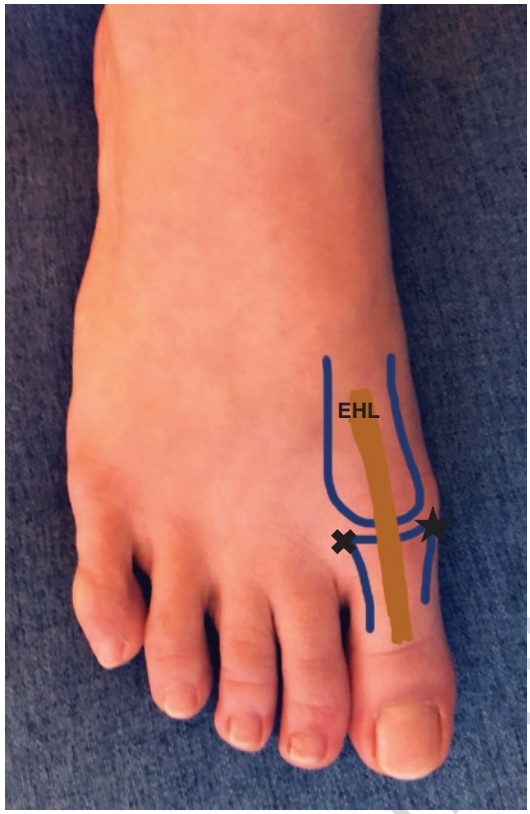

Fig. 19.8 First MTP joint arthroscopy portals: ★ indicates the dorsomedial portal. + indicates the dorsolateral portal. *EHL* extensor hallucis longus

- Central plantar portal: located over the plantar aspect and established by an inside-out method. From the web portal, trocar is inserted and advanced proximally along the deep surface of the intermetatarsal ligament till the trocar tip touches the plantar aponeurosis. Gently perforate the aponeurosis and advance the trocar. The plantar portal is made at this point [8].
- Proximal dorsomedial debridement of dorsal osteophytes.
- Web portal: located over dorsal side of first web space [8, 9]. If this portal is placed more towards the hallux, it might facilitate subsequent screw insertion [8].
- Lateral portal: utilized during arthroscopic lateral soft-tissue release in hallux valgus correction procedure [4]. Lateral portal: 1 cm lateral to the dorsolateral portal, at the level of MTP joint.
- Proximal portal: used in arthroscopic release of the arthrofibrosis and in arthroscopic excision of pathologic medial sesamoid [4].

19.7.5 Procedure

- MTP fusion:
  - Cartilage over the metatarsal head and at the base of proximal phalanx is removed using

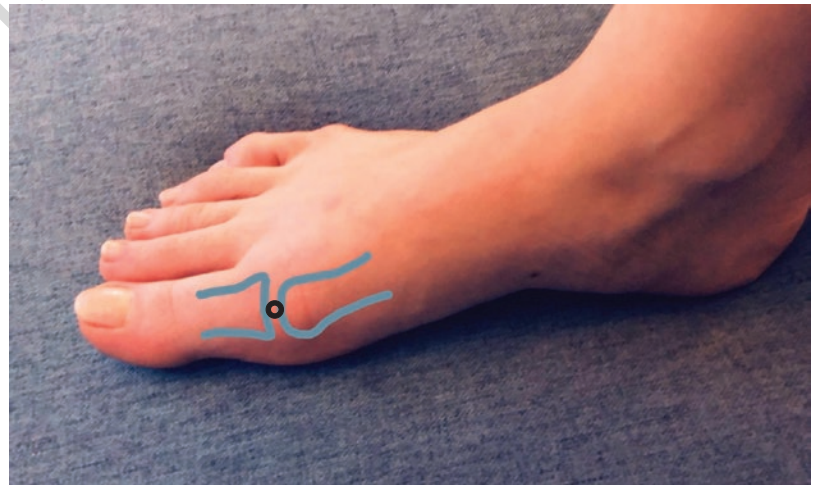

Fig. 19.9 First MTP joint arthroscopy portals: ○ indicates the medial portal

arthroscopic shaver [7]. Instrumentation and visualization can alternate between the dorsomedial and dorsolateral portals until debridement of the joint is achieved. In the three-portal approach, the medial portal is usually used for debridement only [7]. The position of the MTP joint is checked under fluoroscopy. Provisional fixation using K wires can be used to assist in holding the MTP joint in the desirable position for arthrodesis. Fixation is then achieved using percutaneously inserted headless or headed screws [3, 8].

- Lateral release in hallux valgus:
  - The procedure starts with synovectomy if needed. Then, release of lateral suspensory ligament is performed using small arthroscopic knife, to visualize the lateral sesamoid. Releasing of the lateral capsule and the adductor tendon follows this. Care should be taken to avoid injury to the lateral metatarsophalangeal ligament [4]. Others have reported utilizing web portal and central plantar portal to perform this release. The plantar portal is used as viewing portal and the web portal is used as the working portal [8].
- Arthroscopic release for arthrofibrosis in hallux rigidus:
  - J.H Ahn et al. reported on using three portals with proximal portal to perform the debridement. Through the dorsomedial and dorsolateral portals, excision of dorsal metatarsal and phalangeal spur is done using a burr or a shaver. All loose or delaminated articular fragments are removed. Visualization of medial and lateral gutters, sesamoids, and plantar plate is performed to identify any other pathologies. Then MTP joint is maximally dorsiflexed to ensure that there is no dorsal impingement [6].
- OCL debridement:
  - Using a shaver, synovial debridement is performed as needed to facilitate visualization. Confirm the location of OCL and evaluate the integrity of the underlying bone using a probe. A curette is used to debride the lesion until getting to more stable boarders. The stability of the lesion is reassessed using a probe. K wire can then be used to perform microfracture [5, 10].

19.8 Lesser MTP Arthroscopy

19.8.1 Indications

Arthroscopic interventions can be used in treating lesser toes osteoarthritis, synovitis, chondral lesion, arthrofibrosis, and instability. Corrective procedures of toe deformity involve excisional arthroplasty, arthrodesis of the IP joint, and arthroscopic ganglionectomy of recurrent IP ganglion [1].

19.8.2 Positioning (Fig. 19.10)

Supine position with light manual traction [1, 11]. Some surgeons prefer to use thigh tourniquet [1]. Manual traction is used during visualization and instrumentation. The surgeon is seated at the lateral side of the operated foot with the monitor at the end of the bed [1].

19.8.3 Technique

- To confirm the proper placement of the portals, 18- or 21-gauge needle is used to mark the portal site and inject 2–3 mL of normal saline into the joints. Then the portals are established using "nick and spread" technique, that is, using 11-scalpel blade to incise the skin followed by using mosquito clamp to spread subcutaneous tissue and penetrate the capsule [1, 11]. Some reported using pump system to maintain the normal saline flow and achieve joint distraction [11].
- Instrumentation: 30° 1.9 mm, 2.7 mm arthroscope.
- Portals:
  - Portals for MTP joint:
    - Dorsal-medial and dorsal-lateral portals placed at or slightly distal to the MTP articular joint line, about 4–5 mm medial and lateral to the extensor digitorum lon-

gus tendon [1, 11]. When placing the dorsomedial portal over the second MTP joint care should be taken to avoid injury to the dorsal digital branch of the deep peroneal nerve. This nerve branch runs in the first intermetatarsal space very close to the medial border of the second MTP joint [11, 12]. At the level of other MTP joint, the dorsal digital branches of superficial peroneal nerve are at risk [1].

– Portals of proximal IP joint:
  - Dorsomedial and dorsolateral portal made at the dorsomedial and dorsolateral corners of the IP joint, between the collateral ligaments and the lateral slips of the tendon expansion. Insert the arthroscopic instrument along the dorsal recess and point away from the articular surface. The plantar lateral portal: located at the plantar lateral corner of the joint.

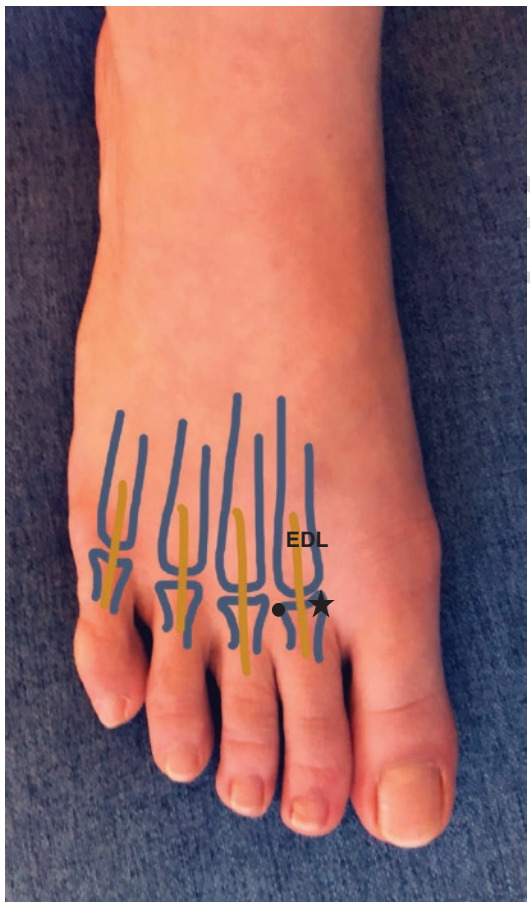

Fig. 19.10 MTP joint arthroscopy ★ indicates the dorsomedial portal, • indicates the dorsolateral portal. *EDL* extensor digitorum longus

19.8.4 Procedure

- Arthroscopic interposition arthroplasty of second MTP in Freiberg disease:
  – This is performed utilizing the dorsomedial and dorsolateral portal, alternating between the two portals as viewing and working portals. Diagnostic arthroscopy followed by debridement of the damaged cartilage, synovectomy, and removal of loose body is performed. A probe is used to measure the distance from one portal to the center of the defect in order to prepare the tendon graft. Harvesting the tendon of the extensor digitorum brevis follows this. The extensor digitorum brevis tendon is identified along the dorsolateral portal incision, which is the lateral to extensor digitorum longus tendon. Then hemostat is passed around the tendon and tension is applied by lifting the tendon. The tendon is traced proximally and through a small transverse incision the proximal end of the EDB tendon is transected. The tendon is then retrieved through the incision of the dorsolateral portal. The graft is rolled into a ball and sutured with long-stay suture using No. 0 Vicryl.
  – The graft is passed through the dorsolateral portal and the stay suture is then passed through the plantar plate while making sure that the suture and then the graft pass over the center of the chondral defect. The stay suture is passed all the way through the plantar skin. The exit point of the suture is dilated via hemostat and the sutures are tied and the tendon graft is stabilized. The insertion of the tendon can be sutured to the dorsal capsule of MTP joint for additional stability of the graft [1].
- Arthroscopic synovectomy:
  – It is indicated for pain and swelling control in metabolic, inflammatory arthritis, or

infections. It is advised to place the portals slightly further from the extensor tendons. To clean the medial gutter, the dorsolateral portal is used to visualize the joint and the dorsomedial portal used as instrumentation portal and vice versa for synovectomy of the lateral gutter [1].

- Arthroscopic-assisted double-plantar plate tenodesis for metatarsophalangeal instability:
  - Two portals, dorsomedial and dorsolateral, are used to perform this procedure. Any associated intra-articular pathology is treated accordingly.
    - The dorsomedial portal is used as the visualization portal and dorsolateral portal is used to pass the sutures. This is performed while holding the MTP joint in flexion. The first limb of the sutures is passed through the lateral part of the plantar plate-fibrous flexor tendon sheath complex and all the way through the plantar skin. A second proximal incision is made over the dorsal aspect of the shaft of metatarsal. Using a hemostat, a blunt dissection starting on medial side of the metatarsal shaft and extending to the plantar aspect of the distal metatarsal is performed. The suture is retrieved through this proximal incision. A second suture is passed through the medial part of the plantar plate and retrieved along the lateral side for the metatarsal shaft through the proximal incision. Then, while holding the ankle in neutral position, the sutures are secured to the extensor digitorum longus tendon at the proximal dorsal wound. These steps are repeated and two figure-of-eight configuration of sutures connecting plantar plate-flexor tendon sheath complex to extensor digitorum longus are constructed [1].

19.9 Excision of Tarsal Coalition

19.9.1 Indications

Resection of talocalcaneal (TC) coalition or resection of a calcaneonavicular (CN) coalition.

19.9.2 Positioning

In talocalcaneal coalition resection: Patient is positioned supine with hip in flexion, external rotation, and abduction and knee in flexion. Tourniquet is applied to the ipsilateral thigh [13–15]. If the resection is performed through posterior arthroscopy to hindfoot, patient will be placed prone.

In calcaneonavicular coalition resection: Patient is laid supine with bump under the ipsilateral hip in order to position the foot in internal rotation [16].

19.9.3 Technique

- TC coalition portal insertion:
  - Posterolateral portal: While ankle is in neutral position, this portal is placed just lateral to the Achilles tendon above imaginary line drawn from the tip of the lateral malleolus to the Achilles tendon. Some surgeons prefer injecting normal saline to subtalar joint prior to skin incision although this is not our routine practice. Longitudinal skin incision is performed followed by introducing a blunt trocar into the lateral aspect of the subtalar joint. Arthroscope is then inserted into the lateral recess of the subtalar joint. The first landmark to be identified is flexor hallucis longus (FHL). Move the great toe assist for identifying the tendon. Keeping the FHL in view helps preventing damage to the neurovascular bundle during the procedure [14].
  - Posteromedial portal: Some authors perform this portal by first inserting 18-gauge needle into the joint. The needle is visualized with the arthroscope to ensure its lateral position to the FHL. Then skin incision is performed followed by blunt dissection using mosquito clamp that is introduced into the lateral aspect of the subtalar joint [14].
- CN coalition portal insertion:
  - Location of the portals is identified under fluoroscopy (see portal description for location of each portal).
  - After skin incision, subcutaneous dissection with a mosquito clamp is performed in

the bone. Under fluoroscopic control, the mosquito is passed around the coalition at its upper and lesser aspects to create a working area [15].

19.9.4 Portals

- TC coalition resection:
  - Viewing portal: two fingerbreadths posterior to the vertex of the medial malleolus.
  - Working portal is created approximately three fingerbreadths inferior to the vertex of the medial malleolus [17].
  - Others have described posterolateral and posteromedial portals:
    - Posterolateral portal: a line is drawn from the tip of the lateral malleolus to the Achilles tendon, parallel to the foot sole. The portal is placed above this line, tangential to the Achilles tendon [13, 14, 18].
    - Posteromedial portal: placed at the same level of posterolateral portal, medial and tangential to the Achilles tendon [13, 14, 18].
- CN coalition:
  - Visualization portal: Under fluoroscopy an oblique view of the foot is taken and the portal is established dorsal to the angle of Gissane. Another fluoroscopy image is taken to conform the portal location over the dorsal aspect of the anterior process of the calcaneus at the proximal and lateral extremity of the coalition [14–16].
  - Working portal: located on the distal and medial aspect to the coalition at the junction of the coalition with the navicular bone. This portal is placed in the space between the talonavicular and calcaneocuboid joints. Fluoroscopy is used to localize the portal site. This portal is less than 1 cm dorsal and medial to the superficial peroneal nerve [14–16].
  - Accessory visualization portal: It is established to visualize the deep part of the anteromedial calcaneal process. It is preformed under fluoroscopy guidance at the medial side of the extensor hallucis tendons at the level of the talonavicular joint [14].
  - Alternative portal: The visualization portal can be placed 0.5 cm anterior to the anterolateral corner of the calcaneus and the working portal at the medial border of the extensor tendons [14].

19.9.5 Procedure

- TC coalition resection:
  - Hayashi described the resection through posteromedial approach. The viewing and working portals are performed (see description in portals). The procedure starts by separating the coalition from surrounding soft tissue using cobb. High perfusion pressure should be maintained to prevent soft tissue from blocking the view. Then using a shaver, continue removing any soft tissue attached to the coalition, as this will facilitate the resection of the coalition. Care should be taken to keep the instruments facing towards the coalition as neurovascular bundle is on the side opposite to the coalition. The resection of the coalition is continued until normal articular surface can be confirmed. Fluoroscopy can be used to confirm the location of the coalition. Bone wax is applied to the resection surface once procedure is completed [17].
  - Others have described the resection utilizing posterolateral and posteromedial portals. Once the two portals are established, synovectomy of the subtalar and ankle joint is performed to achieve optimal visualization of the coalition. Then FHL tendon is identified. The subtalar joint line and FHL tendon are followed until localizing the TC coalition. For most of the time the working portal will be the posterolateral portal and the instruments will be passed through the posteromedial portal. In cases of osseous coalitions, visualization of the subtalar joint might be difficult. The posterior talofibular ligament (PTFL) is used as a landmark to localize the subtalar joint that is located approximately 5 mm below the talar insertion of the PTFL. Arthroscopic burr or shaver is used to resect the coalition until normal articular surface is encountered and

always keep the FHL medial to the instruments. Resection is complete when you can visualize healthy cartilage posteriorly, medially, and laterally, and good subtalar motion can be elicited clinically. A probe can be inserted between talus and calcaneus, and a gentle levering is performed to verify the opening of the joint. Fluoroscopy image is taken to confirm adequate resection [13, 14].

- CN coalition:
  - Two portals are placed under fluoroscopy guidance as described above. After passing the mosquito clamp around the coalition, a shaver is introduced to clear up the soft tissue around the coalition. Minimal release of the extensor digitorum brevis muscle should be done just enough to get access to the coalition [16]. Then tissue debridement is performed laterally up to the anterior process of the calcaneus and the calcaneocuboid joint and medially up to the talonavicular joint. Once complete visualization of the coalition is achieved, resection of the bony bar is performed using a burr. The landmarks to follow are:
    - The anterior and dorsal aspect of the calcaneus and the calcaneocuboid joint laterally
    - Talar head, the talonavicular joint, and the lateral part of the navicular bone medially
  - Using the arthroscope, visualize the inferior side at the talonavicular joint and calcaneocuboid joint to assess if the resection is complete. Once resection is completed, the foot is taken into inversion-eversion motion to confirm the mobility between navicular bone and the calcaneus [14–16].

19.10 Arthroscopy of Total Ankle Arthroplasty for Impingement, Cysts and Aseptic Loosening

19.10.1 Indications

Bony impingement at medial or lateral gutters minimum of 90 days, preferably 6 months post-total ankle arthroplasty [19–21].

19.10.2 Positioning

Patient is positioned supine and thigh tourniquet is applied. No distraction is used [19–21].

19.10.3 Technique

18-guage needle can be introduced to confirm the joint level and then 11-guage scalpel blade is used to perform a skin incision. This is followed by using hemostat to penetrate the capsule. Hemostat can be used to break some of the scar tissue before introducing the trocar.

- Portals: standard anteromedial and anterolateral ankle arthroscopy portals [19–21].

19.10.4 Procedure

- Lateral gutter debridement:
  - In this technique the anteromedial portal is the visualization portal while the anterolateral portal is used as working portal. Start with clearing the scar tissue anterior on the lateral gutter. This can be achieved using a combination of shaver, arthroscopic scissors, and biters and right angled curette as necessary. This gives access to visualize the lateral gutter. Soft tissue in the gutter is debrided to clear the space between lateral malleolus and lateral edge of the talus. A burr is then used to remove any bony impingement starting laterally on the fibula. After cleaning the lateral gutter arthroscope is taken over the top of the talus to look down into the gutter. Any talar shelf bone if present should be cleaned. It is believed that visualization of the peroneal tendons is essential to conform adequate debridement. Care should be taken to avoid excessive bony resection on the talus or any damage to the metal and polyethylene of the TAA implant. This can be achieved by having the shaver in contact with the prosthesis while debridement of the joint is performed [19–21].

- Medial gutter debridement:
  - Arthroscope is introduced through the anterolateral portal and instruments are introduced through the anteromedial portal. Similar to lateral gutter debridement the combination of shaver, burr, and curette is utilized. Start by cleaning soft tissue anterior to medial gutter to be able to visualize the gutter. Then any soft tissue in the gutter is removed until the medial malleolus and medial aspect of the talus are seen. A burr is used to resect any bony prominence on medial malleolus or talus. Some authors believe that a complete debridement requires that the posterior tibial tendon be visualized in the medial gutter [19–21].

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Ankle Alignment Procedures

20

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20.1 Introduction

Articular tibiotalar fractures are burdened with many complications that may be distinguished as early and late.

Whatever is the type of fracture and related complications, the hazard of osteoarthritic evolution is a common issue with prevalence till 1% of the population [1].

In contrast to the osteoarthritis of other joints, such as hip and knee, ankle osteoarthritis is, in most cases, secondary to trauma outcomes [2, 3]. As a consequence, keeping in mind the mean age of distribution of high-energy trauma, ankle arthrosis becomes symptomatic roughly 12 years earlier than compared to other anatomic districts [4].

Since the pathological anatomy is extremely variable, the treatment of the tibiotalar fractures is often complex resulting in poor outcomes. Despite the effort, even if different authors have proposed numerous treatment algorithms, today there is still an open debate about the guidelines and ideal treatments of these conditions. In 2007 Giannini described specific indications for the treatment of tibiotalar posttraumatic outcomes based on the osteoarthritis degree, age, and articular condition, as shown in Table 20.1.

Malunion is often a complication of ankle joint fractures. This complication is caused by three factors: type of fracture, incomplete reduction, and loss of reduction. Other minor factors which may contribute to establish a malunion are gender, osteoporosis, and age. When a malunion occurs in the ankle joint there is a lack of articular matching and an alteration of mechanical axis. This happens due to rotational or angular deformities which in turn affect weight-bearing distribution playing a role in early and progressive joint deterioration.

In order to detect deformities and select the proper treatment for the ankle joint it is crucial to consider some radiographic criteria.

The anatomic axis of tibia can be found by tracing a line starting from the indentation between the two tibial spines proximally and the center of the conjugation cartilage on the tibial plafond distally. Another important parameter to evaluate tibial deformities is the TAS (tibial-ankle surface); this angle is traced by a line passing through tibia's articular surface which crosses the mechanical axis of the tibia at the plafond center forming, frontally, the angle known as TAS (tibial-ankle surface) (Fig. 20.1). This angle is open medially and normally ranges between 91° and 93° in the Caucasian population. This value describes the slight and physiologic valgus

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Table 20.1 Guidelines according to Giannini

Stage	Age	Ankle joint condition		Surgical procedure
2	–	Preserved ankle anatomy		Arthrodiastasis and arthroscopic debridement
		Supra-articular malalignment		Supra-malleolar osteotomy
		Intra-articular malalignment		Joint reconstruction
3	< 50	Preserved or restored ankle anatomy	<25° of motion in other foot joints or arthrodesis refusal	Joint allograft
				Joint prosthesis
	>50		>25° of motion in other foot's joints	Arthrodesis
	–			
	–	Nonrestorable ankle anatomy, chronic infections, neurological disorders, severe osteoporosis		Arthrodesis

of the ankle. On the sagittal plane these lines form the angle known as TLS (tibial lateral surface) (Fig. 20.1), and open anteriorly, and that normally ranges between 81° and 82°. While evaluating a deformity is essential to consider another parameter known in literature as CORA (center of rotation and angulation) (Fig. 20.2), this is defined as the intersection point between the proximal and distal mechanical axis of tibia; the angle between these represents the deformity amount to correct.

Ankle deformity and malunion are often difficult to detect; some authors proposed a classification of hidden types [5] (Fig. 20.3). In unclear cases it may be helpful to perform CT scan and MRI in order to clarify the exact anatomy of the deformity.

20.2 Classification of Malunions

It's possible to distinguish malunions in two categories based on age: adult malunions and growing-age malunions [6].

Adult's malunions are divided into more subcategories based on fracture patterns:

- Supra-malleolar
- Malleolar
- Intra-articular distal tibia
- Talar

Their incidence is variable in a range between 5 and 68% according to literature [6–8]. The alignment failure secondary to one of these fractures is followed by chronic pain and functional alteration until the development of tibiotalar osteoarthritis. This event sequence results from the altered weight bearing which, as demonstrated in many previous studies [8, 9], changes the location and shape of the contact areas between the articular surfaces of tibia and talus, leading to deterioration of articular cartilage and finally premature osteoarthritis.

20.3 Treatment of Malunion in Adults

There are many procedures when it comes to malunion surgical treatment, but in literature there are mainly two trends: articular salvage surgery and articular replacement or blockage surgery [6, 10–17].

The rationale of the articular sparing surgery is to interrupt or slow down the osteoarthritic progression by realigning every existing deformity by reestablishing the correct articular anatomy as much as it's possible. The second approach finds application whenever the articular degeneration is too advanced to such a degree which makes impossible any kind of repair efforts, shifting priorities to pain treatment mainly and whenever is possible to maintain a certain functionality.

Articular salvage surgery may be divided into extra-articular and intra-articular, based on malunion's kind [6]. As for any other kind of surgery it is essential to set the right indications and contraindications in order to guarantee the best possible results (Table 20.2). For these reasons, nowadays, the matter is still a subject of debate [19, 20].

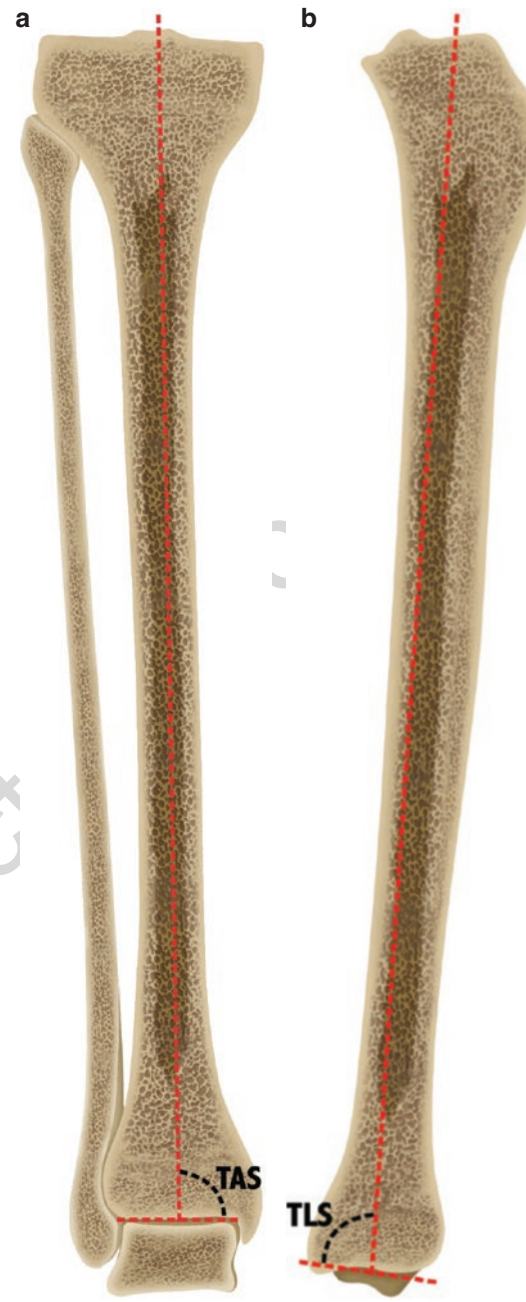

Fig. 20.1 (**a**) Coronal projection of tibial axis, visualization of TAS angle. (**b**) Sagittal projection of tibial axis, visualization of TLS angle

Extra-articular salvage surgery is applied to malunions classified as supra-malleolar and talar. Intra--articular salvage surgery, on the other hand, is applied in malleolar, articular distal tibial fractures, and talar body malunions. The aim of this surgical procedure is to restore the proper articular matching [21].

Malunions may be often associated to soft tissue or articular cartilage lesions: most frequently there are osteochondral lesions and ligamentous laxity of one or more ankle's compartments. An osteochondral lesion is found more often at the medial surface of the talus dome (up to 80% of reported cases). Diagnosis in such cases is suspected when patients

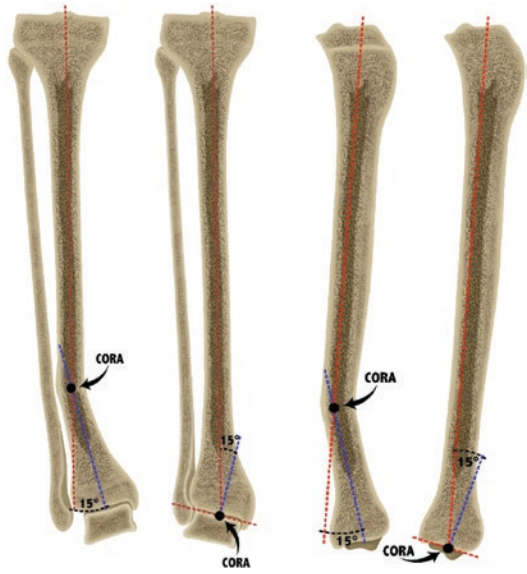

Fig. 20.2 Different localization of CORA angle represents different orientations of the deformity, therefore affecting the treatment choice

refer to symptoms such as pain, swelling, and occasional joint blockage with a positive history of ankle trauma. The first choice for an instrumental examination is the standard weight-bearing X-rays in order to visualize the malleolar clamp; often it is necessary to undertake more detailed examinations such as CT scan or MRI scans. Lesion size is crucial to prognosis; that's why over the years numerous classifications were developed [22, 23]. Ankle ligamentous laxities, when they occur along with osteochondral lesions, are needed to be treated together with malunion.

20.4 Extra-Articular Surgery

20.4.1 Supra-Malleolar Osteotomies

Speed and Boyd in the 1930s suggested for the first time in orthopedic surgery a procedure to

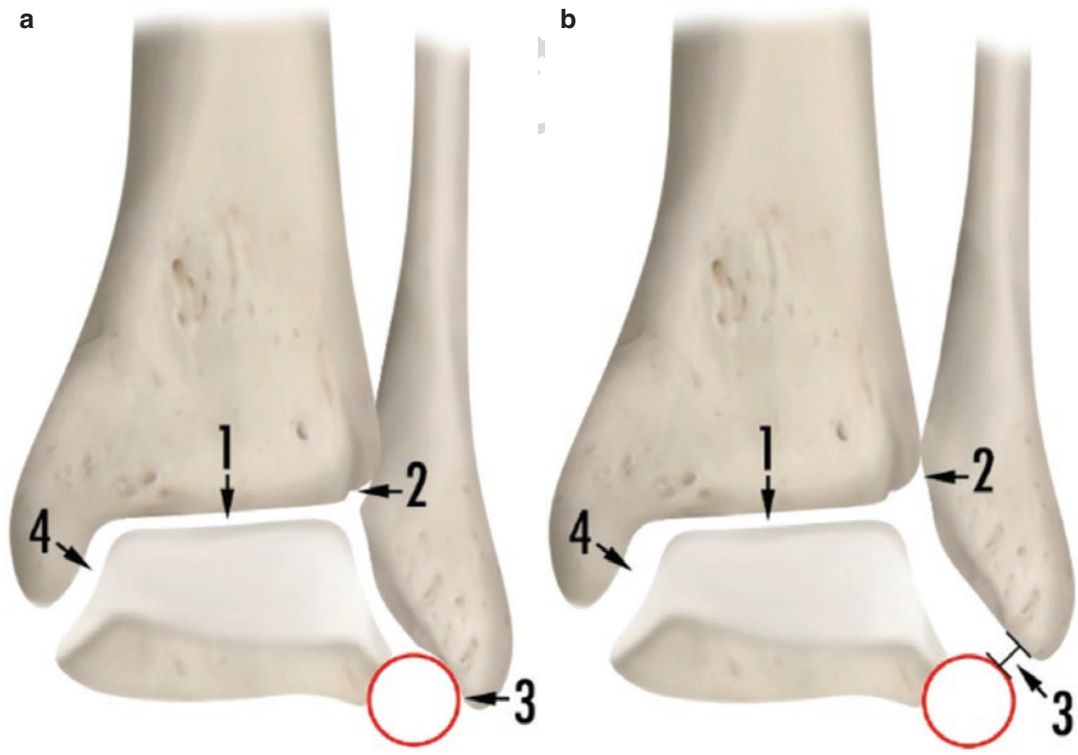

Fig. 20.3 Hidden nonunion according to Yablon. Figure (**a**) represents a normal ankle joint whereas figure (**b**) represents a hidden nonunion. In particular it should be noted: (*1*) Lack of symmetry in joint line. (*2*) Interruption in Shenton line formed by the outline of tibial plafond and the outline of fibula's medial side. (*3*) The fibula's apex doesn't lie in a circle alongside talus lateral joint side. (*4*) Widening of joint line medially

Table 20.2 Tibiotalar reconstruction guidelines according to Mulhern [18]

Indications	Contraindications
• Asymmetric ankle osteoarthritis with varus/valgus deformity and >50% preserved tibiotalar joint surface • Isolated medial/lateral osteochondral lesion of the tibiotalar joint • Physeal growth arrest • Tibial torsion • Tibial fracture malunion • Realignment before total ankle arthroplasty • Tibiotalar arthrodesis malunion • Deformities caused by neurologic/muscular conditions • Congenital talipes equinovarus sequelae • Rheumatoid ankle • Hemophilic arthropathy	**Absolute**: • End-stage ankle osteoarthritis with <50% preserved tibiotalar joint surface • Unmanageable hindfoot instability • Acute/chronic infection • Severe vascular/neurologic deficiency • Neuropathic disorders **Relative**: • Patient noncompliance • Age >70 years • Impaired bone quality of the distal tibia or talus • Tobacco use • Insulin-dependent diabetes mellitus • Chronic skin abnormalities or soft-tissue defects

correct a posttraumatic deformity [24]. Same concept was adopted in the 1960s by Russian authors, which led to several publications [25, 26]. However the first study to systematically report results of patients that underwent a supra-malleolar osteotomy was published in the mid-1990s by Takakura [27]. In this study Takakura reports midterm results of supra-malleolar osteotomies performed on 18 patients affected by a primary form of ankle arthritis with a varus deformity of stages 2 and 3 according to Takakura's own classification (Table 20.3). He reported excellent results in six patients, good results in nine, and sufficient results in three. A later evaluation performed by Takakura himself involved the same procedure applied for the treatment of posttraumatic deformities in nine patients; the procedure showed excellent results in four cases, good results in two, and sufficient results in three cases [28]. Thanks to Takakura's relevant publications over the following decades many international studies were published involving the use of corrective osteotomies in deformity treatment, developing new treatment algorithms and rehabilitative protocols.

20.4.1.1 Preoperative Planning

In the surgical planning of the correction of a deformity it is crucial to correctly identify the deformity site [18]. It's necessary to identify the rotational center and the deformity angle (CORA). Besides the CORA, from a radiographic point of view, it is mandatory to obtain panoramic weight-bearing X-rays of lower limbs in order to identify any other deformity, which may lead to poor results of the surgical treatment. Moreover, the medial and lateral compartments need to be examined to evaluate if it is necessary to reconstruct or release ligaments. An evaluation of the foot dorsiflexion is mandatory to eventually perform an Achilles tendon lengthening. Subtalar joint mobility has to be evaluated to estimate the potential compensation capability after the planned surgery. The last parameter to evaluate is lower limb length to anticipate any possible discrepancy in extension which may influence the kind of osteotomy to perform.

The possible surgeries are the following:

- **In plus osteotomies**: According to the type of deformity they may be performed on the medial or lateral surface of the tibia. It's possible to maintain or minimally increase the length of operated limb. Normally it is applied on a tricortical autologous bone graft collected from same-side iliac crest or instead an allograft can be used. These osteotomies are contraindicated in the presence of previous extended surgical scars, infections, poor tissue regeneration potential, and vascular deficit. Based on the osteotomy angle it is possible to perform multiplanar correction. When compared to osteotomies in minus they seem to require longer to consolidate, even if some studies deny this result [27].
- **In minus osteotomies**: As before, they may be performed on the medial or lateral surface of tibia. The main disadvantage is the shortening of the operated limb, which has to be considered during preoperative planning.

t3.1 **Table 20.3** Takakura's classification [1]

Stage	Radiographic findings
1	No joint-space narrowing, but early sclerosis and osteophyte formation
2	Narrowing of the joint space
3a	Obliteration of the joint space limited to the medial malleolus facet with subchondral bone contact
3b	Obliteration of the joint space advanced to the roof of the talar dome with subchondral contact
4	Obliteration of the joint space with complete bone contact

The absence of grafts is the main advantage of this procedure, besides shorter consolidation time as reported in literature [29, 30].

- **Dome-shaped osteotomies**: Technically the more challenging to perform, they may represent the best choice in some specific cases. Specific guidelines to perform this technique are not yet universally codified. According to Krähenbühl [31] deformities over 10° angle represent the optimal indication; on the other hand Knupp [1] reserves this procedure for cases with preserved joint congruency. The main disadvantage of this procedure is represented by the mono-planar correction, since multi-planar corrections are not possible.
- **Peroneal osteotomies**: Peroneal shortening or lengthening osteotomies find indication in nonunions associated to fractures in external rotation which lead to fibula's rotational shortening. Correcting the fibula's deformity allows to restore a normal tibiofibular syndesmosis.

Barg and Mangone proposed in their publications a simple formula to calculate the degree of correction secondary to both in plus and in minus osteotomies [32, 33] (Fig. 20.4).

In the eq. H stands for the height of wedge to implant or remove, α stands for the amount of deformity and potential degrees of hypercorrection, and Ø represents tibia's diameter at the osteotomy site.

20.4.1.2 Surgical Procedure

If osteotomy's purpose is to correct a deformity it should be performed at CORA's level. If the deformity (and therefore the CORA) is found to be at the joint surface or there is a hidden malunion the osteotomy should be performed approximately 4–5 cm proximally to distal malleolus extremity [29]. An osteotomy performed somewhere else from CORA's level leads to an inevitable misalignment between axis of proximal and distal segment (Fig. 20.5).

In plus or in minus wedge osteotomies (Fig. 20.6): Osteotomy site should be identified avoiding excessive periosteum removal. A Kirschner wire is positioned at the level of the osteotomy site parallel to joint surface to be used as guide. While performing the bone saw cut water irrigation is crucial in order to avoid thermic bone damage. Osteotomy is completed with an osteotome to prevent soft-tissue damage. The periosteum on the opposite side of the osteotomy should be preserved as it's supposed to work as a keystone in obtaining correction.

Dome-shaped osteotomy (Fig. 20.6): The shape of the osteotomy can be created with various techniques. Generally, a single pin is inserted parallel to joint line with the aim to work as osteotomy's rotational center. Bicortical holes are then performed as guide to the following osteotomy with micro-oscillating saw. The distal fragment is then mobilized to correct the deformity.

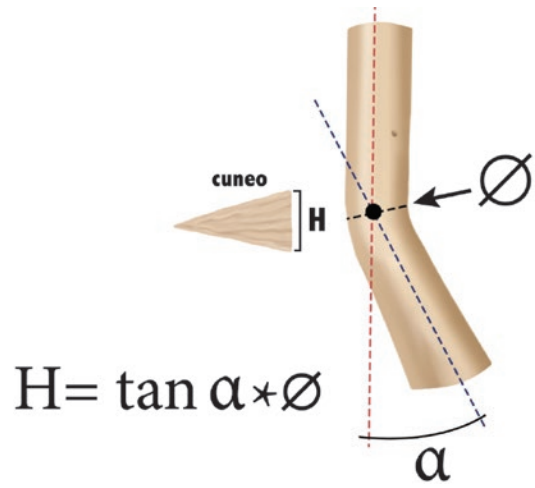

Fig. 20.4 Equation to establish the wedge size to implant or remove in order to gain correction of a deformity. To be noted: α absolute value is represented by the CORA and Ø represents the diameter of the tibia at the level of the CORA

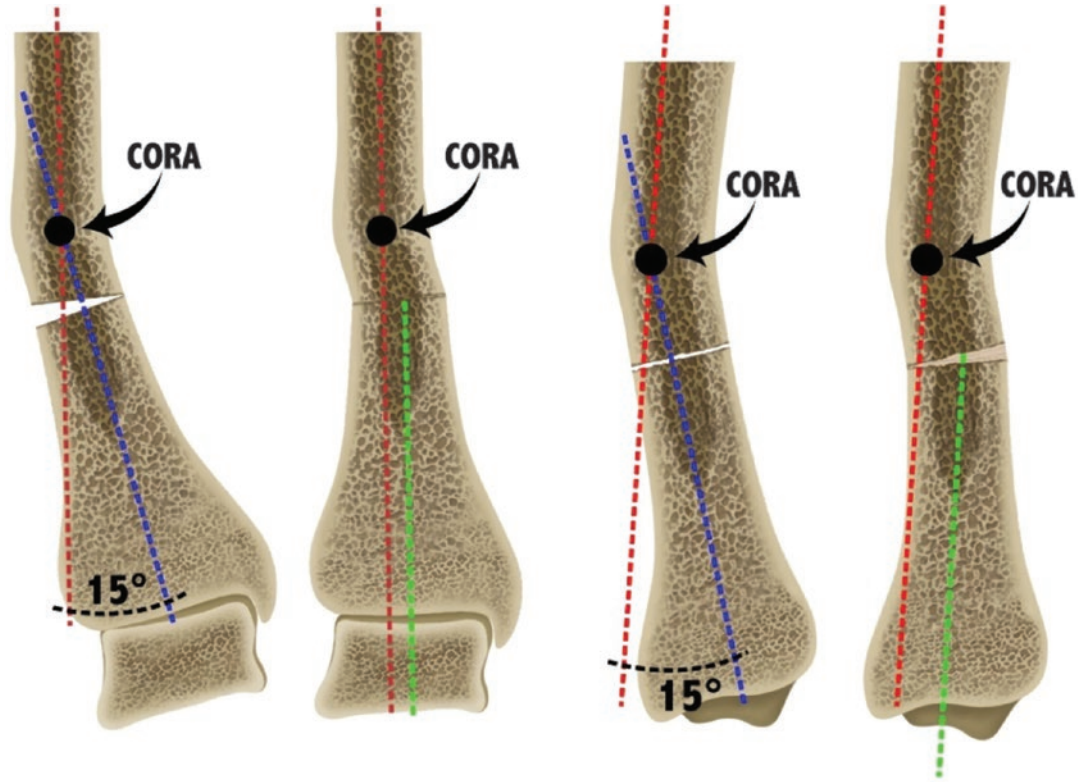

Fig. 20.5 An osteotomy performed away from CORA's level leads to misalignment between the axis of distal and proximal tibia

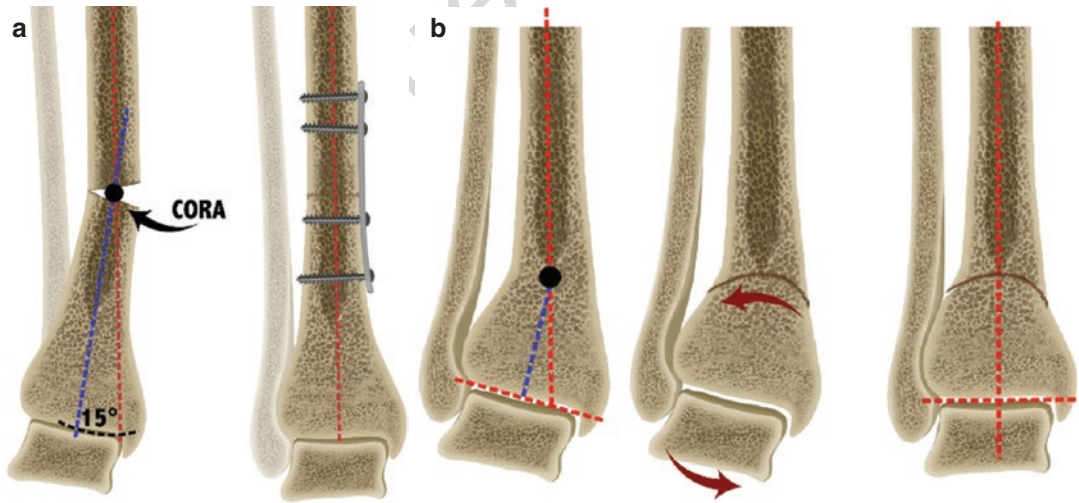

Fig. 20.6 (**a**) Minus osteotomy to correct varus malalignment performed at CORA's level. (**b**) Dome-shaped osteotomy to correct valgus deformity performed at CORA's level

Peroneal osteotomy (Figs. 20.7, 20.8, 20.9, and 20.10): In order to ensure a normal anatomy and appropriate functionality it is often necessary to perform a peroneal osteotomy in plus or in minus. It is mandatory to perform it at the same level of the tibial osteotomy.

20.4.1.3 Postsurgical Treatment

The first postsurgical treatment consists of a boot cast and non-weight bearing. The length of this phase is still debated. Stamatis and Myerson describe a protocol of boot cast excluding weight bearing for 10–14 weeks until obtaining radiographic evidence of bone healing [29]. On the other hand, Mulhern et al. suggest a shorter period (6–8 weeks) with progressive weight bearing in the following month; according to their protocol full functional recovery is fulfilled in 6–12 months [18].

20.5 Intra-articular Surgery

20.5.1 Reconstructive Treatment of Malleolar Malunions

The reconstructive treatments of malleolar malunions are usually performed through fibular reconstructions in order to influence tibiofibular syndesmosis; this procedure is often associated with a medial ligamentous reconstruction. It is mandatory to correct any eventual malunions of posterior malleolus or any embeds in medial tibia's plafond [34].

20.5.2 Reconstructive Treatment in Outcomes of Distal Articular Tibial Fractures

Complex **distal articular tibial fractures** are often associated with chondral and soft-tissue lesions [35]. Most of these fractures are caused by high-energy trauma with axial load, which damages articular cartilage, and, during a period of years, leads to posttraumatic osteoarthritis. Resulting deformities may be treated through articular salvage techniques such as those from Rammelt [35] (Table 20.4).

Surgical procedure (Fig. 20.11)

Distal articular tibial fracture malunions may be approached through anteromedial, anterior, or anterolateral approach. To reach the tibial plafond it may be necessary to perform a medial malleolar osteotomy.

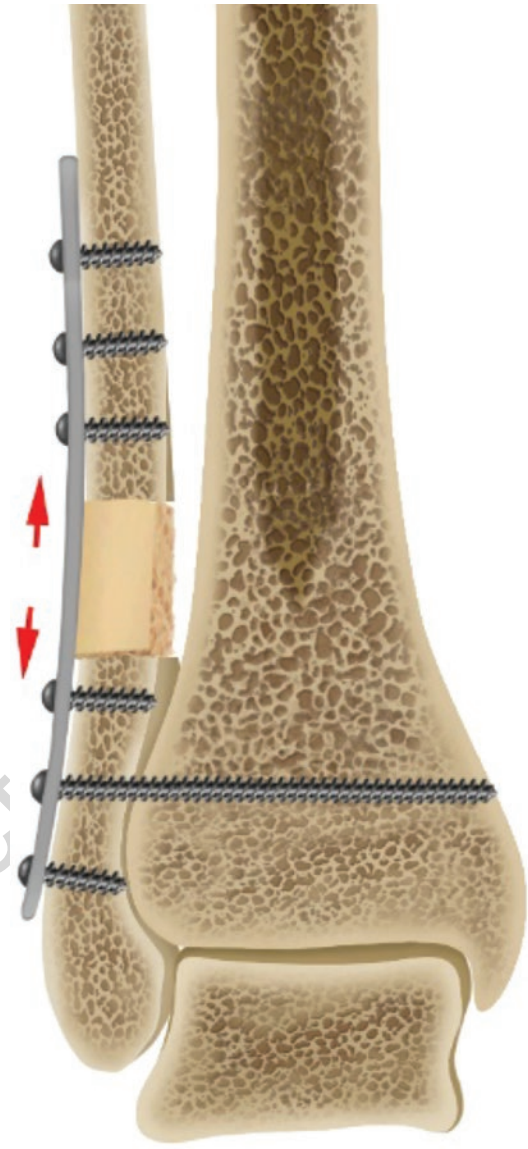

Fig. 20.7 In plus fibular osteotomy to correct length deficit

- *Anterior approach*: A classic dorsal incision is performed followed by capsulotomy and exposition of the tibial plafond and talar dome. The anteromedial and anterolateral

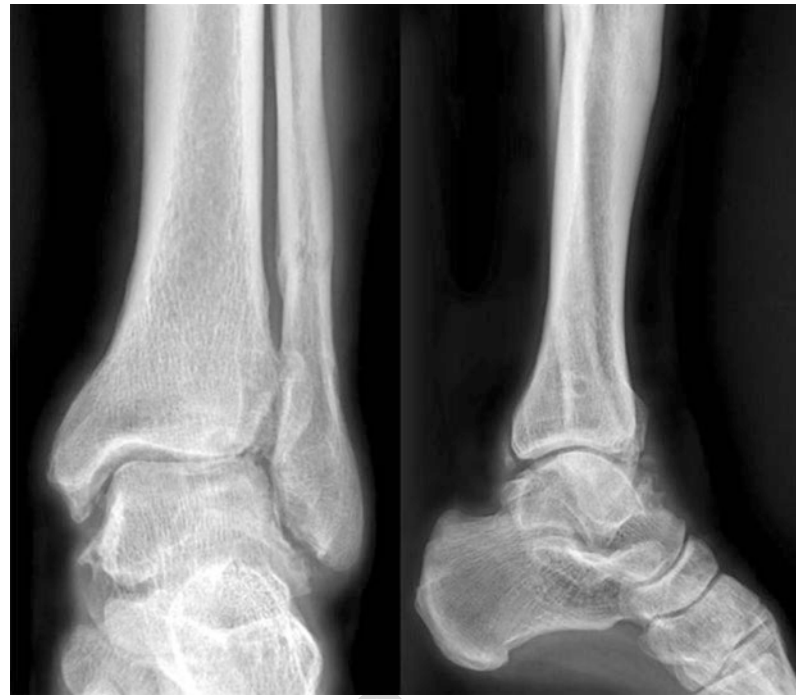

Fig. 20.8 Deformity outcomes in fibular fracture with tibial hyperpressure at the medial talus dome

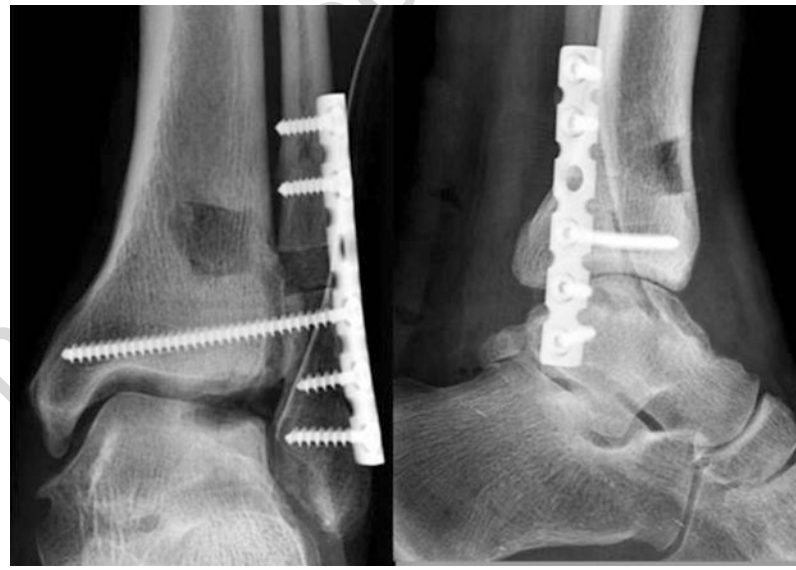

Fig. 20.9 Postsurgical X-ray of fibular lengthening through graft from tibia's bulb and repair of chondral defect, along with cleanup, by placing a scaffold with autologous medullar mononuclear cell concentrate

fracture fragments need to be separated in order to gain access to central or posterior malunion. Unstable intra-articular fragments need to be removed. Minor cartilage deteriorations are usually treated with microfracture procedures. In case of nonunion, debridement of sclerotic/necrotic tissue is performed in order to create a microenvironment suitable for bone healing. In case of fibular malunions a fibular osteotomy is usually performed in order to get free access to and mobilization of distal fragments.

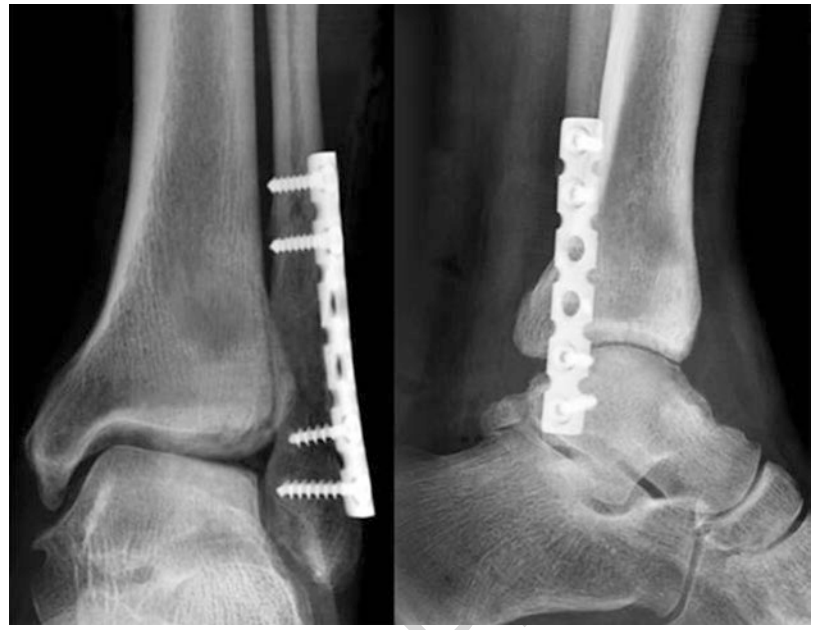

Fig. 20.10 Postsurgical X-ray 18 months later

t4.1 **Table 20.4** Guidelines according to Rammelt

t4.2 Indications	Contraindications
t4.3 Young, active t4.4 patients	Severe osteoarthritis in weight-bearing areas
t4.5 Good bone quality	Impaired bone quality
t4.6 Adequate cartilage t4.7 coverage	Chronic soft-tissue or bone infection
t4.8 Good patient t4.9 compliance	Scarce patient compliance
t4.10	Comorbidities

343 Eventual bone loss is treated with bone
344 grafts. The stabilization of the osteotomy
345 site is obtained via plates and screws.
346 • *Medial malleolar osteotomy*: An important
347 shortening of medial malleolus requires a ded-
348 icated osteotomy in order to restore normal
349 malleolus's clamp anatomy.

350 *Posterior approach*: Isolated malunions of the
351 posterior joint line may be treated through a pos-
352 teromedial or posterolateral approach. Hallux
353 long flexor muscle is retracted medially to protect
354 the vascular-nervous bundle. Any intra-articular
355 fragment needs to be removed. Then, a corrective
356 osteotomy is performed. The stabilization of
357 fragments is achieved via plates and screws.

20.6 Malunions During Growth 358

359 Besides an articular mismatch and evident defor-
360 mities, the outcomes of these malunions are dif-
361 ficult to predict, due to patient's skeletal age. It
362 may cause an early sealing of epiphysis and bone
363 rod formation.

364 These synostoses are classified according to
365 Ogden [36] in:

366 • Peripheral: predisposing to angular deformities
367 • Linear: extending from front to rear in the sag-
368 ittal plane
369 • Central: in the plafond's midportion, causing a
370 deformity of the joint line

371 In case of bone rod the excision surgery has to be
372 performed when there's a 2 years of growth expec-
373 tancy and less than 50% of tibia's plafond involved
374 [37]. Angular deformities associated to rod have
375 been described as liable of spontaneous correction

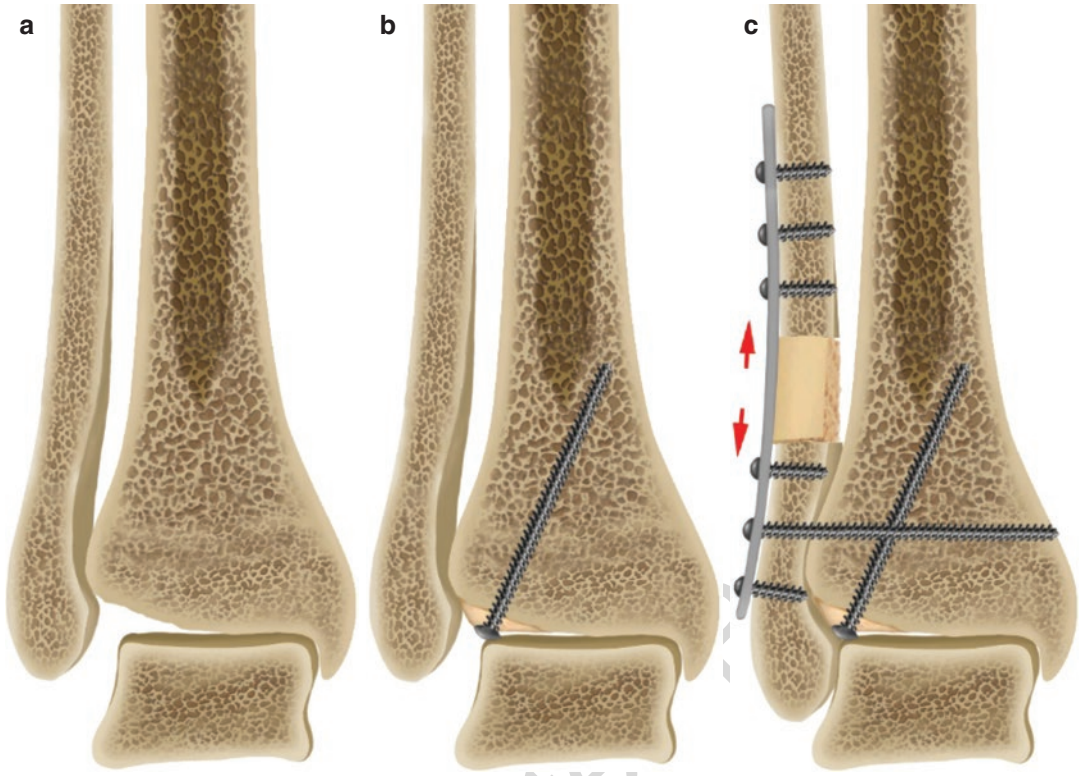

Fig. 20.11 Treatment of an intra-articular malunion through lateral plafond-plasty associated to a peroneal lengthening osteotomy. (**a**) Initial deformity. (**b**) Plafond-plastic with screw and bone graft. (**c**) Peroneal lengthening osteotomy

if <15° in patients under 10 years or <10° in children older than 10 years. If the deformity exceeds these values a corrective osteotomy is required.

20.7 Conclusions

In tibiotalar fracture among many outcome scenarios malunions are among the most frequent and feared complications. The treatment rationale of the described techniques is to preserve the joint and to block or, at least, slow down the osteoarthritic progression through a realignment of the loading axis. In literature there are reports of good long-distance results in a percentage between 70% and 90% of patients [30, 38–41]. The described procedures offer also benefits even in case of successive progression of the osteoarthritis allowing, in virtue of obtained articular alignment, a better performance in potential replacement or articular blockade surgeries.

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21 Current Concepts in the Treatment of Osteoarthritis of the Ankle

Yasuhito Tanaka

21.1 Introduction

Osteoarthritis (OA) occurs in the ankle less frequently than in the knee or hip, which are also weight-bearing joints, comprising about 4% of all OA [1]. The level of subjective impairment in end-stage OA of the ankle is equivalent to that of end-stage nephropathy or congestive heart failure, and it causes a comparable level of impairment of hip OA [2, 3]. Posttraumatic OA is a common form of ankle OA in young people that can lead to long-term, potentially lifetime impairment [3, 4]. Therefore, the role of effective therapies is significant.

Post-fracture OA, most commonly from malleolar and pilon fractures [5], makes up a large proportion of ankle OA cases [6]. Other causes include joint instability and abnormal ankle morphology, such as varus deformity of the articular surface of the distal tibia. Treatment must take the cause into consideration, as well as the stage of the disease, especially in varus-type OA of the ankle (Fig. 21.1) [7, 8]. This chapter presents a general outline of treatment strategies.

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21.1.1 Conservative Therapy

The chondrocytes of the ankle differ from those of the knee in that they are more resistant to degeneration and better able to repair themselves after being damaged [9, 10]. Therefore, the effects of the appropriate conservative therapy can be significant. However, while sufficient evidence regarding conservative therapy for the more common OA of the knee has been accumulated, few reports have discussed the effects of conservative therapy for OA of the ankle.

21.1.1.1 Lifestyle Guidance

While no studies have demonstrated the efficacy of dieting for OA of the ankle, it is considered appropriate to recommend that patients lose weight. Patients can also be advised to avoid movements that cause pain in their daily lives or to reduce the weight-bearing load on the ankle by using handrails or canes.

21.1.1.2 Pharmacotherapy

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the standard internal analgesic, and there are several skin patches and liniments that contain NSAIDs. The subcutaneous tissue of the ankle is thin, which makes it easier for external medications to exert their effects.

Intra-articular steroid injections are effective during periods of severe inflammation accompanied by intra-articular edema. Hyaluronic acid

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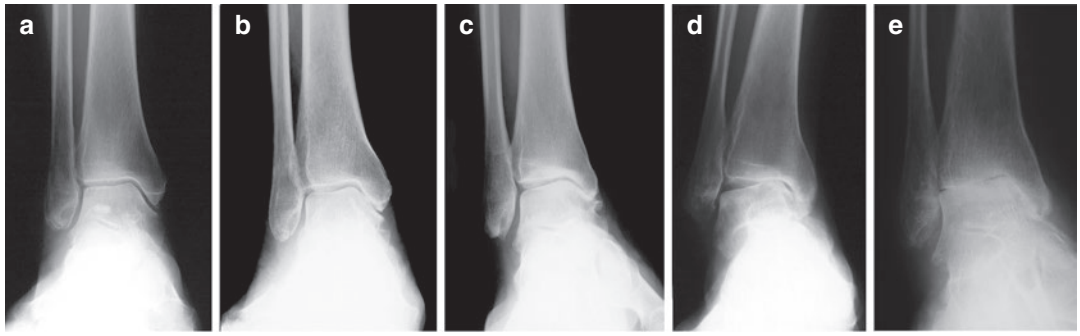

Fig. 21.1 Takakura-Tanaka classification of varus-type ankle osteoarthritis. (**a**) Stage I: no narrowing of the joint space, but early sclerosis and formation of osteophytes. (**b**) Stage II: narrowing of the medial joint space. (**c**) Stage IIIa: obliteration of the joint space with subchondral bone contact was limited to the medial malleolus. (**d**) Stage IIIb: obliteration extended to the roof of the dome of the talus. (**e**) Stage IV: obliteration of the whole joint space with complete bone contact

injections are also commonly used. The efficacy of hyaluronic acid, including its anti-inflammatory effects, lubricating action, ability to supply nutrients to the cartilage, and ability to improve pain thresholds, has been demonstrated by many studies [11–13].

21.1.1.3 Exercise Therapy

Posttraumatic ankle OA frequently involves limited range of motion (ROM) in adjacent joints as well as the ankle. Pain can arise from improper weight distribution on the foot. Therefore, active ROM training is important, and care should be taken to stretch the tendons around the ankle. Training of the peroneal muscle is particularly important in OA following injury to the lateral ligament. Standing on the toes so that the head of the first metatarsal presses against the ground can strengthen the peroneal muscle.

21.1.1.4 Physical Therapy

Heat therapy is performed as in cases of regular OA. Hot packs and underwater jet massage are used to warm the surface layers, while ultra-microwave, microwave, and ultrasonic therapies can be used to heat deeper areas. Regular use of a thermal insulation layer is also beneficial.

21.1.1.5 Orthotic Therapy

Ankle instability sometimes causes OA of the ankle. Joint incongruity and instability can markedly increase stress inside the joint [14, 15]. Braces that have been developed for the lateral ankle ligament injuries are also effective for ankle OA. If ankle OA is caused by a foot deformity, prosthetic shoes may improve load disparities inside the ankle.

21.1.1.6 Shoe Inserts

Custom-made shoe inserts have shown effectiveness in varus and valgus-type OA of the ankle. In varus OA, an insert is used to wedge the anterior and lateral sides, which disperses the weight concentrated on the anterior and medial ankle (Figs. 21.2 and 21.3) [16, 17]. Valgus OA is treated with an insert with medial arch support.

Treating varus-type ankle OA using a shoe insert with a lateral wedge is effective up to stage IIIa, in which obliteration of the joint space stops at the medial malleolus, but not in advanced cases of stage IIIb or higher. Up to stage IIIa, valgus of the subtalar joint compensates for varus of the articular surface of the distal tibia; however, this compensatory function is known to break down when stage IIIb is reached [18]. Treating stage IIIb cases with a lateral wedge is ineffective because it pushes the heel from the lateral side, which causes increase of the varus deformity.

21.1.2 Surgery

If conservative therapy is unsuccessful, surgery can be considered. Owing to the variety of potential surgical approaches, accurate diagnosis of the disease stage is critical.

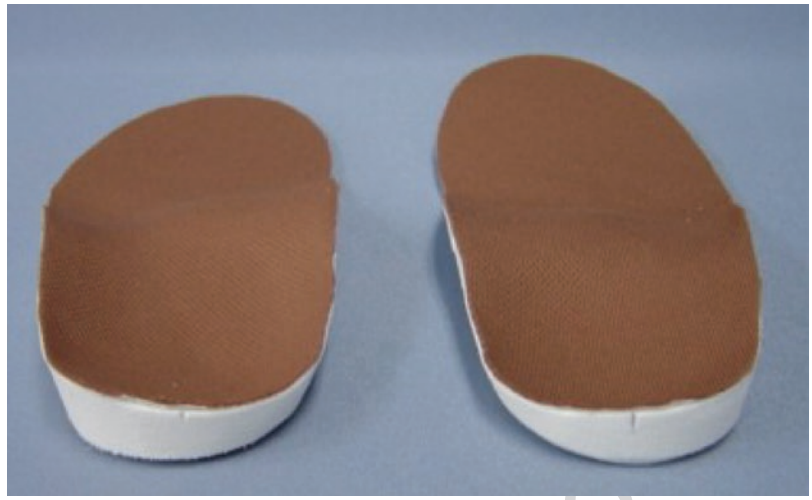

Fig. 21.2 Insoles with lateral wedges

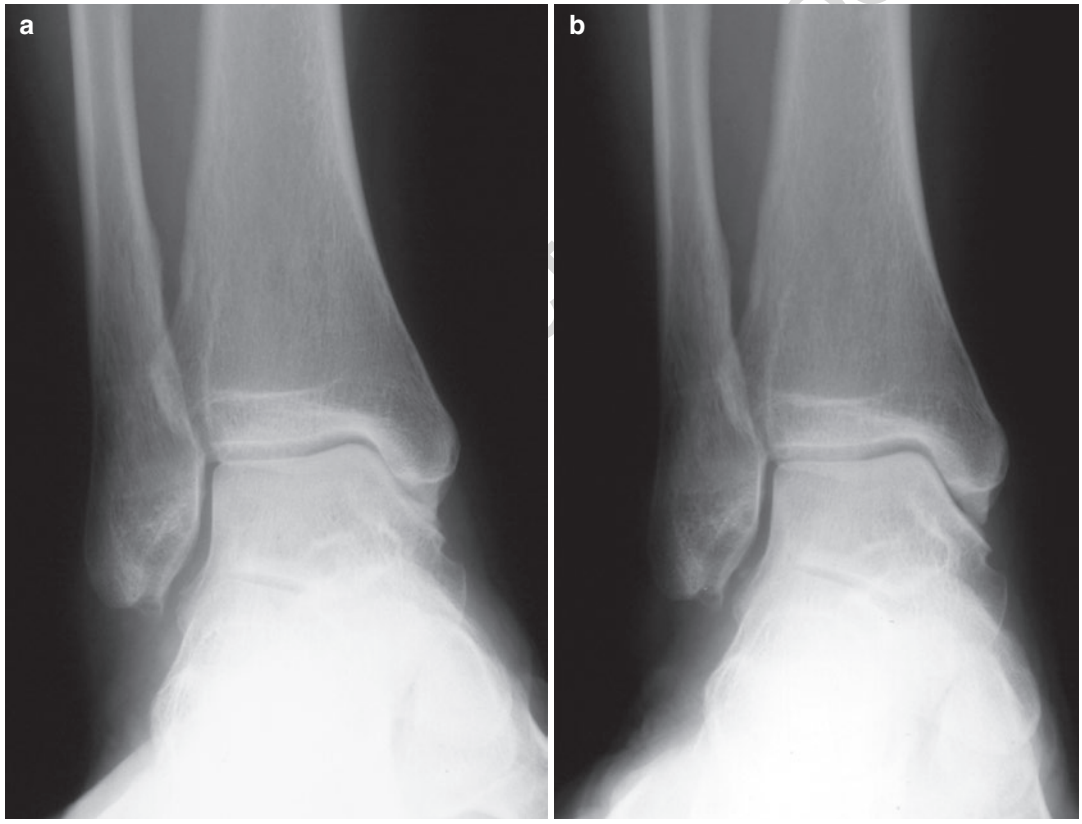

Fig. 21.3 Effect of an insole with lateral wedge. Medial joint space is opened with an insole with lateral wedge. (**a**) Without an insole. (**b**) With an insole

21.1.2.1 Joint Preservation Surgery

Arthroscopic Debridement

Arthroscopic debridement yields positive outcomes in anterior impingement syndrome caused by bony or soft tissue [19], though its effects are limited in cases of advanced ankle OA [20]. Symptomatic improvement in cases with no cartilage remaining is difficult using arthroscopic debridement alone.

Distraction Arthroplasty

While arthrodesis is often indicated for posttraumatic OA in young people, distraction arthroplasty may be indicated if there is a desire to conserve joint mobility. In this procedure, a joint external fixator is used to stretch the joint while mobilizing it until the cartilage can repair. This is reported to produce positive short-term outcomes [21]. While much about its mechanism remains unclear, it is a good option to try before considering total ankle arthroplasty (TAA) or arthrodesis.

Ankle Lateral Ligament Reconstruction and Distal Tibiofibular Ligament Reconstruction

Many studies have demonstrated a close relationship between ankle instability and OA of the ankle [22, 23]. Under loading, the talus subluxated anteriorly onto the tibial pilon, which creates a shearing force on the articular surface that can cause OA [24]. Lateral ligament reconstruction is effective for ankle OA accompanied by ankle instability, and Takao et al. [25] reported that ligament reconstruction and arthroscopic debridement produced positive outcomes in stage II ankle OA. However, it does not appear that joint instability alone can cause OA. Löfvenverg et al. [26] examined the long-term courses (18–23 years) of old lateral ligament injuries and found osteoarthritic changes in only 6 of 46 cases (13%), which suggests the involvement of bony factors besides instability. Lateral ligament reconstruction is often combined with surgery to correct bone alignment [27]. Since tibiofibular ligament injury can also cause OA of the ankle, ligament reconstruction may be indicated in such cases.

Corrective Osteotomy

Osteotomy is an important surgical style for improving a joint's biomechanical environment and achieving functional recovery. In cases of malunion after malleolar fracture with mild osteoarthritic changes, the aim is to correct the deformity. Anatomical correction of the lateral malleolus is important [28]. In cases of shortening, the talus is displaced laterally, for which lengthening of the lateral malleolus is indicated (Fig. 21.4) [29]. If there is persistent subluxation of the ankle, scar tissue filling the medial gutter must be removed. Low tibial osteotomy is indicated in cases with remnant cartilage, such as in early closure of the epiphyseal line or tibial fracture malunion [30, 31].

The morphological characteristics of varus-type OA of the ankle include inversion and anterior opening of the articular surface of the distal

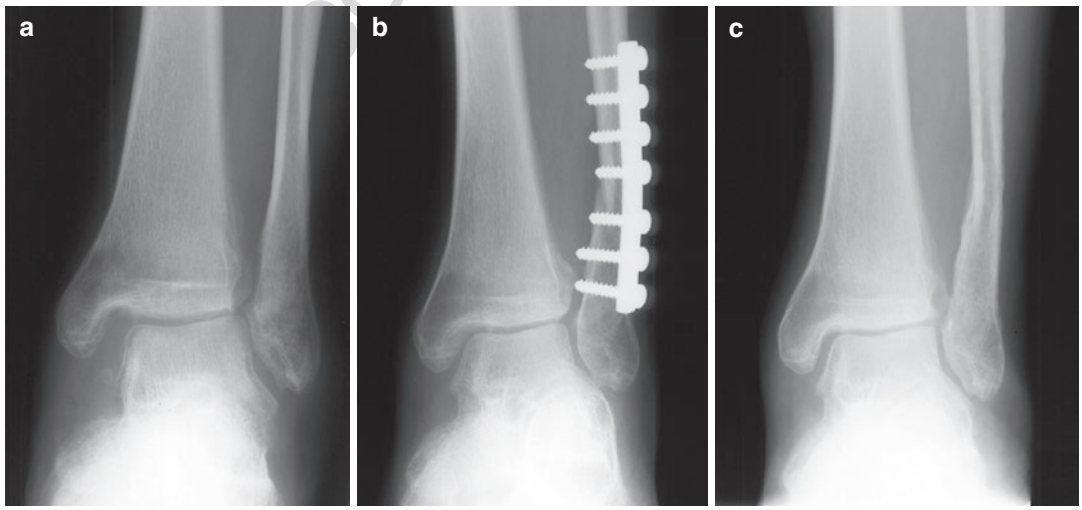

Fig. 21.4 Fibular osteotomy for posttraumatic osteoarthritis (Maisonneuve fracture). (**a**) Pre-op. (**b**) Post-op 9 months. (**c**) Post-op 2 years

tibia, which are evaluated by measuring the angles formed by the tibial shaft and the articular surface of the distal tibia—the tibial anterior surface (TAS) angle and tibial lateral surface (TLS) angle on anterior-posterior (AP) and lateral weight-bearing ankle radiographs [32, 33]. These angles are smaller in the presence of varus-type OA of the ankle. Stage II or IIIa cases with this kind of deformation are good indications for low tibial osteotomy [8]. In some cases, there is a large opening of the lateral joint space of the ankle on weight bearing. This procedure is also indicated when weight-bearing talar tilt angle—the angle between the articular surface of the distal tibia and the superior surface of the talus—is less than 10° on anteroposterior weight-bearing ankle radiographs [8]. The operation involves am open-wedge osteotomy at 5 cm proximal to the tip of the medial malleolus, followed by the creation of a wedge for an autogenous bone graft or an artificial bone or an artificial bone, while simultaneously performing osteotomy on the fibula (Figs. 21.5, 21.6, and 21.7). The objective is to correct the varus deformity of the articular surface of the distal tibia in order to redistribute the load concentrated on the medial ankle to the lateral side and to slightly overcorrect the TAS angle to 93–96° and the TLS angle to 81–84°. Better outcomes are observed with overcorrection than with undercorrection. However, studies of clinical outcomes indicate that there are limits to what can be achieved with this kind of regular low tibial osteotomy in stage IIIb cases. Teramoto et al. [34] reported positive outcomes using distal tibial oblique osteotomy (DTOO) in stage IIIb cases. This involves no osteotomy to the fibula and creation of an oblique osteotomy from 4 to 5 cm proximal to the tip of the medial malleolus to just superior to the tibiofibular joint (Figs. 21.8 and 21.9). The objective is to shut the opening of the lateral ankle gutter and achieve the stability of the ankle. It is also indicated in cases with large weight-bearing talar inclination.

21.1.2.2 Ankle Arthrodesis

Ankle arthrodesis is currently considered the gold standard treatment for end-stage ankle OA. It is indicated in stage IIIb or IV cases with advanced osteoarthritic changes and in individuals who use their feet relatively often, such as in jobs that require prolonged standing. In cases of posttraumatic OA after a pilon or talar fracture, it can be difficult to preserve joint functionality; therefore, arthrodesis is often selected. As compared with arthrodesis for the knee or hip joints, patients who undergo this procedure for the ankle report minimal inconvenience to their daily lives. However, progression of OA to adjacent joints remains a potential issue [35, 36].

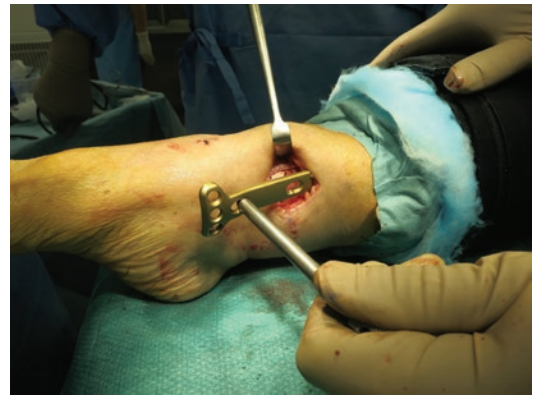

Fig. 21.5 Minimally invasive plate osteosynthesis technique during low tibial osteotomy

Although more than 30 surgical techniques have been reported, they can broadly be categorized according to the approaches, which include anterior, posterior, lateral, and endoscopic. Selection of the surgical style varies by institution. We often use an anterior sliding graft in cases with severe varus or valgus deformity of the ankle. This involves collecting a prism-shaped graft fragment from the anterior surface of the tibia that includes cortical bone and embedding it in a triangular hole created in the talar neck [36]. Staples are used to immobilize the talocrural joint, and screw fixation is used between the bone graft and the tibia. Partial weight bearing with a walking cast begins at 2 weeks postoperatively, with full weight bearing at 4 weeks and cast removal after 5 or 6 weeks. Allowing weight bearing in the early postoperative stages puts traction on the posterior articular surface of the ankle from the tension of the Achilles tendon. The limb is fixed slightly externally rotated and in between plantar flexion and dorsiflexion and varus and valgus. Failing to correct varus deformities in particular can lead to

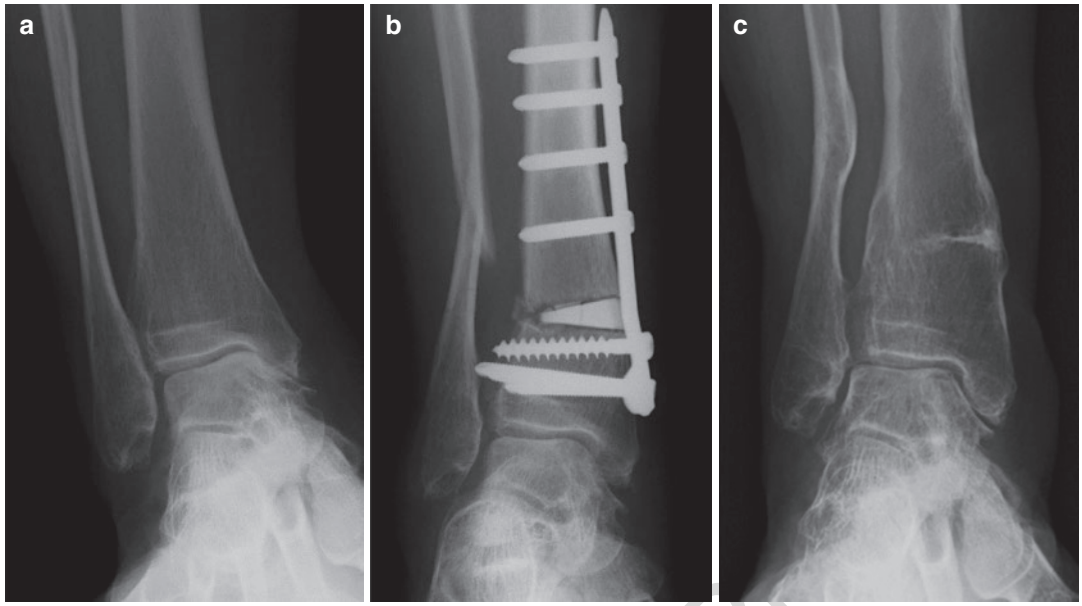

Fig. 21.6 Low tibial osteotomy (stage III-a 64-year-old female). Joint space opened after surgery. A low tibial osteotomy is a good indication for osteoarthritis stage III-a. (**a**) Pre-op. (**b**) Immediate after operation. (**c**) Post-op 5 years

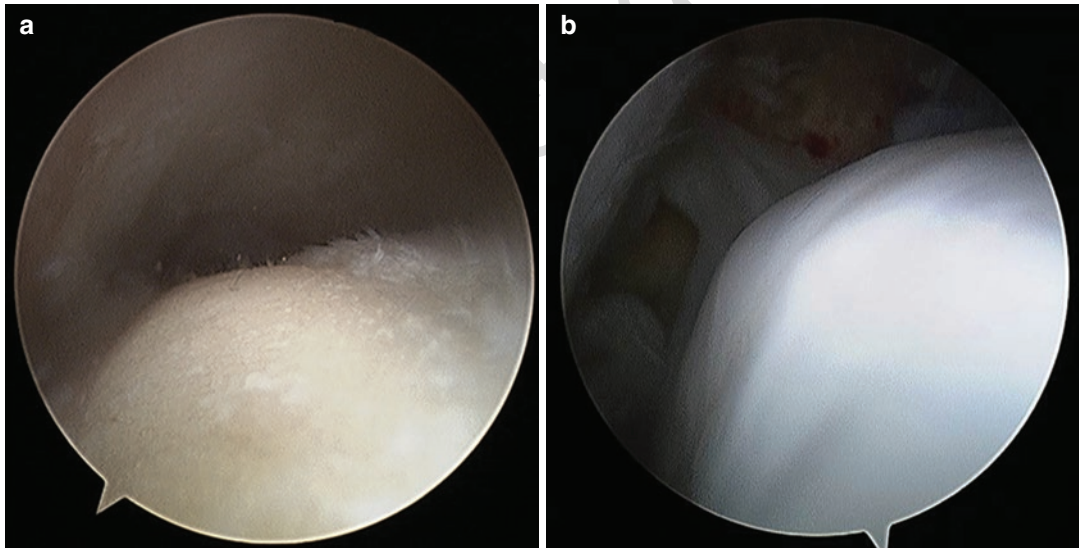

Fig. 21.7 Regenerated fibrocartilage. (**a**) Articular cartilage was completely disappeared at the time of low tibial osteotomy. (**b**) Regenerated fibrocartilage was totally covered on the same area 1 year after the surgery

postoperative pain [36]. Arthroscopic fixation is possible if ankle varus or valgus is no greater than 15° and the osseous defect is not large. Arthroscopic curettage of remaining cartilage and the subchondral plate is performed with a sharp curette and ablator until bleeding from the graft bed is confirmed (Fig. 21.10), at which point fixation is performed using cannulated screws from the medial side of the distal tibia with fluoroscopic guidance. The duration of cast immobilization is the same as in the anterior approach. It is also similar to the anterior approach in that there is little postoperative pain and synostosis occurs relatively quickly (Fig. 21.11). If the ankle and

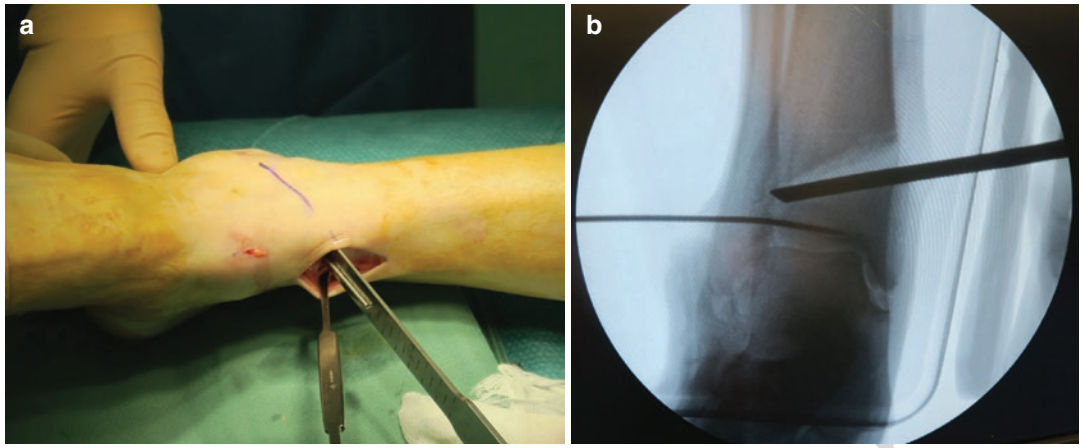

Fig. 21.8 Osteotomy technique of distal tibial oblique osteotomy. (**a**) An oblique cut was made using a thin osteotome following multiple drilling with a Kirschner wire. (**b**) The distal tibiofibular joint was temporally fixed with a Kirschner wire for preventing iatrogenic intra-articular fracture. The osteotomy site is gently opened using a thin osteotome

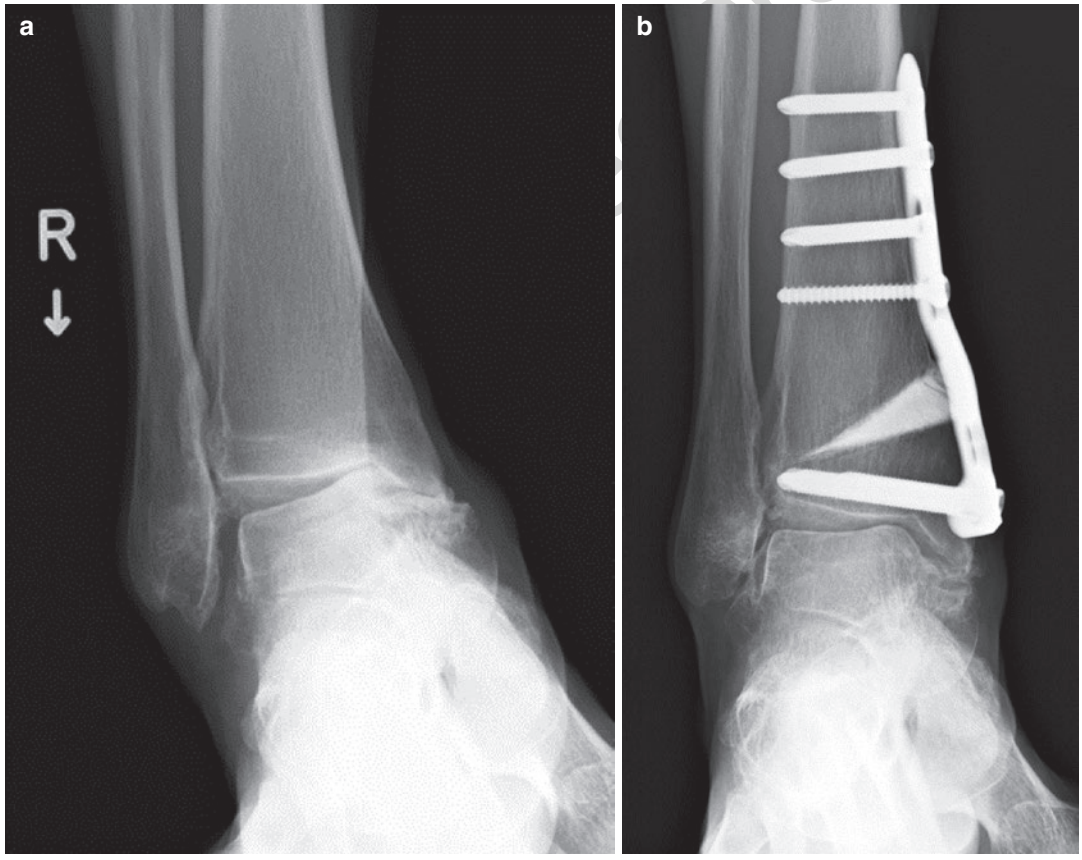

Fig. 21.9 Distal tibial oblique osteotomy (53-year-old female). (**a**) Pre-op. (**b**) Post-op 2 years and 3 months

subtalar joint need to be immobilized simultaneously owing to severe foot deformity or osteoarthritic changes to the subtalar joint, it is helpful to use a lateral approach with transverse locking screws in intramedullary nails. Using this method, good visualization can be obtained and the bone graft collected from the fibula can be used to perform immobilization.

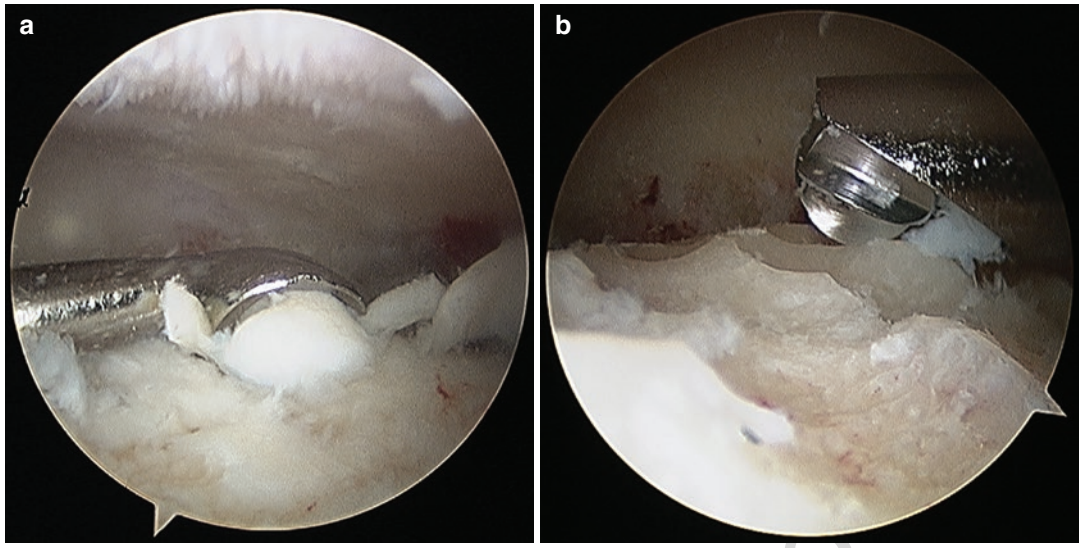

Fig. 21.10 Arthroscopic ankle arthrodesis. (**a**) Removing residual articular cartilage with a curette. (**b**) Subchondral bone plate was removed using an abrader

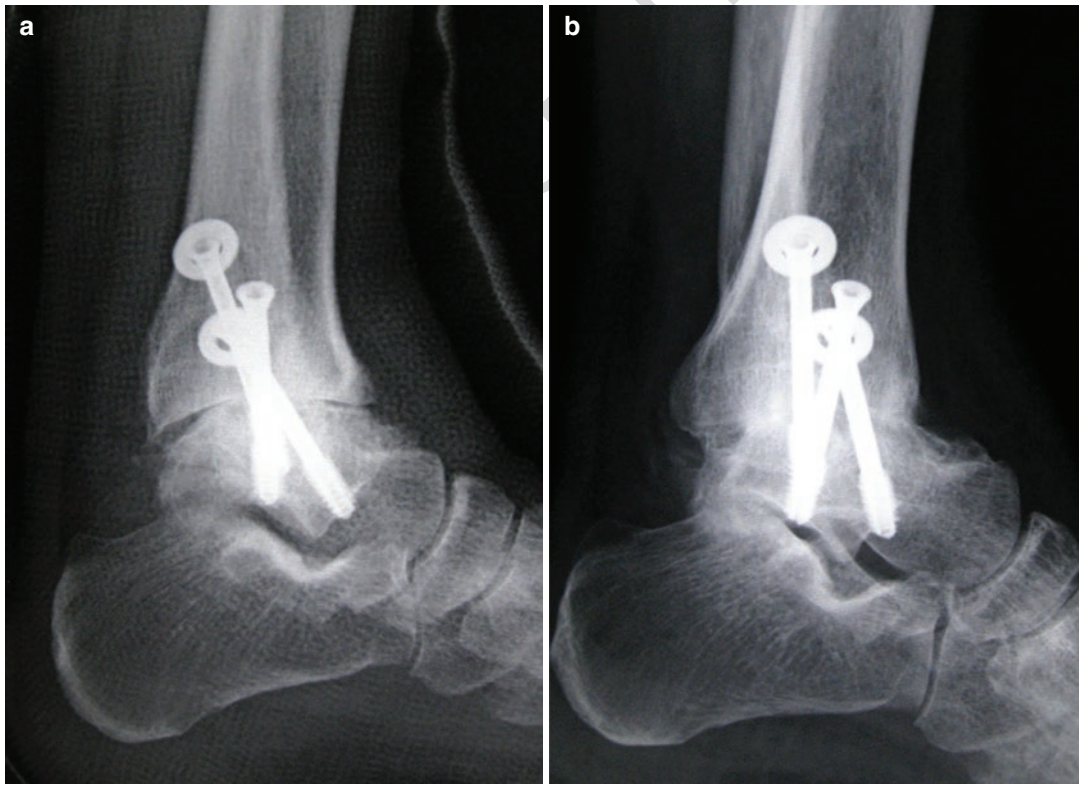

Fig. 21.11 Arthroscopic ankle arthrodesis (57-year-old male). There were gaps at an anterior and posterior side of the ankle at immediate after surgery. Bone union achieved at 2 months after surgery and gaps were filled out by regenerated bone. (**a**) Immediate after surgery. (**b**) Post-op 2 months

21.1.2.3 Total Ankle Arthroplasty (TAA)

Ankle replacement designs were associated with poor outcomes up to the 1980s, and TAA received little attention for many years. However, advances in biomaterials and surgical techniques starting in the 1990s led to the development of several new types of artificial ankle joints that markedly improved outcomes [37, 38]. According to our research, more than 30 different types of ankle replacements are currently used worldwide. Unconstrained joints with three-component designs are mainstream, and positive outcomes have been reported using the STAR ankle since the 1980s [39]. However, 5-year survival rates of 70% have been reported, with 16% of patients opting not to repeat the surgery, which indicates a degree of dissatisfaction [40]. Artificial joints with two-component designs have continued to improve and are now being used around the world. The TNK ankle developed at Nara Medical University is a semi-constrained variety of this type [41].

In terms of indications, it can be difficult to determine when to use TAA and when to use ankle arthrodesis. Haddad et al. [42] carried out a systematic review on the differences in outcomes between TAA and ankle arthrodesis. In ten reports (852 total cases) on the intermediate outcomes of TAA, the reoperation rate was 7%, with the primary reason being loosening of the implant. In 39 reports (1262 total cases) on ankle arthrodesis, the reoperation rate was 9%, with the primary reason being nonunion. According to the score of American Orthopaedic Foot and Ankle Society, the mean clinical assessment of TAA was 78.2 points, while that of ankle arthrodesis was 75.6 points (max. 100 points), leading to the conclusion that the outcomes were roughly equivalent. That said, the procedure is generally recommended in patients who are at least 50 years old and have varus or valgus deformity ≤15°, though successful outcomes can be difficult to achieve in cases of severe obesity. TAA is the preferred choice for patients who are affected bilaterally or who have impairments in adjacent joints, as arthrodesis can severely limit ROM.

The TNK ankle we use is made of alumina ceramic, which has had its surface treated with beads to improve adhesion with the bone since 1991 [41]. Calcium phosphate paste came into clinical use starting in 2000. At our institution, its normal concentration is diluted two to five times before being applied to surfaces. To further increase bone bonding, the joint is placed on bone marrow aspirate when it is inserted. Overall, TNK ankle outcomes are characterized by low revision rates. Of the first 70 cases, the revision surgeries were performed in only 3 cases. Furthermore, nowadays we devised an artificial total talar prosthesis which was made by alumina ceramic (Fig. 21.12) [43]. If

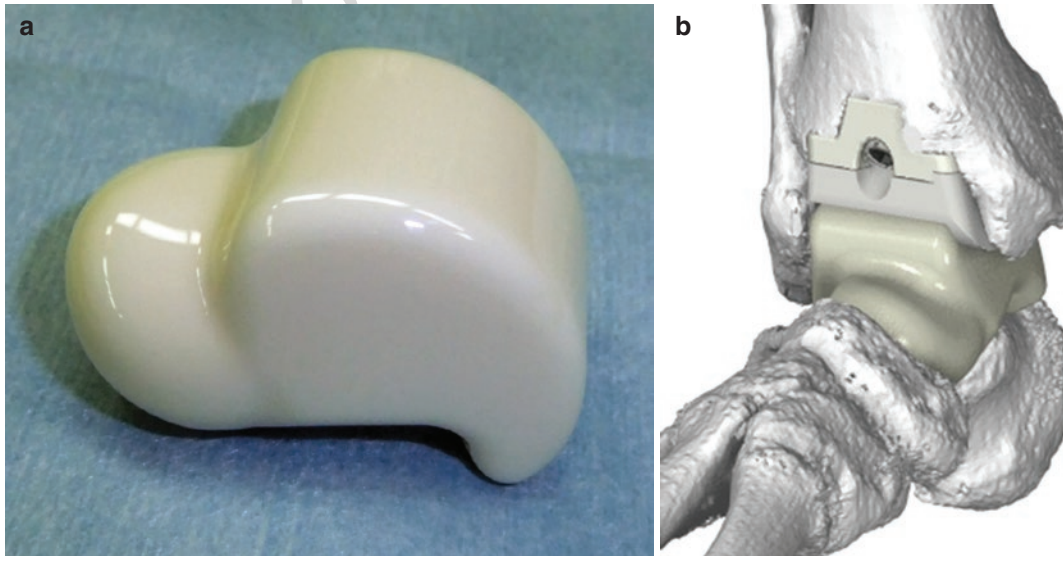

Fig. 21.12 Alumina ceramic total talar prosthesis. (**a**) Alumina ceramic total talar prosthesis. (**b**) Combined total ankle arthroplasty

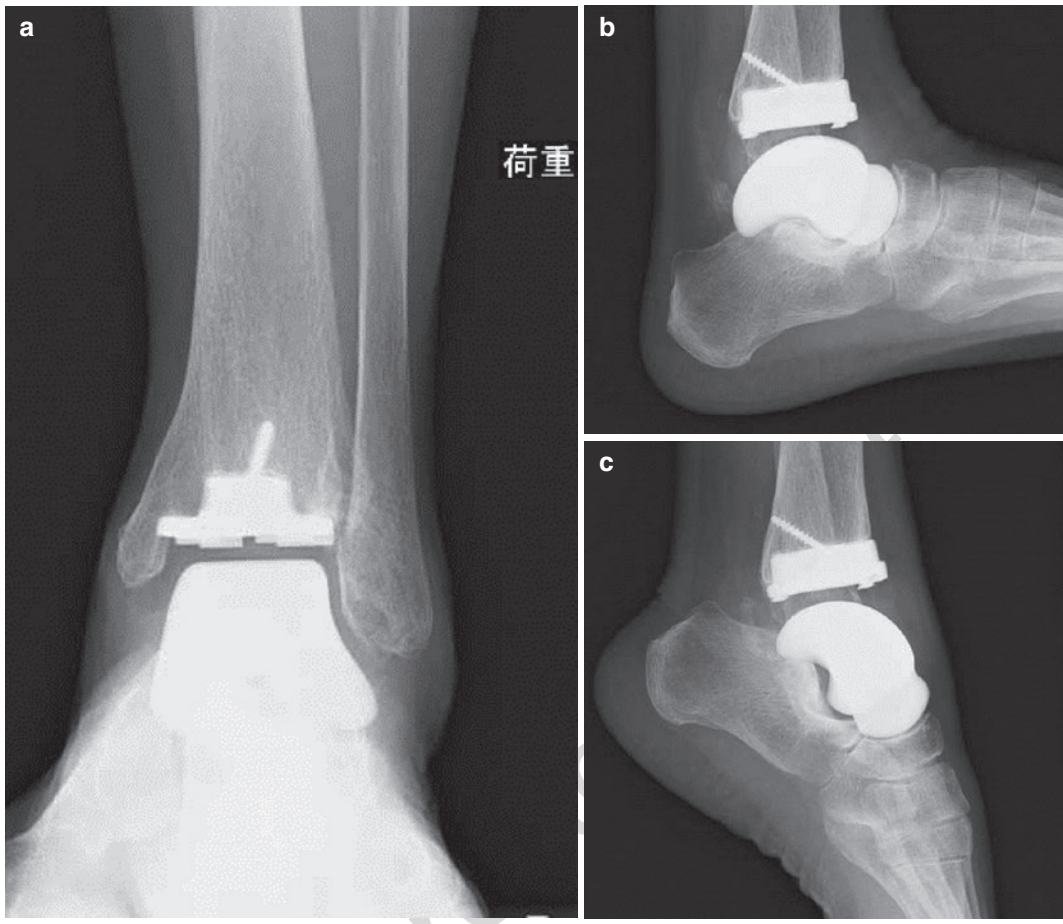

Fig. 21.13 Total ankle arthroplasty using an artificial talus (4 years after the surgery). (**a**) AP weight-bearing view. (**b**) Dorsiflexion. (**c**) Plantar flexion

the talus in the patients is severely destroyed, total ankle arthroplasty using a total talar prosthesis (combined TAA) is selected. Combined TAA using an artificial talus can also be performed in cases of severe talar deformation (Fig. 21.13).

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Jones Fractures

22

K. C. Doan and Kenneth J. Hunt

22.1 Introduction

Fractures involving the base of the fifth metatarsal were first described in 1902 by Sir Robert Jones. In his paper, "Fracture of the Base of the Fifth Metatarsal Bone by Indirect Violence," Jones introduced the first controversy surrounding fractures of the base of the fifth metatarsal. Over the years, understanding of fractures about the base of the fifth metatarsal has evolved; however treatment controversies have persisted. Multiple classification systems, treatment strategies, and surgical techniques have been developed over the years to address this diverse group of fractures. Due to the complex bony and ligamentous anatomy, mechanics, and blood supply of the proximal fifth metatarsal [1], these fractures continue to challenge orthopedic surgeons and the patients in which the fractures occur.

22.2 Epidemiology and Importance

Fractures of the fifth metatarsal are the most common fracture in the foot. The fifth metatarsal is the most commonly fractured metatarsal, comprising about 68% of all metatarsal fractures [2]. The Jones fracture, or fracture of the base of the fifth metatarsal at the meta-diaphyseal junction entering the fourth–fifth inter-metacarpal joint (Fig. 22.1), accounts for about 3–10% of all meta-

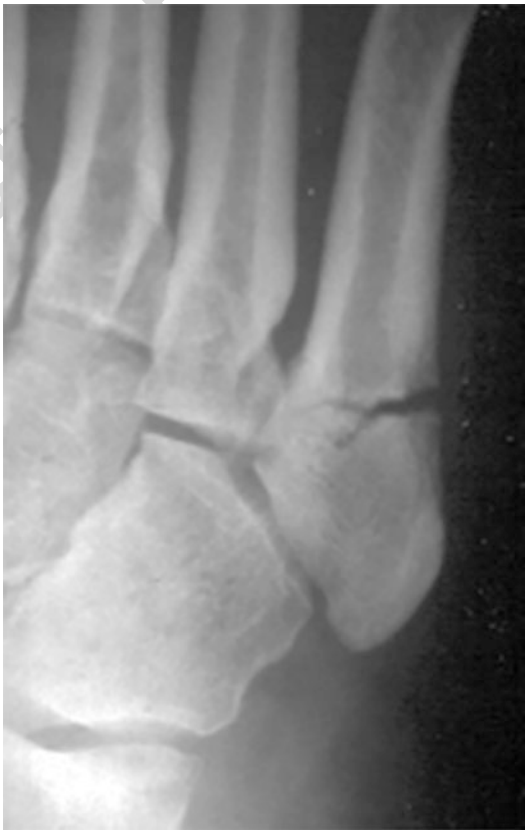

Fig. 22.1 Oblique radiograph demonstrating Jones fracture

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tarsal fractures. Fractures more proximally and more distally to this are also common, with fractures of the tuberosity proximally being the most common [1]. These are avulsion-type fractures of the peroneus brevis attachment and are commonly, and perhaps inaccurately, referred to as "pseudo-Jones" fractures. Stress fractures often occur more distally approaching the diaphysis, and have been reported to be rare, constituting only about 1% of all metatarsal fractures in a large series [3]. These are important to recognize due to their underlying mechanism of injury, healing rates, and subsequent treatment strategies.

Base of the fifth metatarsal fractures are common in a large patient demographic and do not distribute in the standard bimodal distribution seen in other traumatic orthopedic injuries. These fractures, conversely, are seen often in athletic populations and show the highest incidence between the ages of 20 and 50 years [2]. As Jones originally described, this is likely due to the mechanism of injury. Recently, new evidence also suggests that intrinsic anatomic factors may play an important role. Base of the fifth metatarsal fractures are reported after a wide variety of acute mechanisms, or can be of insidious onset. They can also present with subtle or difficult-to-interpret symptoms of pain to the lateral aspect of the foot. It is important to note that a large portion of these injuries arise from relatively minor trauma, or from repetitive impact athletic activities, making it imperative to have a high index of suspicion when dealing with all patients, especially athletes with lateral mid-foot pain.

22.3 Evaluation and Treatment Considerations

It is important to obtain a thorough history and physical examination of the patient being evaluated for a fracture of the base of the fifth metatarsal. Often, in the history the patient will recall several weeks of discomfort prior to the inciting event that brought them for evaluation [4]. Conversely, these fractures may be the result of an acute trauma in isolation such as an MVC, jump or fall from height, or a cutting-type maneuver in athletes. This differentiation is important as subacute and stress fractures of the fifth metatarsal are more prevalent in patients with cavovarus deformity and metatarsus adductus [2, 5], and successful treatment of this specific injury pattern is highly dependent on recognition of this deformity. In addition to a comprehensive history and physical examination, it is necessary to evaluate the patient medical comorbidities. Blood supply to the area of the base of the fifth metatarsal is tenuous as it is a well-recognized watershed area. Treating any underlying medical comorbidities which contribute to poor vascular supply could theoretically improve postoperative outcomes both in the surgical and nonsurgical settings. Appropriate imaging should always be obtained including weight-bearing anteroposterior, oblique, and lateral foot radiographs at the time of initial evaluation. Given the subacute or chronic nature of some of these fractures however it can occasionally take 3–6 weeks before radiographs show evidence of fracture reabsorption [3]. In this and other instances, it may be appropriate to obtain advanced imaging to evaluate patients with a concerning history or physical exam but without radiologic evidence of fracture. MRI, or technetium bone scan, can show acute inflammation in the area of an occult fracture, and CT can be of particular use when evaluating for refracture or bone healing and cortical union.

In order to successfully treat fractures about the base of the fifth metatarsal, multiple classification systems have been described. The two most common classification systems in use today are the anatomic classification system first described by Lawrence and Bottle, and the radiographic appearance classification system described by Torg [1, 4]. The anatomic classification system divides the base of the fifth metatarsal into three zones. Zone 1 is the most proximal at the level of the peroneus brevis insertion on the tuberosity which can involve the metatarsal–cuboid joint. Zone 2 is located at the metadiaphyseal junction at the level of the fourth/fifth intermetatarsal joint—the so-named Jones fracture. Zone 3 extends distally an additional 1.5 cm into the diaphysis of the fifth metatarsal.

Torg's radiographic appearance classification system attempts to qualitatively describe the chronicity of the fracture. Type I therefore represents an acute fracture with sharp margins and minimal cortical hypertrophy or periosteal reaction. Type II is a more delayed healing picture demonstrating early intramedullary sclerosis, bone reabsorption, and associated periosteal reactions. Type III is a fracture nonunion with wide fracture line, periosteal new bone, and complete obliteration of the metal medullary canal with sclerotic bone. Both of these classification systems help guide treatment strategies by identifying which fractures will do well when treated nonoperatively with or without orthotics [6], which patients should be evaluated for a deformity leading to their fracture [5], and which will require specific surgical interventions for optimal outcome [7, 8].

The majority of fractures of the base of the fifth metatarsal will heal without surgical intervention [1, 4, 6, 9]. Zone 1 fractures typically do well with nonoperative management, even in the setting of displacement greater than 2 mm [9]. Some unacceptably displaced fractures, particularly with a stepoff at the joint, may require fixation with percutaneous pinning, tension band, or hook plate internal fixation. It is also important to consider orientation of fracture lines in this zone in the skeletally immature, such as in Iselin's disease, or apophysitis of the fifth metatarsal will show only the normal longitudinally oriented apophysis on radiographs. Zone 2 fractures also often heal regularly with nonoperative management; however there are some drawbacks to nonoperative management especially in the athletic population which will be discussed in depth in subsequent sections. Zone 3 fractures need consideration of mechanism of injury and foot alignment to develop appropriate treatment approach [3, 5].

22.4 Nonoperative Management

Nonoperative management requires prolonged non-weight bearing, initially at least 6–8 weeks until radiographs show evidence of healing and there is no longer tenderness to palpation at the fracture site. The average time to union is sited around 15–19 weeks [10]. Additionally, nonoperative management has nonunion rates approaching 30% and an increased rate of refracture when compared to operative management [10]. Operative management conversely has union rates around 96% which occur on average at 6–8 weeks and also allows earlier return to sport with less risk of refracture [10]. Zone 3 injuries should be differentiated between acute and chronic. Acute injuries in a nonathletic population can be offered a trial of nonoperative management; however subacute and chronic fractures especially in athletes are recommended to undergo surgical fixation possibly with bone graft augmentation [5]. A recent decision analysis model indicated that given current healing rates, operative treatment is the preferred treatment approach in elite athletes, consenting patients who prefer to limit the risk of nonunion, and patients with evidence of stress fractures with delayed or nonunion [11].

22.5 Surgical Techniques

Outpatient surgical fixation of base of the fifth metatarsal fractures can be preformed using percutaneous, limited open, and open techniques depending on the specific fracture pattern, fixation construct, and other factors, like chronicity. Regional block with monitored anesthesia care such as a popliteal or ankle block is reasonable; however the authors prefer general anesthesia for ease of positioning as well as limiting risks of nerve complications. Appropriate patient positioning is critical to allow adequate fluoroscopic imaging to be obtained (Fig. 22.2). The authors recommend ensuring that all views can be obtained adequately before draping is preformed, especially if a percutaneous approach is going to be used. The patient is positioned supine on the operating table with a bolster under the affected extremity. This allows for internal rotation and adequate exposure of the lateral aspect of the foot. A well-padded tourniquet is placed on the affected thigh; however it is not routinely

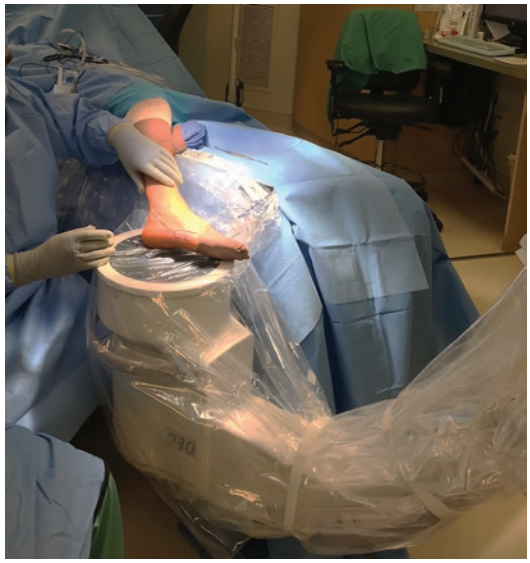

Fig. 22.2 Photographs demonstrating positioning for Jones fracture fixation with the sterile image intensifier adjacent to the operating table

inflated. Intravenous antibiotics are administered prior to incision, and the extremity is prepped and draped in standard fashion, usually with the entire limb to the level of the tourniquet exposed to allow for adequate knee flexion to obtain fluoroscopic imaging. The importance of fluoroscopy for assessing fracture reduction, screw length, screw diameter, hardware starting point, intramedullary position, and fracture compression cannot be overstated, as each fixation method has specific pearls and pitfalls for optimizing fixation and mediating risk of failure or iatrogenic injury.

22.6 Intramedullary Screw Fixation

The most common method of zone 2 and 3 fifth metatarsal fractures remains the single intramedullary screw. This section focuses on intramedullary screw fixation for primary fixation of acute or chronic fractures of the base of the fifth metatarsal.

Use of a single percutaneous intramedullary screw begins with identification of the surface landmarks. The tuberosity can be palpated along with the metatarsal shaft distally. The intramedullary canal can be assessed in relation to these using fluoroscopy. A single incision is used proximal to the base of the tuberosity in line with the intramedullary canal. The incision is traced at appropriate level proximal to the tuberosity about 2 cm in length. After the skin is sharply incised, blunt dissection is carried down to the base of the fifth metatarsal. It is important to realize that the sural nerve lies superficially here, and the peroneus brevis and lateral band of the plantar fascia are at risk with this approach; all should be protected throughout the procedure. A Kirschner wire is introduced and appropriate starting point is assessed. It is important to utilize the "high and inside" start point in order to get appropriate trajectory within the intramedullary canal. This correlates with the most dorsal and medial aspect of the base of the fifth metatarsal without entering the metatarsal-cuboid joint. The K wire is then advanced using fluoroscopic imaging. The position of the guidewire must be centered within the medullary canal on all views, and advanced to the level of the distal metatarsal shaft where there is consistently a curvature which limits screw length. A cannulated drill with soft-tissue protector is then used, again utilizing fluoroscopy and alternating between forward and reverse functions, to ream over the wire and prevent cortical perforation. The wire is maintained in the metatarsal, and a second wire can be used to measure the length of the partially threaded screw. The screw length is then checked prior to insertion by obtaining an image to confirm all threads are distal to the fracture line.

A solid screw of at least 4.5 mm diameter is selected. The largest diameter screw that will fit in the canal is selected. Most of the time, this is a 5.5 or 6.5 mm diameter solid screw. The screw is finally inserted under fluoroscopy to confirm compression and prevent malrotation or iatrogenic fracture (Fig. 22.3). It is important that the screw is not of excessively large diameter that could risk fracture of the metatarsal from circumferential stresses. It is also important that the screw is not too long as the fifth metatarsal canal is nonlinear and if a cortex is engaged distraction can occur (Fig. 22.4). Once final images are

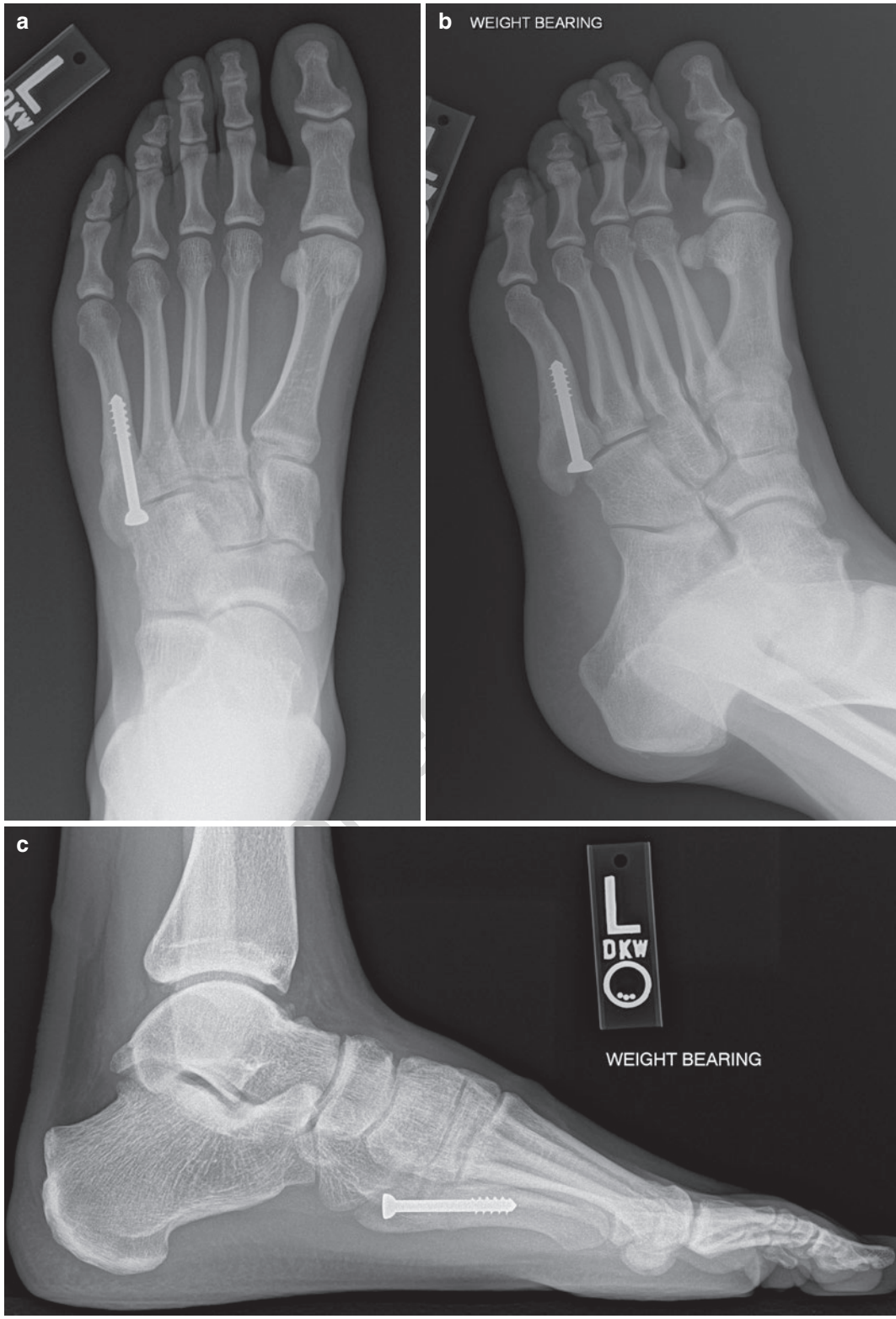

Fig. 22.3 (**a**) Anteroposterior, (**b**) oblique, and (**c**) lateral radiographs demonstrating healed Jones fracture with intramedullary screw fixation

obtained, the wound can then be irrigated and closed in layers above the screw head. If possible, the periosteum should be closed over the screw head and the rest of the wound closed in layers.

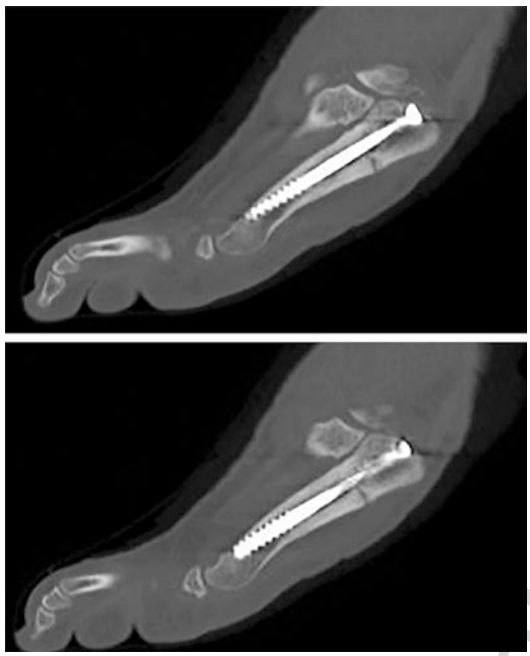

Fig. 22.4 CT scan images demonstrating a long screw abutting distal fifth metatarsal cortex. Fracture nonunion is evident

22.7 Biologic Adjuncts

Use of bone graft and biologics in the treatment of both acute and revision fractures is also a consideration for operatively managed fractures. This has increased relevance with the surge of new products on the market and in the media. New methods for less invasive harvesting, along with improved processing, have made the availability of these products much greater. Given the vascular watershed of the fifth metatarsal, some authors are proponents of routine use of bone graft or other biologic materials, while others use this only in at-risk cases or in the revision setting.

In cases with significant sclerosis and nonunions, the addition of orthobiologic adjuncts can be helpful. In fractures with significant sclerosis, the senior author's preferred technique includes percutaneous curettage and debridement of the fracture site (Fig. 22.5a). A curette is used to remove sclerotic bone and a K wire to drill into bone to promote bleeding (Fig. 22.5b). The resulting gap can be filled with autograft or allograft bone, or a combination. We have previously reported on this technique with excellent results, including elite athletes [12]. Further outcome studies are required to differentiate which fracture types or patient populations would benefit from grafting or biologics in the acute scenario.

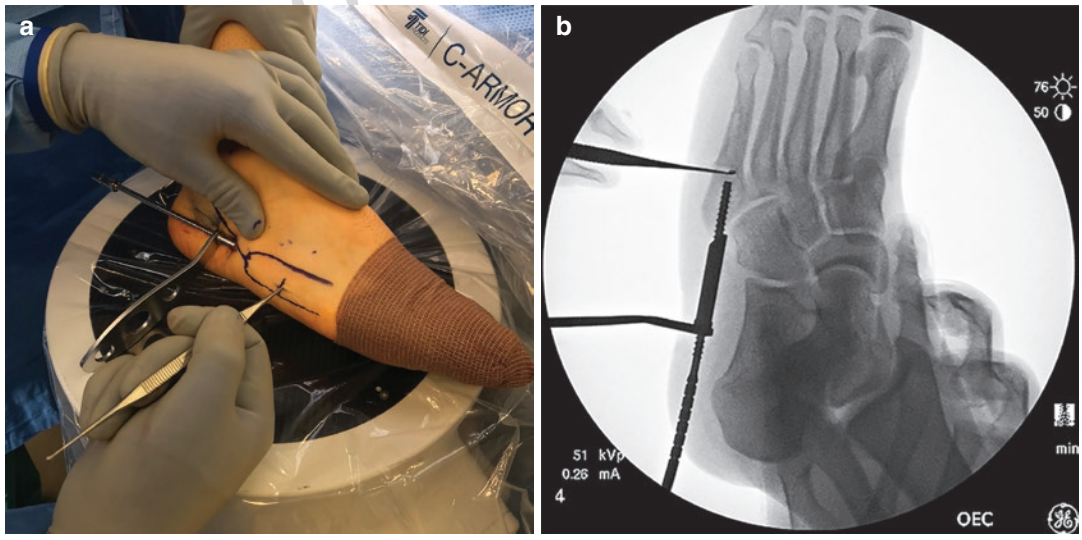

Fig. 22.5 (**a**) Photograph and (**b**) fluoroscopic image demonstrating curettage of non-united fracture site

22.8 Compression Plate Fixation

Plating of fifth metatarsal fractures can be very useful for certain fracture patterns, revision cases, and nonunions (Fig. 22.6), and occasionally for athletes who are at high risk of nonunion or refracture [13]. Specific low-profile plantar plates have been developed to prevent prominence and counter traction forces seen especially in the competitive athletic

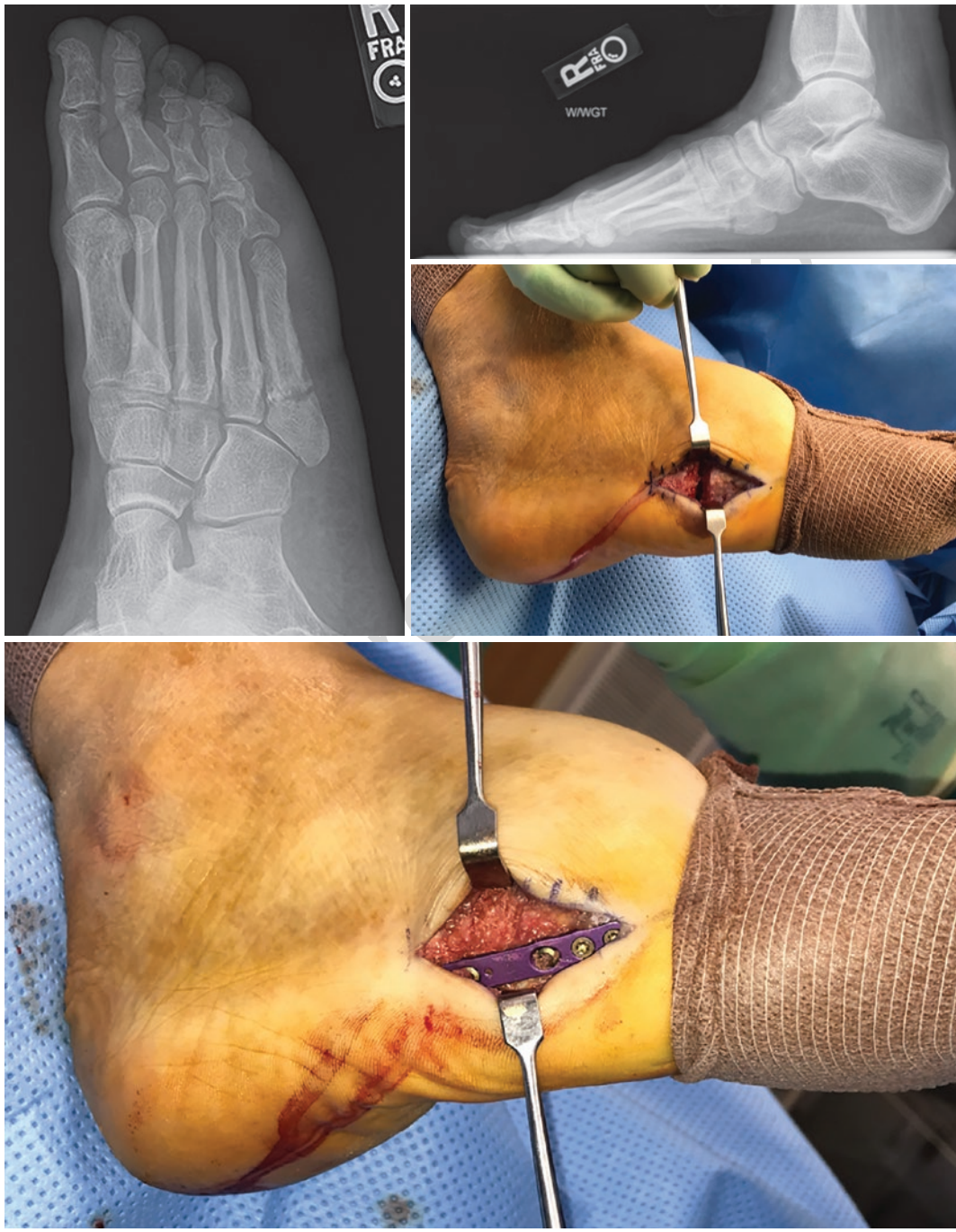

Fig. 22.6 Plate fixation for Jones fracture nonunion

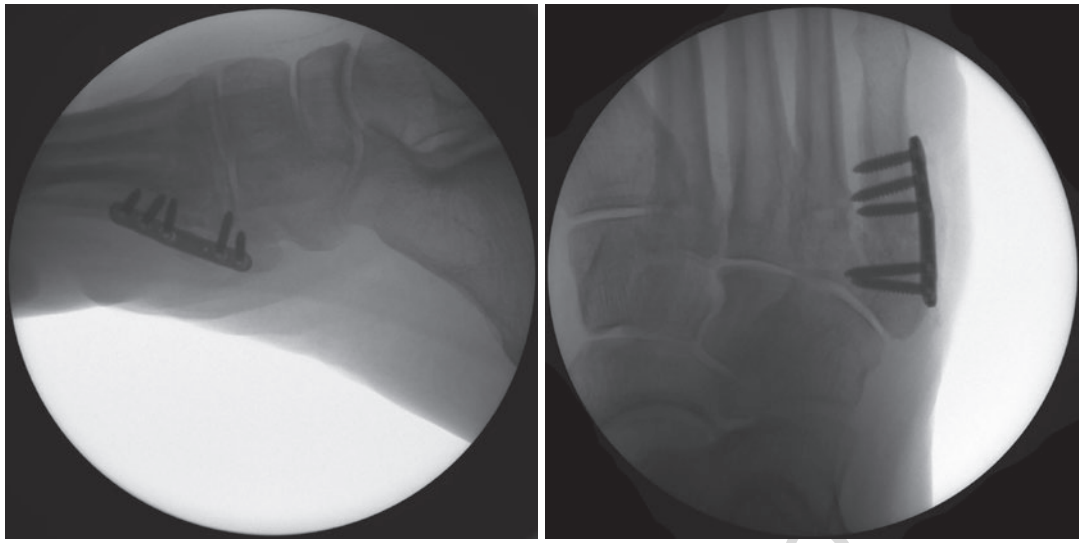

Fig. 22.7 Fluoroscopic images following plate fixation

population [10]. The incision for a plate fixation construct is centered over the fracture site over the lateral foot, slightly plantar. Skin and soft tissues are dissected carefully down to bone with care to preserve superficial sensory nerves if encountered (usually sural nerve is more dorsal than incision). Periosteum is incised and fracture site debrided, minimizing exposure to preserve vascular supply using standard principles of fracture compression and fixation. Once the fracture is reduced and provisionally fixed using either manual reduction or fracture reduction clamps, the plate is applied. Pre-contoured, low-profile compression plates optimize fit and allow fracture-site compression with eccentric screw placement in dynamic holes. The fracture reduction, compression, and fixation should then be confirmed radiographically in orthogonal planes. The incision is then copiously irrigated and closed in layers, again with the attempt to close periosteum over hardware. Figure 22.7 demonstrates final fluoroscopic films of the plate osteosynthesis technique.

22.9 Revision IM Screw and Plate w/Grafting

In revision settings, or in the case of delayed or nonunion, we recommend the routine use of biologic augmentation to the previously described reduction and fixation strategies. Either a single or two-incision approach can be used, but the fracture site should be universally exposed. This is to allow fracture-site debridement of any callus or fibrous scar, removal of any prior fixation, and confirmation of anatomic reduction. The sclerotic fracture margins should be drilled with small-diameter K wire to promote and confirm blood flow with the "paprika sign." [11] The author prefers the use of autologous cancellous bone graft from the iliac crest applied directly at the fracture site. In the case of revision IM nail, a screw of at least 1 mm diameter larger than the prior screw is then inserted as described in prior sections. In the rare case of a large or segmental bone deficit, we recommend the use of iliac crest cortico-cancellous autograft in combination with bone marrow aspirate concentrate.

22.10 Postoperative Care

Postoperatively the patient is immobilized in a cast or splint. Any sutures are removed at the 2-week postoperative appointment. The patient is progressed to weight bearing as tolerated in a short walker boot at the 2–4-week timeframe. Between 6 and 8 weeks, the fracture site is usually minimally tender to palpation and shows radiographic evidence of healing. If the patient is able to ambulate in clinic without pain, they are then transitioned to supportive athletic shoe with custom orthotic insert at the 6–8-week post-op visit. For both primary and revision fixation, athletic patients will typically begin noncontact running and sport-specific rehabilitation programs with anticipated return to sport around 10–12 weeks. When the athlete returns to sport, an orthotic that has a full-length lateral post that extends proximal to the cuboid is utilized. This is also beneficial to accommodate hindfoot varus and metatarsus adductus.

22.11 Outcomes

Base of the fifth metatarsal fractures comprise a diverse group of injuries, and an equally diverse body of literature regarding treatment strategies and outcomes. In two large systematic reviews, acute fractures treated nonoperatively were noted to have about a 75% union rate, while operatively treated fractures with an intramedullary screw had a union rate approaching 96%. Revision and nonunion cases have similar successful results with multiple studies showing union rates >95% [7, 8, 11, 12]. Another advantage of surgical fixation is the faster rate of fracture union. The average time to union of surgically fixed fractures is about 8–10 weeks sooner than nonoperatively managed fractures [9, 14]. This can be of critical importance for return to sport, especially when taking into consideration that nonoperatively managed fractures have higher rates of nonunion and refracture. Multiple studies on professional athletes in the NBA and NFL have shown safe and successful return to sport with operative fixation.

The decision between fracture fixation with intramedullary screw and plantar lateral plating should be tailored to individual fracture patterns and patient needs. The optimal screw size and other properties such as cannulation vs. solid shaft, fully or partially threaded, or variable angle pitch are subjects of contention in the literature. Biomechanical studies comparing strength and resistance to stress of different screw properties are numerous [4, 6, 15], and current consensus from the American Orthopaedic Foot and Ankle Society states "Operative intervention in the form of an intramedullary solid screw is the treatment of choice." In addition, a shared decision-making model analyses indicated strong preference for surgical treatment [16]. The decision to use plantar lateral plating can also be tailored to specific patient needs, and recent biomechanical studies have shown increased cyclical and maximal load to failure of this construct over IM screws [13]. This is an important consideration in athletes where quicker return to sport is desired and the most stable and durable construct is desirable. Further research comparing the many IM screw and plate fixation strategies focusing on healing rates, return to sport, and clinical outcomes is needed before definite recommendation could be made, but reduction of the fracture, an adequate biologic and mechanical environment, and a stable construct are indicated to optimize healing.

22.12 Summary

Fractures of the base of the fifth metatarsal are a diverse and challenging problem for the treating orthopedic surgeon. As surgical fixation strategies have evolved, outcomes are well understood, and patient demands continue to increase, the threshold for surgical intervention has lowered. During the shared decision-making process, all factors including union rates, time to union, and

increased risk of refracture associated with non-operative management should be discussed with the patient. The optimal fixation strategy and decision of the most appropriate fixation construct should be tailored to the individual fracture pattern and patient factors. If performed well in a compliant patient, excellent outcomes with early return to sport can be anticipated both in acute and revision scenarios. There is a trend toward a more conservative return-to-sport strategy following surgical repair to further reduce refracture rates. Further study will help delineate which patients will benefit further from realignment surgeries, specific fixation constructs, as well as biologic supplementation as the treatment of base of the fifth metatarsal fractures continues to evolve.

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Hallux Rigidus

23

Stephanie L. Logterman and Kenneth J. Hunt

23.1 Background

Hallux rigidus refers to osteoarthritis of the first metatarsophalangeal (MTP) joint and is the most common degenerative joint disease in the foot [1]. In athletes, hallux rigidus is the most common pathology of the first MTP joint and causes considerable disability in this population [2]. Despite its relative frequency in athletes, hallux rigidus has received minimal consideration in the sports literature. During the gait cycle, the first MTP joint receives about 119% of the bodies' weight with each step [3]. Hallux rigidus is characterized by joint pain and limited motion of the first MTP joint, specifically dorsiflexion. The natural history of the disease involves cartilage degeneration with osteophyte formation dorsally and associated pain that gradually progresses to involve the entire first MTP joint [4]. The exact etiology of the disease has yet to be fully elucidated; however, several potential causes exist. Traumatic injury and osteochondral lesions to the articular surfaces, in addition to biomechanical and structural factors such as hallux valgus, hypermobility of the first ray, and metatarsus adductus, are just a few associated factors and potential causes [5–7]. In the athletic population, repetitive hyperextension of the first MTP joint during push-off is oftentimes the inciting mechanism.

Patients often present with pain and stiffness of the first MTP joint. Initially, the pain is located dorsally and then progresses to diffuse joint pain. Patients usually report pain during activity, especially with toe off. They may also note a dorsal prominence that becomes painful from inflammation due to shoe wear. On exam, patients often have tenderness to palpation at the first MTP joint in addition to restricted dorsiflexion (usually <30°), dorsal osteophytes, and synovitis. Hallux rigidus is classified radiographically with three grades. On X-ray, grade I hallux rigidus is characterized by mild to moderate osteophyte formation with preservation of the joint space, while grade II involves moderate osteophyte formation with evidence of joint space narrowing and subchondral sclerosis (Fig. 23.1). Grade III changes on X-ray demonstrate significant osteophyte formation with severe loss of the first MTP joint space and subchondral cyst formation [1].

Initial nonoperative management of hallux rigidus includes foot orthosis, shoe modifications, or steroid injections [1]. Activity modification is not practical option for high-level athletes. For symptomatic patients who have failed conservative management, many different surgical options exist. The most common surgical interventions include cheilectomy, arthroscopic cheilectomy, and arthrodesis. Joint-destructive procedures such as arthrodesis provide definitive

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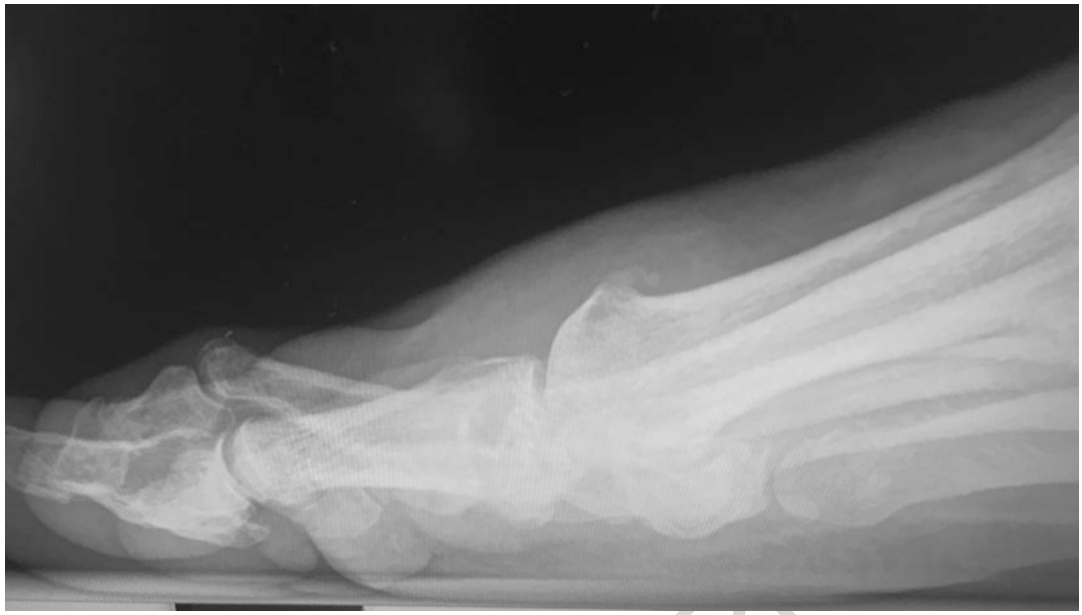

Fig. 23.1 Lateral radiograph of the forefoot demonstrating a dorsal osteophyte, joint space narrowing, and subchondral sclerosis

and predictable results; however, motion-preserving surgeries like hemiarthroplasty or synthetic cartilage implant may be more advantageous in some patients, although there is little data in athletes. Currently in the literature, there is fair evidence in support of arthrodesis (grade B) and poor evidence (grade C) for cheilectomy and implant arthroplasty for the treatment of hallux rigidus [8].

23.2 Cheilectomy

Cheilectomy involves resection of both the dorsal osteophyte and the dorsal one-third of the metatarsal head articular surface. Furthermore, any loose bodies are also removed and a synovectomy is performed. The procedure was first described by Mann and DuVries in 1979 [9]. Currently, there are multiple methods for performing a cheilectomy including open, arthroscopic, and percutaneous. Surgeons need to consider the athlete's functional expectations and the clinical examination when choosing the type of cheilectomy to perform. The size of the dorsal osteophyte, presence of loose bodies, and presence of a lateral osteophyte seen on radiographs also help to guide decision-making as a lateral spur is not amenable to minimally invasive cheilectomy and may necessitate the use of an accessory portal if an arthroscopic procedure is pursued. Open cheilectomy remains the gold standard for treatment of early hallux rigidus; however, percutaneous and arthroscopic techniques are minimally invasive. It is recommended to obtain an MRI preoperatively to assess for any joint degeneration that may not be obvious on X-ray when considering a percutaneous procedure as a percutaneous cheilectomy does not allow for visualization of the joint surface. An arthroscopic cheilectomy is typically chosen when the surgeon desires a minimally invasive procedure but also needs to also assess the articular surface, such as when a central osteochondral lesion is suspected.

Cheilectomy is most often indicated for early-stage hallux rigidus (grades I and II); however, some authors advocate for the procedure regardless of stage of involvement [10–12]. It is usually performed for athletes presenting with mild first

MTP joint dorsiflexion stiffness and dorsal pain without through-range symptoms, rest pain, or plantar pain and with a negative grind test [13]. Cheilectomy may be considered so long as no significant bone loss exists despite radiographic evidence of advanced joint degeneration. This is due to the fact that the radiographic grading system does not correlate well with the potential for joint-preserving surgery nor is it predictive of outcome [14].

The open cheilectomy technique is typically performed through a dorsomedial or dorsolateral incision. Great caution must be used to avoid injury or scarring of the EHL tendon and dorsomedial cutaneous nerve. Release of plantar adhesions can be helpful to restore motion, but this is not typically necessary in athletes. It is important to achieve at least 80° of dorsiflexion intra-op since dorsal scar formation will limit ROM in some cases post-op (Fig. 23.2). Arthroscopic and minimally invasive cheilectomy techniques have been described and are associated with less postoperative swelling and improved motion postoperatively [15] (Figs. 23.3 and 23.4).

Cheilectomy offers many advantages including preserving motion and maintaining joint stability. It also has a low morbidity and may allow for secondary procedures in the future. In addition, arthroscopic and percutaneous techniques may offer less swelling and a shortened recovery time after surgery. While cheilectomy relieves pain in athletes, it does not result in normal hallux

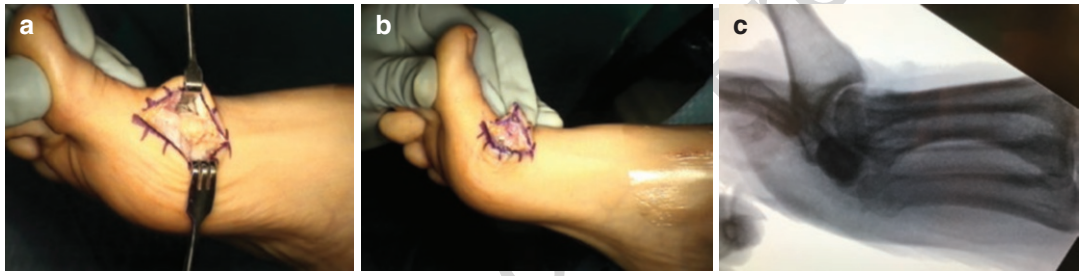

Fig. 23.2 (**a**) Intraoperative view of the first MTP joint after dorsal osteophyte excision. (**b**) Improved dorsiflexion to at least 80° after cheilectomy. (**c**) Shows radiographic evidence of improved postoperative first MTP dorsiflexion

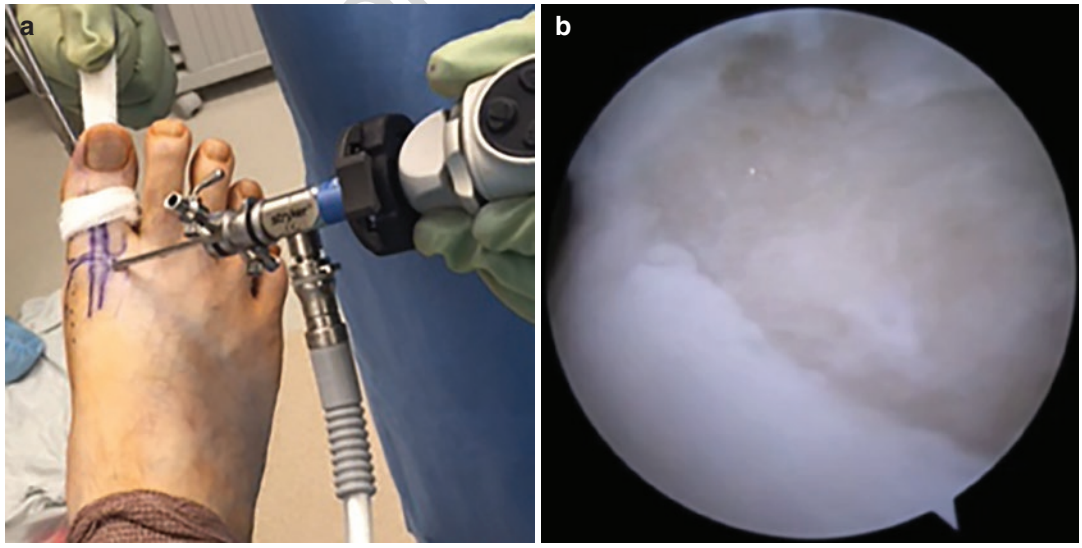

Fig. 23.3 (**a**) Lateral portal placement for arthroscopic cheilectomy. (**b**) Arthroscopic image demonstrating the dorsal metatarsal after osteophyte excision

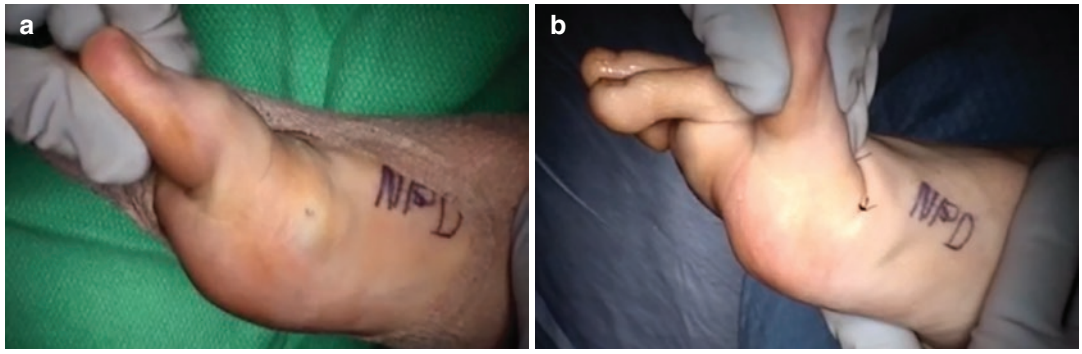

Fig. 23.4 (**a**) Preoperative and (**b**) postoperative first MTP dorsiflexion, respectively, following MTP arthroscopic cheilectomy

function [2]. It is also important to consider the impact of alignment on the condition and treatment outcomes. Patients are more likely to have associated hallux valgus interphalangeus deformities with hallux rigidus. Osteotomy techniques, like a combined Moberg and Akin osteotomy, can be a helpful adjunct to restore the alignment and mechanics of the hallux [16].

The success rates for cheilectomy range from 72 to 100% for early-stage hallux rigidus with worse results for advanced joint degeneration in multiple retrospective case series [17–20]. In athletes, open cheilectomy offers 90% good and excellent results at a mean 5-year follow-up [2]. Two studies examining the results of arthroscopic cheilectomy found 67% good to excellent outcomes; however, these studies both had small sample sizes [21, 22]. In two different matched comparisons of percutaneous vs open cheilectomy, both groups demonstrated high patient satisfaction postoperatively [23, 24]. Loveday et al. showed a 94% satisfaction rate after percutaneous cheilectomy at mean 12-month follow-up with those patients who were dissatisfied were noted to have grade III degenerative changes [25].

23.3 MTP Arthroplasty

Joint replacement implants were originally designed to not only preserve the motion of the first MTP joint but to also relieve pain. Currently, there is little to no evidence of effectiveness and longevity for MTP implants in athletes. Due to the magnitude and direction of forces at the hallux MTP joint, it is likely that many athletic activities would put substantial stress on an MTP implant putting it at risk of early failure and potentially leading to further degenerative changes, deformity, and performance challenges.

However, for the nonathletic population, there are a number of implants available for end-stage hallux rigidus. These include metallic implants, interposition arthroplasty, and nonmetallic hemiarthroplasty (Fig. 23.5). Results of polyvinyl alcohol hemiarthroplasty (Cartiva) were shown in one series to be equivalent to MTP fusion for grade 2 hallux rigidus with greater maintenance of motion and significant improvement in FAAM scores [4]. This implant provides a smooth articular surface and a buffer space in the joint which can help reduce bone friction with dorsiflexion of the MTP joint. There is currently no data on compatibility of this technique with athletic activities. However, in the right patient, these options may prove to be useful tools to provide pain relief and maintenance of motion.

23.4 Arthrodesis

Arthrodesis involves fusion of the first MTP joint. Arthrodesis is usually considered for end-stage degenerative changes (grade III or IV) or after failure of joint-sparing procedures. It should

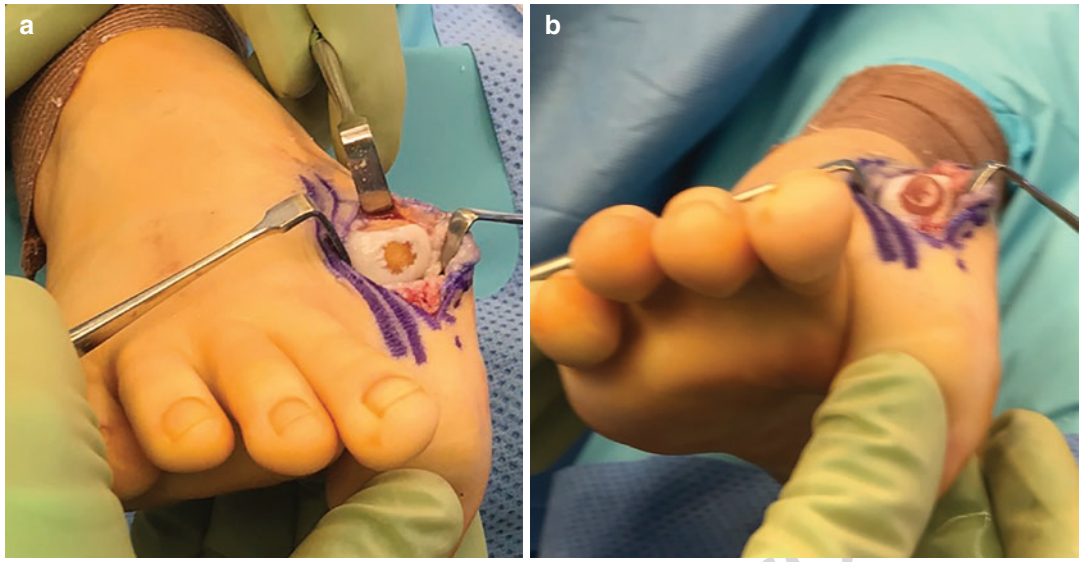

Fig. 23.5 (**a**) Intraoperative image demonstrating a large, central osteochondral defect of the first metatarsal articular surface. (**b**) Photograph of polyvinyl alcohol hemiarthroplasty applied to the defect

rarely be considered for first-line treatment of hallux rigidus in athletes. Multiple techniques exist to promote arthrodesis including lag screw with dorsal plate, oblique lag screw, staple, or crossed Kirschner wires. Furthermore, there are many different techniques for preparing the joint surfaces including simple cartilage excision and use of saw, cone, or socket for planar cartilage excision and finally conical reamer [26–28]. While the ultimate fixation method may be surgeon dependent, the ideal type of fixation should lead to high fusion rates, have low complication rates, and be reproducible. In a study by Politi et al., fixation with an oblique interfragmentary lag screw with a dorsal plate produced the most biomechanically stable construct to promote first MTP joint fusion and was nearly twice as strong as an oblique lag screw alone [29]. Arthroscopic fusion techniques have been described, but there is little available outcomes data to show short- or long-term superiority over open techniques [30]. Complications from this procedure include nonunion, malunion, infection, symptomatic hardware, and stress fracture of the metatarsal. Historical nonunion rates are as high as 30% in the literature [29]. However, more recent literature on later generation implants which provide both compression and rigid fixation demonstrates significantly lower nonunion rates.

23.5 Summary

Hallux rigidus is a common problem in athletes that causes pain and limited motion of the first MTP joint. Most athletes will note improvement or resolution of symptoms with conservative management. For those with persistent symptoms, the standard surgical technique includes an open debridement with cheilectomy. In patients with hallux rigidus interphalangeus, this technique can be successfully augmented with an osteotomy to correct alignment and joint mechanics. Arthroscopic and minimally invasive techniques are also growing in popularity. While first MTP joint arthrodesis provides predictable pain relief, joint fusion limits great toe dorsiflexion, which can impair athlete's ability to participate in running and jumping sports. Additional research is necessary to determine the long-term outcomes of some modern techniques and implants.

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Hallux Valgus for Athletes

24

Yasuhito Tanaka

24.1 Pathogenesis

Many athletes complain of painful hallux valgus. With respect to its pathogenesis, however, studies have found that lesion severity is not greater than that in ballet dancers who put their forefeet to very hard use compared with ordinary people [1, 2], and no evidence supports that hallux valgus occurs more frequently among athletes. In terms of the association between the onset of its symptoms and sports, although it is true that individuals with healthy feet who play sports do not develop hallux valgus, those who engage in intense sports who tend to develop hallux valgus may be more likely to exhibit symptoms, which may progress more rapidly [3]. Particularly, when the metatarsophalangeal (MTP) joint of the big toe is valgus, the medial collateral ligament becomes damaged and causes the onset of hallux valgus [4, 5] (Fig. 24.1). Excluding such cases of post-traumatic hallux valgus, the causes of hallux valgus in athletes are the same as those in nonathletes. That is, feet with metatarsus primus varus or "Egyptian feet" whose big toe is longer than the second toe are prone to develop hallux valgus. In flat feet, the medial longitudinal arch drops, causing the foot to pronate, and it tends to become valgus because the medial side of the big toe bears weight. Joint laxity may also be involved because hallux valgus occurs more commonly in women athletes.

Valgus of the big toe and varus of the first metatarsal are closely related [6]. The causal site of the first metatarsal varus is the first tarsometatarsal (TMT) joint, which is affected by the varus of the surface of the first TMT joint [7, 8]. The bony prominences of the medial side of the head of the first metatarsal are important as a pathology of hallux valgus, and large bony prominences may cause symptoms in athletes, even if the valgus of the big toe is only mild.

If valgus of the big toe progresses, the first MTP joint becomes subluxated, disturbing the balance of the muscles around the big toe [9]. The flexor hallucis brevis (medial head/lateral head), abductor hallucis, and adductor hallucis (transverse head/oblique head) muscles, which are all intrinsic muscles, all terminate at the first proximal phalanx via the plantar plate. The two sesamoid bones also lie within the plantar plate, and this structure is termed the "sesamoid complex." The big toe pronates, and the abductor hallucis is twisted around the underside, causing subluxation of the first MTP joint (Fig. 24.2).

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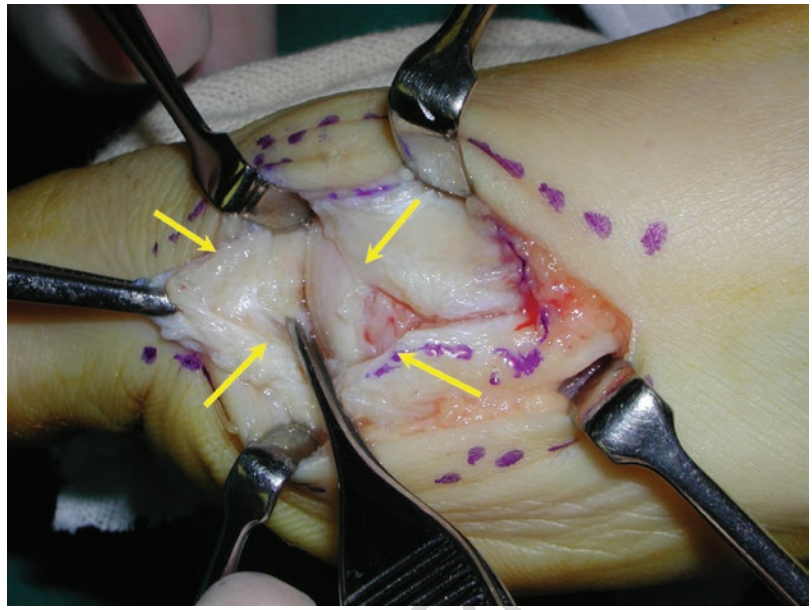

Fig. 24.1 Hallux valgus caused by the medial collateral ligament injury. Arrow: Torn portions of the medial collateral ligament of the first metatarsophalangeal joint

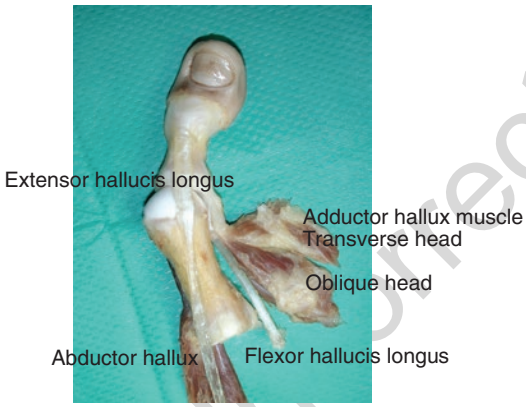

Fig. 24.2 Intrinsic and extrinsic muscles around the first metatarsophalangeal joint in hallux valgus

24.2 Diagnosis

24.2.1 Clinical Symptoms

Pain is the main symptom, and the most common manifestation is pain due to the medial protrusion of the head of the first metatarsal. In most athletes, it normally improves when the shoes are removed, but the intensity of their movements is such that skin erosion may form even if the deformity is mild. The skin turns red in the acute phase, and the patient still complains pain at rest in extreme cases. Bursitis-induced swelling may also be present. If the big toe pronates, the dorsal cutaneous nerve is twisted around to the medial side and compressed, causing pain. Weight bearing is also concentrated at the bottom of the medial side of the head of the metatarsal, resulting in callus formation. Many athletes without hallux valgus are already calloused in this area, and calluses are not a symptom even if hallux valgus is present in most cases. Rather, callouses are concentrated on the heads of the second and subsequent metatarsals because the deformity prevents full weight bearing on the big toe, causing painful calluses. Many patients with hallux valgus also have hammer toe in the second to fifth toes, causing midfoot pain and callus formation on the backs of the toes. Although severe hallux valgus presenting as crossing of the big toe over the second toe is rare in athletes, first ray dysfunction means that weight is placed on the lateral ray, which may cause stress fracture of the second or subsequent metatarsals or Morton's disease [10].

24.2.2 Radiographic Diagnosis

The basic method of evaluating a deformity is weight-bearing dorsoplantar foot radiography. Severity is assessed in terms of the hallux valgus angle (HV angle) formed between the axes of the first proximal phalanx and the first metatarsal (normally <15°). HV angle of <20°, between ≥20° and <40°, and ≥40° are considered mild, moderate, and severe, respectively. Some athletes may complain of pain if the angle is <15°. The intermetatarsal angle formed between the axes of the first and second metatarsals (M1M2 angle, normally <10°) is evaluated as an indicator of metatarsus primus varus. The congruence of the first MTP joint and arthritic changes are also scrutinized. Although joint congruence is preserved in the early stages, subluxation occurs if the condition progresses. In younger patients, joint congruence may be maintained despite severe hallux valgus deformity because the distal joint surface of the first metatarsal becomes valgus [11] (Fig. 24.3a). This must be considered when deciding on surgery, and the distal

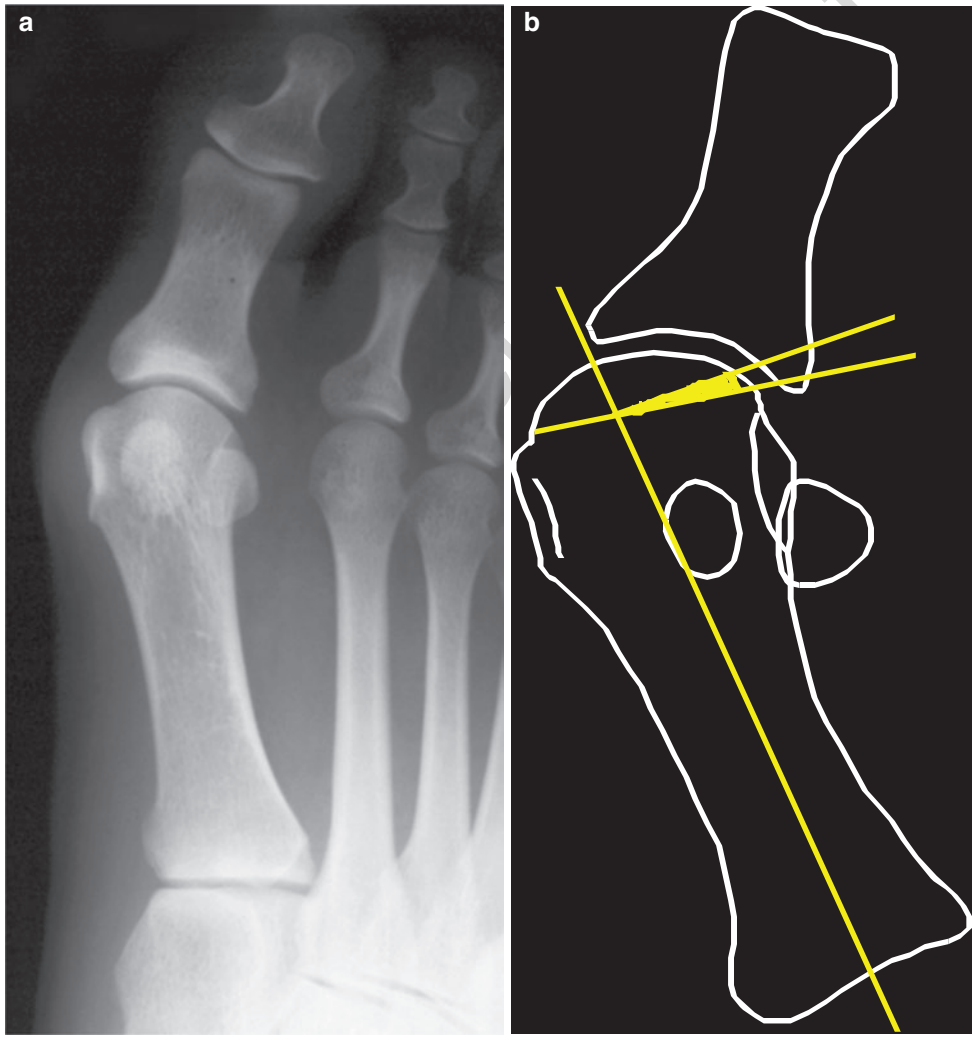

Fig. 24.3 Distal metatarsal articular angle (DMAA). (**a**) A hallux valgus foot with large DMAA. (**b**) The measuring method of DMAA which is an angle between a line perpendicular to the axis of the first metatarsal and the joint surface of the first metatarsal head

metatarsal articular angle (DMAA) between a line perpendicular to the axis of the first metatarsal and the joint surface of the first metatarsal head should be measured (Fig. 24.3b). Displacement of the sesamoid bones also indicates an abnormal course of the intrinsic muscles.

24.3 Treatment

24.3.1 Conservative Treatment

24.3.1.1 Shoes

The sports shoes normally used by the patient should be checked initially. Approximately 1-cm space should be present beyond the toes, and moving the toes freely while the shoes are on is important. In addition, the instep should be checked whether it is firmly held by the laces, preventing the foot from sliding forward within the shoe. If the shoes fit poorly, the patient should be advised to change them. If they severely compress the inside ball area, pushing out that part of the shoes from the inside before putting them on may help alleviate compression of the medial side of the first MTP joint. If pain occurs only when the sports shoes are worn, the shoes should be taken off whenever possible, and avoid wearing these for long periods.

24.3.1.2 Stretching and Therapeutic Exercise

Preserving joint flexibility by stretching is important. The midfoot is held down on both sides with one hand to correct the varus of the first metatarsal, and the other hand slowly pulls the big toe inwards while keeping it under traction. Plantar/dorsiflexion stretches are performed at the same time. Stretching the Achilles tendon and plantar fascia is also important to reduce the strain on the forefoot [10]. Patients should perform these stretches together with whole-body stretches when warming up. Improving toe function, including that of the big toes, is essential for alleviating midfoot pain. Towel gathering and other exercise therapy techniques are effective, and patients should also practice flexing the toes intentionally while walking. They should also be taught how to perform hallux valgus exercises, such as varus movements of the big toe, to correct the deformity and eliminate pain (Fig. 24.4) [12]. Initially, attempts to move this toe outward result in its plantar flexion, but with manual intervention and persistence, many patients with even advanced hallux valgus can move the toe in the varus direction. This technique should always be taught because it is useful for athletes, who are highly motivated.

24.3.1.3 Insoles, Orthoses, and Taping

Insoles are prescribed to correct the alignment of the feet or legs and distribute the concentration of pressure that causes pain. If there are calluses on the undersides of the heads of the second to the fourth metatarsals, the function of the big toe will be impaired, and the weight-bearing pattern during walking is often shifted to the lateral side. The aims of the prescription are to create a medial arch to correct the forefoot pronation and metatarsus primus varus and to form a midfoot pad to decompress the heads of the second to fourth metatarsals that cause midfoot pain. Boosting the abductor hallucis muscles also creates a force that acts to correct the hallux valgus deformity during weight bearing. Corrective orthoses that do not fit inside shoes cannot be used during sports activities; hence, soft orthoses worn between the toes should be used instead. Taping is readily accepted by athletes, and they should be taught simple taping methods for correcting hallux valgus.

24.3.1.4 Ultrasound-Guided Neurolysis

Many patients with hallux valgus develop a pseudoneuroma of the digital nerve on the dorsal side of the first MTP joint, for which ultrasound-guided neurolysis may be effective in some cases (Fig. 24.5). It may be worth attempting conservative treatment just once. Excessive dorsiflexion

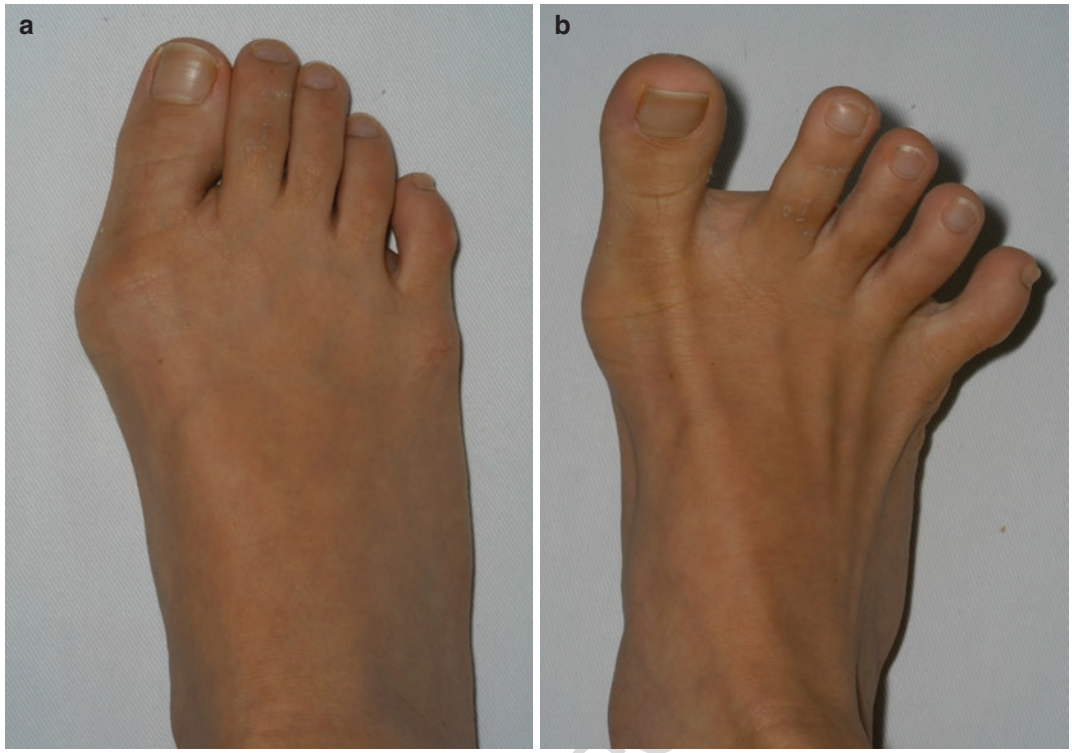

Fig. 24.4 Abductor hallucis exercise. (**a**) Close the big toe. (**b**) Open the big toe

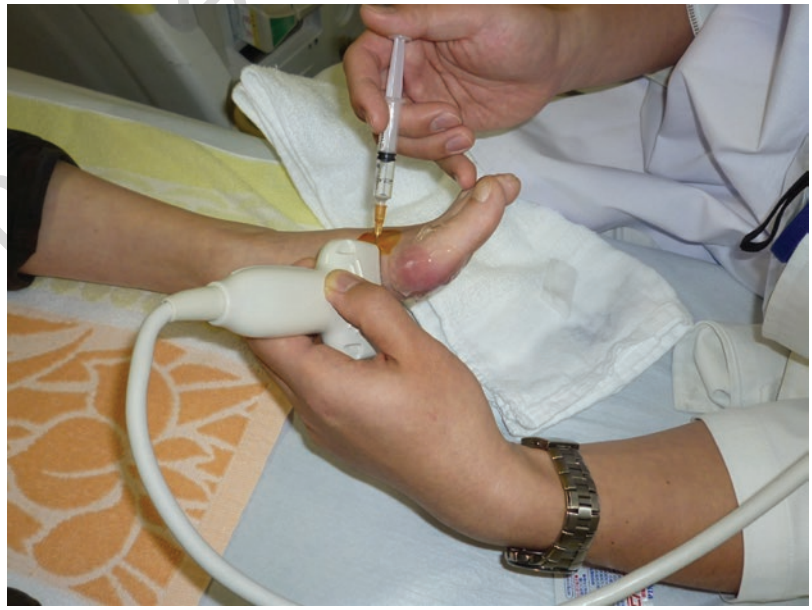

Fig. 24.5 Ultrasound-guided neurolysis

of the MTP joint is required particularly by dancers, and because hallux valgus surgery necessarily reduces the range of motion of the joint, in principle, conservative therapy is used if they are still dancing.

24.3.2 Surgical Treatment

In cases of post-traumatic hallux valgus, surgery is performed to repair the medial collateral ligament [5]. However, the decision to treat athletes surgically must be made with caution. In surgical procedures that require the release of soft tissue around the first MTP joint, changes in joint alignment may reduce the range of motion [12]. This can significantly reduce the competitiveness in disciplines that require excessive dorsiflexion of this joint, such as dancing and sprinting in athletics. Osteotomy of the first metatarsal is currently the most popular procedure, but for athletes, the correction should be performed while the deformity is still mild because soft tissue release is also required in severe cases [13]. Because good range of motion is maintained when distal osteotomy alone is performed using Chevron or Mitchell osteotomy, it is recommended if pain is affecting competitiveness (Fig. 24.6) [10, 13–15]. Patients in their late teens who have recently stopped growing recover particularly rapidly, and in these patients, this surgery is performed if the deformity is mild or moderate [13]. Restricted range of motion can be prevented by minimizing the damage to the soft tissue region. For active dancers, however, neurotomy (Fig. 24.7) is sometimes considered as a procedure because it does not diminish the range of motion of the first MTP joint. Curative surgery for severe cases in adult patients should only be recommended after they have retired from dancing [13, 16, 17]. One study has reported the treatment of such patients who nevertheless request surgery by double osteotomy comprising distal osteotomy and proximal phalangeal osteotomy without soft tissue release [18].

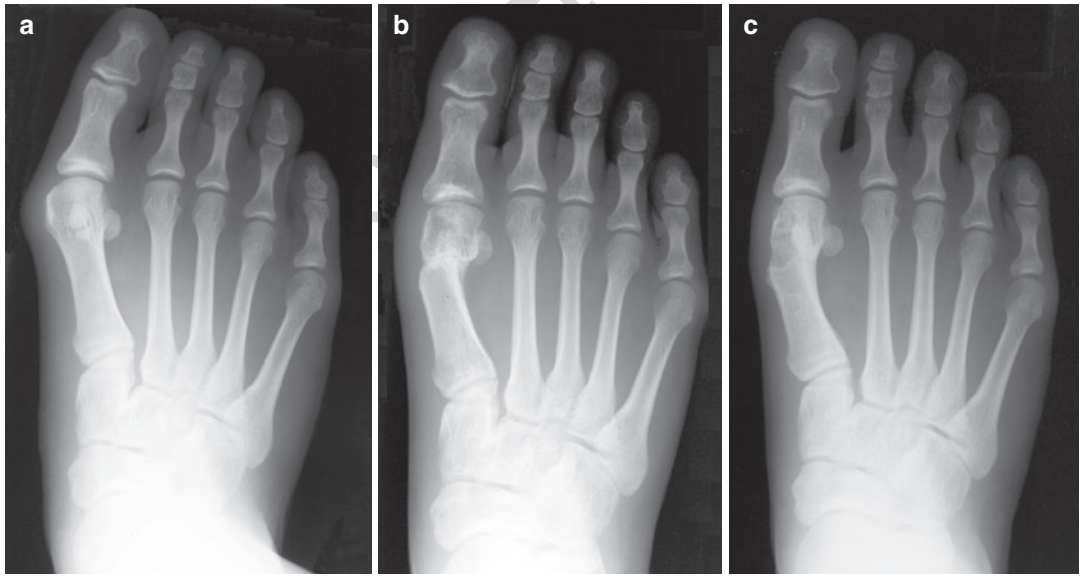

Fig. 24.6 Mitchell procedure. Eighteen-year-old female. Semiprofessional softball player. (**a**) Before operation. (**b**) Eleven weeks after the surgery. (**c**) Four years after the surgery. She could attend the national athletes meet 1 year after the surgery and continues playing softball 4 years after the surgery. Dorsiflexion of the first metatarsophalangeal joint is 70°

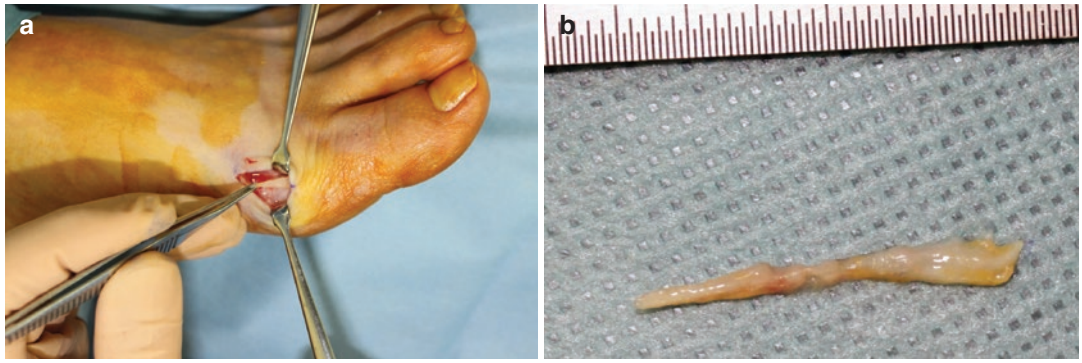

Fig. 24.7 Neurotomy of the medio-dorsal cutaneous nerve of the hallux. (**a**) A neurotomy is performed at the proximal side of pseudoneuroma. (**b**) A resected nerve

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25 Special Consideration and Perioperative Management for Turf Toe Injuries

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25.1 Introduction

Foot injuries are the third leading cause of foot injuries in NCAA athletes that have to be removed from competition, with a large number attributed to the hallux metatarsophalangeal (MTP) joint [1]. Turf toe injuries occur when there is a strain to the first MTP joint complex. An increasing rate of musculoskeletal injuries in athletes has been linked to athletic activity on artificial surfaces. Artificial turfs tend to be less shock absorbent resulting in greater force distributed throughout the body. Strain on the forefoot, as occurs in turf toe injuries, are common in athletes that participate in football or other contact sports. Prior surveys have revealed as many as 83% of turf toe injuries in professional football players occur on an artificial turf [1]. Athletes are 14 times more likely to sustain a turf toe injury during active competition compared to practice [2]. Biomechanical studies that investigate the impact of artificial surfaces suggest that greater torque and strain is applied to the forefoot compared to natural grass [3]. Turf toe injuries can be quite debilitating or even career ending. With less severe injury patterns, players who sustain the injury have a mean return to play of 10 days [2].

25.2 Anatomic Relationship

The first metatarsophalangeal (MTP) joint connects the midfoot to the forefoot of the great toe and is stabilized by several anatomical structures that make up the plantar complex. The flexor hallucis brevis (FHB) runs along the plantar surface of the metatarsal bone and inserts at the base of the proximal phalanx, crossing over the MTP joint. The FHB splits into the medial and lateral tendons that conjoin with the abductor and adductor hallucis, respectively (Fig. 25.1). The tibial and fibular sesamoids are two osseous structures that run within the sheath of the flexor hallucis tendons and are connected by the intersesamoid ligament (Fig. 25.2). The sesamoids share the load placed on the forefoot with full weight bearing. The medial head bears more of the weight bearing force, as it sits more directly under the metatarsal head. They also function similarly to the pulley system on the thumb and aid in mobility of the flexor tendons during planted dorsiflexion of the great toe. There is a thick, fibrous plantar structure that envelopes the sesamoids and makes up the capsular ligamentous complex. On the medial and lateral aspect of the MTP joint are collateral ligaments that ensure proper alignment from varus and valgus forces. These structures work in con-

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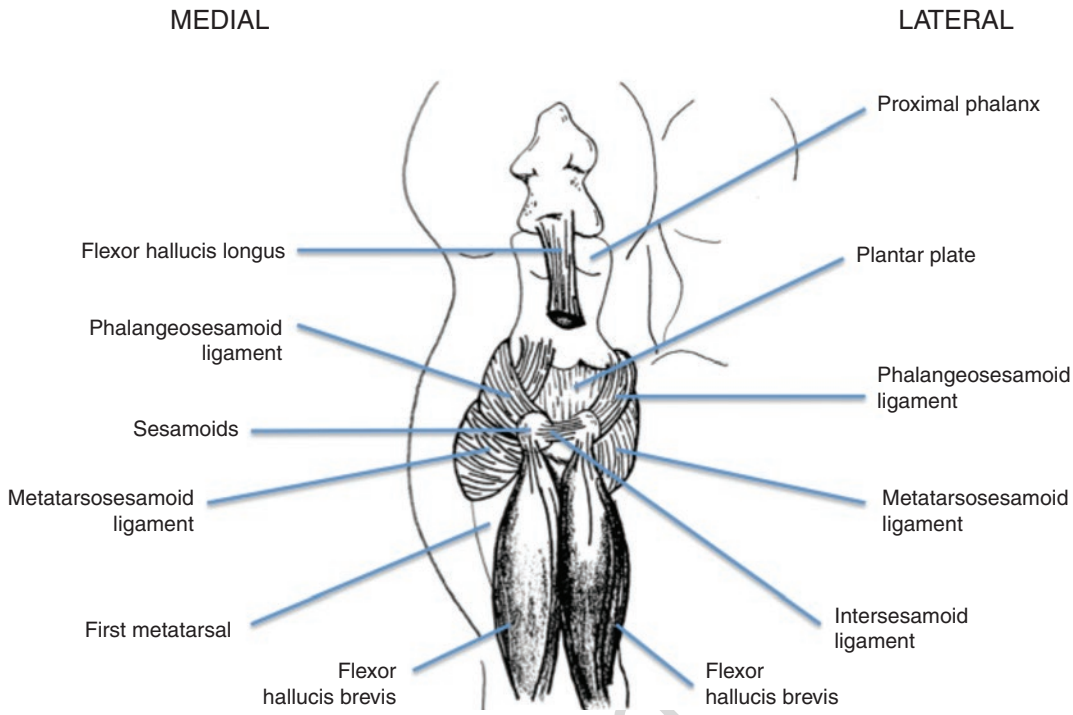

Fig. 25.1 A plantar view of the plantar plate ligament complex of the first MTP joint. *(Original artwork by Stephanie M. Jones, BA; Universityof Pittsburgh, Pittsburgh, PA)*

Fig. 25.2 A medial view of the plantar-sesamoidal complex. The flexor hallucis brevvis muscle runs along the plantrar surface of the 1st MT and the medial sesmoind lies just beneath the 1st MT head. *(Original artwork by Stephanie M. Jones, BA; University of Pittsburgh, Pittsburgh, PA)*

cert to provide the stability necessary in daily and high-level athletic activity.

25.3 Initial Evaluation

The initial evaluation should assess for risk factors related to turf toe injuries, such as type of athletic shoe or surface when the injury occurred. Detail should focus on the mechanism of injury, force applied, and the structures that are likely damaged. A keen assessment of specific signs and symptoms can help distinguish turf toe from other hallux injuries. Other injuries may include claw toe, mallet toe, MTP dislocation, fracture, and/or sesamoid injuries.

25.3.1 Mechanism of Injury

The plantar complex bears up to 60% of the body's normal weight and stabilizes the MTP joint during dorsiflexion [4]. The capsular MTP complex can sustain up to eight times body weight during jumping and running activities [5]. In turf toe injuries, one or more structures of the plantar complex of the first MTP joint are injured. Patients will often report an injury that involves planting the foot in fixed equinus with forced dorsiflexion of the first MTP. This typical mechanism of forward momentum during axial loading

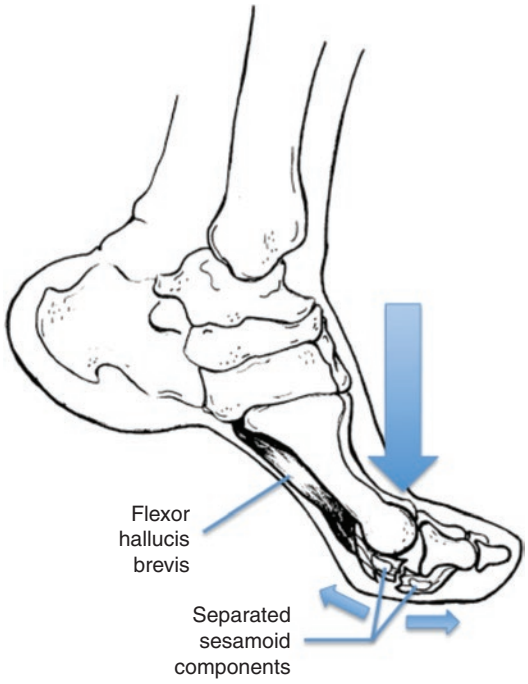

Fig. 25.3 Demonstrates mechanism that occurs in turf toe injuries with an axial load on the hyperextended foot *(Original artwork done by Stephanie M. Jones, BA; University of Pittsburgh, Pittsburgh, PA)*

causes forced hyperextension of the plantar plate and sesamoid complex of the first MTP joint (Fig. 25.3) [6]. Patients may complain of a stiff great toe that is swollen and tender with shoes or socks touching the toe. Patients with limited dorsiflexion may have concomitant tendinous injuries.

25.3.2 Physical Examination

Patients often have signs of ecchymosis or swelling of the MTP joint or on the plantar surface. Localized tenderness to palpation may be present on the plantar or medial surface. Malalignment of the hallux may also be present and should be visualized both on physical and radiographic examination. One should check for gross instability with a vertical Lachman (dorso-plantar drawer) test, as well as varus and valgus force to the MTP joint to assess the collateral ligaments. Both passive and active range of motion may be compromised based on the severity of injury and involvement of the flexor and/or extensor tendons. Additionally, patients should be assessed for integrity of the flexor hallucis longus (FHL) and the ability to dorsiflex the great toe. Patients may also have changes in gait evidenced by a quick return to heel strike during the gait cycle.

25.3.3 Diagnostic Evaluation

Patients with hyperextension injuries must undergo diagnostic evaluation with weight bearing anteroposterior, lateral, and oblique foot radiographs. An axial sesamoid and forced dorsiflexion view can also be obtained. Normal radiographs allow for comparison of the sesamoids relative to the joint, as well as proximal displacement of the medial sesamoid that normally sits directly under the metatarsal head. Patients who sustained significant axial force may have a sesamoid fracture.

Discrepant radiographic evaluation, relative to the clinical evaluation, should be further investigated with more advanced imaging. A bone scan reveals increased inflammation around the MTP joint and may signal a stress fracture of the proximal phalanx or the sesamoids. If a bone scan is positive, then pursing magnetic resonance imaging (MRI) would be appropriate. A MRI would also assess for a stress fracture of the proximal phalanx and would more clearly show a disruption or partial tear of the plantar plate complex (Fig. 25.4).

25.4 Preoperative Optimization and Risk Assessment

Injuries may vary in severity from a soft tissue damage to a dislocated MTP joint. Turf toe injuries have been classified based on the structural damage to the plantar plate complex. Injury classification helps to guide management and aids in determining prognosis (Table 25.1). Patients with Grade I and II injuries are often managed conservatively with RICE therapy and immobilization with a walking boot. Anti-inflammatory medica-

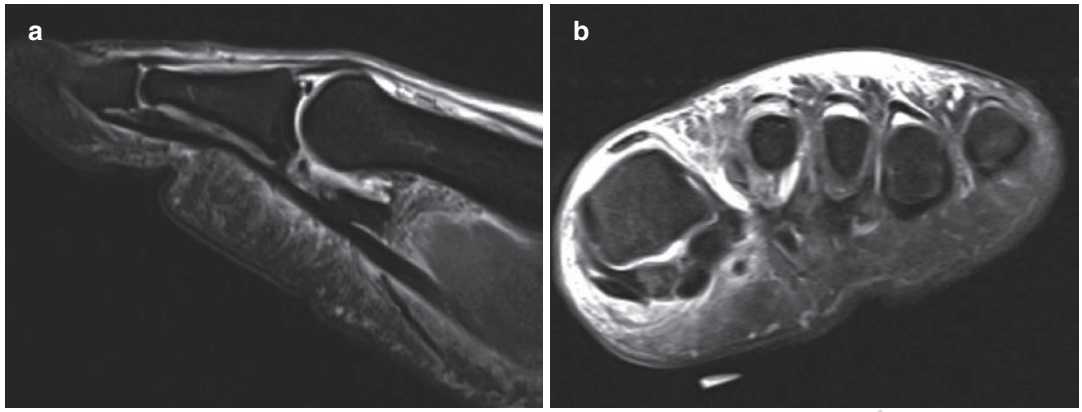

Fig. 25.4 Sagittal (**a**) and axial (**b**) MRI of the hallux MTP joint depicting high-grade partial thickness disruption of the plantar plate complex

Table 25.1 Management of Turf Toe Injuries by Classification

Grade	Short name	Description/symptoms	Management	Prognosis
I	Sprain	• Stretched plantar complex • Point tenderness • Minimal effusion	• RICE protocol • NSAIDS • Tapping	Return to play WBAT
II	Partial tear	• Widespread tenderness and bruising • Limited ROM due to pain and moderate effusion	• Walking boot • 3–14 day rest • Surgical repair if nonoperative trial fails	Return to play in 2 weeks ~10–14 days activity lost
III	Complete tear	• Severe swelling/bruising • Pain w/passive ROM • Difficultly weight bearing	• Cast • Surgical repair	Longer recovery (3–4 months post-op)

tion can also help reduce acute inflammation and help manage symptoms of pain and swelling. Athletes remain weight bearing as tolerated during this period. Gradual progression into low impact activities should be trialed prior to full return to play.

Patients who fail conservative management or have more severe injuries should be prepared for surgical intervention. In the young athlete, one should not forego proper assessment of comorbidities and risk for poor surgical outcomes. A thorough discussion regarding the patient's future aspirations for athletic activity should be explored to appropriately align expectations. Coaches, players, and family members can be a part of this discussion. However, medical decision should be guided based on what is in the best interest of the patient's physical, mental, and emotional best interest.

25.5 Surgical Techniques

Surgical intervention for turf toe injury is often undertaken after nonoperative management has failed for mild injuries or in the case of severe injuries. Severity is imparted by retraction of sesamoids, fracture of sesamoids with diastasis, hallux-valgus deformity, and intra-articular fracture fragments. These factors cause a relative discontinuity between the sesamoids and hallux MP joint during motion. Discontinuity of structures causes instability of the MTP joint [7]. Relative indications include loss of push off strength, progressive deformity, or clawing of toes [8]. Intrinsic minus position of toes is caused by MTP extension and interphalangeal joint flexion [8]. Competitive athletes in particular will often identify a significant loss of plantar restraint when attempting to push off during

play. Criteria for surgical intervention in the athletic population include sport of choice and primary position.

The goal of surgery is restoration of function via restoration of anatomy [9]. The plantar plate is advanced and reattached to the base of the proximal phalanx, thus enabling the sesamoid to move in conjunction with the hallux during dorsiflexion. Plantar soft tissue repair is performed end to end with nonabsorbable sutures if there is only a capsular defect [8]. If there is no residual soft tissue connection, it is recommended to trans-osseous bone anchors or bone tunnels [8].

In the case of mixed injury pattern that includes a valgus deformity and associated loss of push off, special attention must be paid to injury of the medially based structures. The hallmark of surgical intervention includes a relief of the deforming force via tenotomy of the adductor and repair of medial structures (including the abductor and joint capsule) with correction of resultant deformity via a modified McBride [9]. The patient is positioned supine with a lower extremity tourniquet and prepped and draped in the usual fashion. Intraoperative fluoroscopy is essential for dynamic evaluation of pre- and postoperative dorsiflexion of the great toe.

A medial plantar incision is most often used for the approach and may be carried out in an extra- or intra-articular fashion. Other options for surgical approach include dorsolateral or purely plantar approach [7]. Key tenants of intervention include repair or excision of sesamoid based on fracture pattern, repair of fracture, debridement of obvious osteochondral defects and repair or reconstruction of plantar plate.

25.5.1 Plantar Approach

The plantar approach is carried over the intermetatarsal space via a curvilinear incision on the border of the metatarsal fat pad or a "J" extending along the flexor crease at the base of the hallux [10]. The lateral plantar digital nerve courses over the lateral sesamoid. It is retracted medially with the metatarsal fat pad for protection. Sharp dissection of the tendons of the adductor hallucis and flexor hallucis brevis is used to expose the sesamoid. Once the sesamoid is exposed, a small rongeur or curette may be used for local debridement. The soft tissue is then examined including the flexor hallucis longus and plantar plate. Primary repairs are performed lateral to medial. Stability may be evaluated clinically or with intraoperative fluoroscopy. Further repair may be achieved with suture anchors or trans-osseous tunnels and headless screws to the base of the proximal phalanx [10]. The defect in the capsule overlying the sesamoid is then closed with 2-0 absorbable suture. Subcutaneous tissue is approximated with 3-0 absorbable suture. Skin may be closed with 3-0 nylon with simple interrupted stitches or vertical mattress stitches. Final immobilization may be in a standard AO posterior slab splint with side bars or a short-leg cast. Special attention is paid to ensure that there is a plantar plate built in to protect the toes.

Sutures are removed at the first postoperative clinic visit which is usually ~14 days postoperatively. Postoperative protocol emphasizes early passive range of motion to prevent contracture and limited motion of sesamoids. Patients are non-weight bearing immediately post-op and begin progressive weight bearing after first post-op appointment. Patients are transitioned from rigid immobilization to a hard-soled shoe prior to resuming preoperative footwear. The expected return to sport is usually around 3–4 months postoperatively.

25.6 Postoperative Course

After surgery, the great toe should be immobilized in 5–10° of plantarflexion with a toe spica splint. Patients are non-weight bearing immediately post-op and begin progressive weight bearing after first post-op appointment. However, postoperative protocol emphasizes early passive range of motion to prevent contracture and limited motion of sesamoids. Passive range of motion may begin 1 week after surgery. Range of motion exercises help to minimize the formation of arthrofibrosis at the sesamoid-metatarsal

articulation. Excessive dorsiflexion should be avoided in the postoperative period to protect the surgical reconstruction [11]. Patients should remain non-weight bearing for 4 weeks with a protective boot or removable splint. While sleeping, a removable bunion splint with a plantar restraint should be worn. After 4 weeks, protected weight bearing in a boot may be initiated [6]. Pool therapy may be initiated at this time. The progression weight bearing activity should be determined clinically based on the individual patient's level of pain and stiffness [11]. Around 8 weeks, the patient may discontinue wearing a protective boot and transition into a stiff-soled shoe. The patient may increase activity to medium-impact activities, such as elliptical training [8]. When able to comfortably perform medium-impact activities, the patient may proceed to high-impact activities such as jogging and running. Activities involving cutting and jumping should only be initiated once the patient is able to sprint without pain [11]. Most patients return to full activity in 16 weeks. However, it may take 6–12 months for full recovery [11].

25.7 Complications

During operative management of first MTP joint sprains, care should be taken to reduce the risk of infection and neurovascular damage, particularly as the plantarmedial digital nerve may be predisposed to injury during the surgical approach [10]. Hallux rigidus is a late sequela of turf to injuries. However, depending on the level of severity, hallux rigidus may require treatment with cheilectomy or arthrodesis. Turf toe injuries may also lead to progressive forefoot deformities such as hallux valgus, hallux varus, or "cock-up toe" due to hallux-interphalangeal joint flexion contracture [6].

25.8 Outcomes

Most turf toe injuries are mild and when diagnosed early can be managed nonoperatively. However, severe turf toe injuries have the potential to be career ending [12]. Patients with grade III turf toe injuries who have failed conservative management have been shown to have successful outcomes with operative intervention [13]. Multiple studies have reported on the respective operative and nonoperative outcomes of turf toe injuries. Anderson et al. performed a study on 19 athletes with severe turf toe injuries [14]. Of those athletes, nine required operative repair and no postoperative complications were observed. Additionally, only two athletes were unable return to full athletic activity. Coker et al. [15] and Clanton et al. [1] both report joint stiffness and pain as the most common long-term complications in their respective study groups. A more recent study by Brophy et al. evaluated previous turf toe injuries in professional American football players and reported increased hallux plantar pressure, as well as decreased passive metatarsal phalangeal dorsiflexion [16]. With regard to rehabilitation, Nihal et al. [12] report a 25–50% incidence of limited dorsiflexion and pain after 6 months of rehabilitation.

25.9 Conclusion

Turf toe injuries continue to plague athletes who participate in high-impact or contact sports. Athletes who have sustained a turf toe injury may experience a significant increase in missed days of competition or career ending sequela. Keen assessment and management of players with turf toe injuries likely has a major impact on prognosis. For patients with less severe injuries, return to play is often achieved without operative intervention. The hallmarks of operative interventions include sesamoid excision and/or fixation as well as tendon transfer when soft tissue repair is insufficient. However, if indicated, operative intervention may be necessary to restore pre-injury function. Surgical management for turf toe injuries often includes sesamoid excision, sesamoid fixation, and/or tendon transfer. The modified McBride technique is also often used to restore medial and lateral soft tissue balance. Futher investigation is necessary to identify both player-specific factors and environmental fac-

tors, such as hallux valgus deformity and athletic surface, that may predispose to turf toe injuries. Better follow-up is also needed to assess long-term functional outcomes and likelihood of reinjury following turf toe injuries in the athletic population.

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26 Ankle Arthroplasty

Jin Woo Lee and Kwang Hwan Park

26.1 Introduction

26.1.1 Total Ankle Arthroplasty (TAA)

Most orthopedic surgeons are well aware of the treatment options for end-stage osteoarthritis of the hip joint or knee joint since the protocols are well established. However, selecting the appropriate treatment option for ankle osteoarthritis is challenging from the initial stages for most orthopedic surgeons and ankle specialists. Primary (degenerative) osteoarthritis rarely arises in the ankle joint, as in the knee or hip joints; however, secondary osteoarthritis due to trauma is more common in the ankle [1]. Surgical treatment options for end-stage ankle osteoarthritis include total ankle arthroplasty and arthrodesis. Arthrodesis, which is recognized as the treatment of choice for end-stage ankle osteoarthritis to date, may cause excessive loading to nearby joints, and result in osteoarthritis, and may affect normal gait due to limited range of motion. Conversely, TAA enables near-normal gait since the range of motion is mostly preserved. Moreover, patients find it easier to walk on uneven surfaces and TAA reduces the load to nearby joints and prevents osteoarthritis [2]. Courville et al. [3] reported that TAA is more cost-effective than arthrodesis. During the past 30 years, TAA in its early days showed high failure rates due to inadequate implant design, defective surgical instruments, lack of adequate surgical techniques, unskilled cement use, and excessive bone cutting, which resulted in the loss of proper joint stability and normal joint mechanics [4]. Despite such disappointing results of TAA in its early days, dissatisfaction with arthrodesis, and promising results of arthroplasty in the knee and hip joints have enabled continued research for TAA. Following the development of second-generation implants, third-generation implants have been developed and are currently in clinical use. Recently developed implants, which have compensated the defects of conventional implants, show improved implant survival and postoperative clinical outcomes [5]. Results of TAA are promising as Pyevich et al. [6] recently reported a satisfactory rate of 93% in a 3- to 10-year follow-up study and Knecht et al. [2] reported an implant survival rate of 90%, 5 years after the procedure.

26.1.2 Brief History of Total Ankle Arthroplasty

TAA was introduced by Lord and Marotte [7] in 1970 using first-generation implants, consisting of polyethylene tibial and metal talar implants.

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The first-generation implants included unconstrained type models of Smith and Newton, and the constrained type models Mayo, Oregon, and TPR. Results of TAA using first-generation implants were generally poor. In a 3-year follow-up study using the Smith implant, Dini and Bassett [8] reported that satisfactory results were obtained only in 50% of patients with posttraumatic arthritis and in 40% of patients with rheumatoid arthritis. Kitaoka and Patzer [9] reported an implant failure rate of 36% in a 2-year follow-up study after using Mayo's implants. The reasons for the failure of the first-generation implants include the use of cement, over- or under-constraint, and lack of understanding of the soft tissue and ligament balancing, by means of surgical techniques. Constrained type implants resulted in excessive loosening since the stress was concentrated to the cement-bone junction, and unconstrained type implants showed high incidence of dislocations. In addition, the first-generation implants in general showed high rates of subsidence and osteolysis.

Based on the failures of the first-generation implants, second-generation implants, with several improvements, were developed. In the second-generation implants, the porous coating on its surface allowed fixation to be achieved by press fit instead of cement, and the durability of the polyethylene was improved. Implants were designed in a more anatomical and more biomechanical approach. The second-generation implants can be classified into 2 groups according to the number of components and by the bearing material used: the 2-component system consists of a fixed bearing and the 3-component system is equipped by a mobile bearing. Since the polyethylene bearing and tibial implant are adhered, the 2-component system with fixed bearing possesses higher constraint and conformity. This leads to lower dislocation rates, but the implant may be under higher shear force. However, the contact between the talar implant and the bearing remains low and may lead to lower constraint and higher polyethylene wear. The 3-component system with mobile bearing may lower shear force by maintaining balance between conformity and constraint. However, the surgical technique may be difficult and possesses a risk of dislocation of the bearing, and thus may result in higher rates of polyethylene wear between the tibial implant and the bearing. The 2-component prosthesis system with fixed bearing includes Agility, INBONE, Eclipse, SALTO Talaris, ESKA Rudigier, and TNK, while the 3-component system with mobile bearing includes HINTEGRA, STAR, Mobility, Buechel-Pappas, and Ramses.

The third-generation implants are non-cemented and are based on a design that emphasizes soft tissue balancing. The design minimizes bone cutting and most implants have adopted movable bearing systems [10]. Movable bearing systems are superior in terms of positional movements, owing to the second interface between the tibial area and the polyethylene insert. Minimal stress is applied to the ligaments of the ankle joint and wear rates of the polyethylene implants are improved [11]. Metals used for the implants include alloys such as cobalt-chromium and cobalt-chromium and titanium, with an additional porous coating with hydroxyapatite or titanium for implant fixation to the bone resection margin.

26.1.3 Total Ankle Arthroplasty vs. Ankle Arthrodesis

TAA and ankle arthrodesis (AA) are accepted surgical treatment options for end-stage ankle arthritis. Although arthrodesis has been considered the surgical standard for end-stage arthritis, it may result in functional limitations due to alterations in gait and loss of range of motion of the ankle [12, 13]. TAA provides restoration of ankle kinematics and more natural ankle function [14, 15]; however, it has the disadvantages of higher reoperation and complication rates [9, 16]. There is an ongoing debate concerning the superior surgical treatment for end-stage ankle arthritis; although, the available evidence is insufficient to conclude that one procedure is superior to the other [2, 3].

A systematic review of 13 level IV studies reported that the overall failure rate for TAA with

second- or third-generation implants was approximately 10% at 5 years with a wide range (0–32%) reported among different centers [17]. A literature review of the national registry data from Norway, Sweden, and New Zealand reported that the average revision rate was 21.8% at 5 years and 43.5% at 10 years after TAA with second- or third-generation implants [18]. A literature review of AA has described a nonunion rate ranging from 3% to 15% after AA [19]. In a recent level II study comparing these two procedures, the revision rate in the TAA group (17%) was approximately twofold higher than that in the AA group (7%) [6]. The higher revision rate after TAA seems to be due to the complexity of ankle replacement surgery and the unique biomechanics of the ankle joint [6]. The complication rate of TAA has been reported to be greater than that of AA, and the mean revision rate at the 5-year follow-up has been reported to be 11% for AA and 21% for TAA [6, 9, 16].

26.2 Patient Selection

26.2.1 Indications

TAA was developed to reduce pain and retain motion of the ankle joint in patients with ankle arthritis. With improvement of surgical outcomes, indications for TAA have increased recently. In general, TAA is indicated in patients with end-stage ankle arthritis who have sufficient bone stock available in the tibia and talus to support prosthesis. Unlike the hip and knee, ankle osteoarthritis mostly arises as a consequence of trauma [20, 21]. Optimal candidates for TAA include young, nonobese patients, nonsmokers, patients with low activity levels, patients with no ankle deformity, and ROM-preserved ankles.

Other common indications for TAA are systemic (rheumatoid) arthritis [12, 13, 19].

Secondary osteoarthritis due to pathologies, such as hemophilia [14, 15], gout [16, 22], postinfectious arthritis [6], and avascular necrosis [23], may be candidates for TAA, but due to various surgical outcomes, indications for surgery remain controversial. Patients with bilateral ankle osteoarthritis are good candidates for TAA because bilateral ankle fusion generally has a detrimental influence on gait and functional outcome [17, 18, 24].

Another indication for TAA is the salvage of painful nonunion or malunion of a prior ankle fusion [25, 26]. Conversion of fused ankle to TAA is a technically demanding procedure that should be performed only if remaining bone stock is sufficient and soft tissue conditions are not overly compromised [26].

26.2.2 Contraindications

Acute or chronic infections and Charcot neuroarthropathy are absolute contraindications for TAA [5, 27].

In patients with avascular necrosis of the talus, the use of a standard prosthesis component may lead to significant subsidence and loosening of the talar component and failure [28, 29]. Avascular necrosis of the talus is considered to be an absolute contraindication for TAA. Neuromuscular disorders, and poor-glucose control or diabetic polyneuropathy in diabetic patients are considered contraindications for TAA. Relative contraindications of TAA include patients with severe instability, or patients with significant varus or valgus deformity (>10°) [30, 31].

The relative contraindications for TAA also include severe osteoporosis, immunosuppressive therapy, and smoking [5]. Smoking is another relative contraindication because it is associated with higher risk of perioperative complications, including wound breakdown [32]. The negative effects of smoking have been studied relative to fusion, and fracture healing. It is well known that smokers have more difficulties with wound healing as compared with nonsmokers in TAA [33].

26.2.3 Preoperative Considerations

Age is an important factor in a patient's long-term outcome following TAA. The higher physical demands of younger patients may lead to

prosthesis failure. The ankle is more often affected by posttraumatic arthritis; these patients may already have some soft tissue injury from the previous trauma. The anterior soft tissue envelope of the ankle is relatively thin when compared with those of other joints where arthroplasty is performed. For these reasons, medical issues that may compromise healing need to be evaluated.

History of diabetes, smoking, inflammatory arthritis (RA), vascular disease, neuropathy, immunosuppression, neurologic disease, and osteoporosis must be verified before any procedure. Althoff et al. [34] found that age <65 years, low body mass index, obesity, diabetes, inflammatory arthritis, peripheral vascular disease, and hypothyroidism are strongly associated with an increased risk of postoperative infection after TAA. Whalen et al. [32] showed that there is a statistically significant increase in the incidence of wound breakdown in TAA patients with a long history of smoking.

Uncontrolled diabetes and vascular insufficiency are also known to have a deleterious effect on healing postoperative incisions around the foot and ankle. However, diabetic patients with good glycemic control without neuropathy can be treated by TAA.

Raikin et al. [35] demonstrated that rheumatoid arthritis is a leading risk factor for wound infection; patients with inflammatory arthritis are more likely to require additional treatment or surgery to manage wound complications than those without inflammatory arthritis. Immunosuppressive treatments are often indicated as rheumatoid arthritis (RA) increases the risk of wound dehiscence and postoperative infections.

Neurological disease can affect the survivorship of an implant as well as postoperative function. Varus or valgus malalignment of the ankle due to muscle spasticity can lead to edge loading and early failure of the implant.

In young patients, high demands for physical activity can cause edge loading and prosthesis wear that may lead to prosthesis failure [36, 37]. Running or excessive exercise should be restricted in these patients.

Ankle range of motion, muscle function (e.g., tibial and peroneus muscles), and ligament stability should also be assessed. The decreased dorsiflexion of ankle often makes Achilles tendon contracted and shortened. Posterior tibial tendon dysfunction with hindfoot valgus can lead to laxity of the medial ligament complex. Peroneus tendon dysfunction with hindfoot varus can lead to laxity of the lateral ligament complex. The latter must be verified before surgery to determine whether additional operations should be performed together with the TAA.

Osteoporotic patients may have poor bone quality and quantity in the distal tibia or talus to support the prosthesis. This can lead to poor bony ingrowth and instability of the implant. In particular, the tibial components lose fixation and subsidence occurs more often. To reduce the occurrence of these problems, larger alternatives can be used, but medial malleolar fractures may occur.

Weight-bearing radiographs should be reviewed for any coronal or sagittal plane malalignment to allow proper planning for correction interventions. It is critical to evaluate the alignment of the hip and knee as well. Neutral alignment is essential to maximize the prosthesis longevity. If malalignment is present, radiographs from the hip to the ankle may be required. Any signs of avascular necrosis (AVN) of the distal tibia or the talar body should be noted. Collapse and subsidence of the prosthesis may occur more commonly in patients with AVN, since bone ingrowth is deteriorated in such patients. Magnetic resonance imaging (MRI) is helpful in assessing the presence and severity of AVN [38].

26.3 Preoperative Planning

Detailed history taking and physical examination is necessary. Evaluation for limb alignment, gait, range of motion, and muscle function should also be conducted. Besides clinical examination, radiologic examination should also be performed.

26.3.1 Clinical Examination

Inspection of the foot and ankle in many positions (sitting, standing, and walking) should be performed, which allows the differential comparison of changes on weight-bearing vs. non-weight-bearing movements. Skin and soft tissues should be carefully evaluated, with special attention given to previous surgical scars. The eventually observed pathological findings should be compared with the unaffected limb. Ankle alignments are generally performed with the patient in standing position, while Hindfoot stability assessment is performed with the patient in the sitting position [39, 40]. Visual alignment assessment is often not sufficiently accurate, which means careful interpretation is needed for ankle and hindfoot assessment [41].

A goniometer is used to assess the range of motion of the tibiotalar joint and is positioned along the lateral border of the leg and foot. These measurements using the goniometer are performed in a weight-bearing position according to the method described by Lindsjo et al. [42] The Iowa Ankle Range of Motion is another useful option for the assessment of ankle dorsiflexion and stiffness [43]. Basic muscle function should be assessed routinely because lower leg muscle atrophy is common in end-stage osteoarthritis [44].

26.3.2 Radiologic Examination

Weight-bearing radiographs with an anteroposterior view of the ankle and anteroposterior and lateral views of the ankle are used on radiologic examination (Fig. 26.1). Weight-bearing is important in radiographs because non-weight-bearing radiographs are commonly misread [45, 46].

Any deformities or potential degenerative changes in the adjacent joints should be identified and carefully analyzed. Deformities may occur on any level in patients with osteoarthritic ankles**.** The standing position is appropriate for comparison of radiographs before and after the surgery. To measure the hindfoot deformities, the hindfoot alignment view is needed.

It is necessary to thoroughly evaluate malalignment and instability during preoperative planning. Both conditions can result in subluxation and edge loading of the UHMWPE insert, progressive deformity, and high early failure

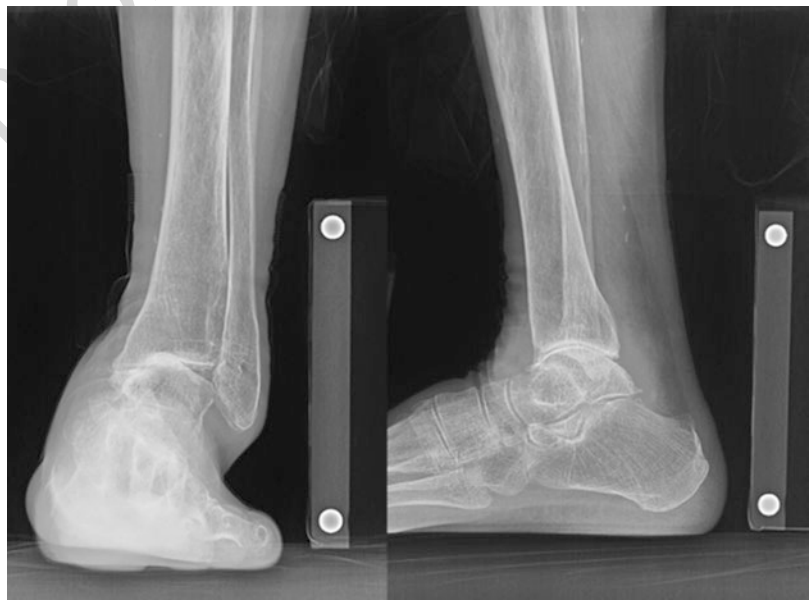

Fig. 26.1 A 84-year-old-women with osteoarthritis of left ankle. Preoperative standing AP and Lateral view shows the varus ankle

rates [47–50]. Most authors agree that correction of varus or valgus deformity may be limited, and a deformity more than 20 ° has been suggested as a contraindication for TAA [51].

In the frontal plane, the degree of alignment of the ankle is formed by the anatomic axis of the tibia and a line perpendicular to the articular surface of the dome of the talus on a standing anteroposterior radiograph [47, 52]. For angle alignments of less than 10 ° of varus or valgus, the joint is thought to be neutral, and is only considered to be varus or valgus when the alignment is above 10 ° [47]. The talar tilt angle is defined by the tibial and talar articular surfaces of the ankle joint on a standing anteroposterior radiograph. For talar tilt angles above 10 °, the joint is defined as incongruent [48]. Deformities can be located at the joint level (usually owing to anatomic joint line malalignment or to ankle degeneration) or proximally (usually due to a tibial fracture) [53]. If an abnormal alignment of more than 10 ° in any plane is present above the level of the ankle joint, corrective osteotomy must be undertaken at the site of the deformity before total ankle replacement [51]. If the deformity of the ankle is located at the joint level, an algorithmic approach to soft tissue balancing in varus ankles is recommended, including gradual release of the medial deltoid ligament, along with additional procedures [54].

A computed tomography (CT) scan can be used to assess joint mismatch or bone defects. In patients with degenerative changes of the adjacent joint, single photon emission computed tomography (SPECT-CT) might be used in the adjacent joint to analyze changes in form and biological activity [55, 56]. Preoperative MRI can be used to assess pathological changes of the tendon, avascular necrosis, and ligament injuries [57].

26.4 Prosthesis

26.4.1 Buechel-Pappas Prosthesis

The Buechel-Pappas prosthesis is rotationally unconstrained, mobile-bearing system

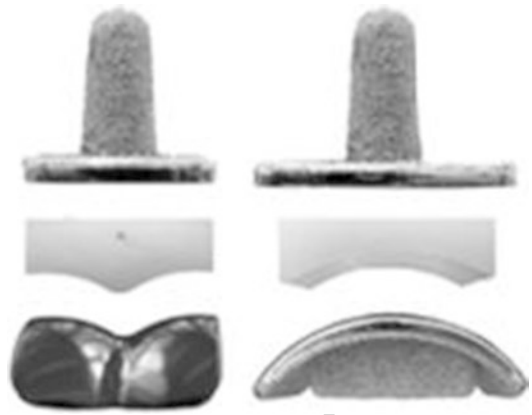

Fig. 26.2 Buechel-Pappas prosthesis

Fig. 26.3 Agility prosthesis

(Fig. 26.2). The prosthesis is composed of flat tibial plate, polyethylene inlay, and biconcave trochlear talar component. Deep trochlear sulcus angle prevent bearing subluxation. Buechel et al. [58] reported a survival rate of 92% after 12 years. Doets et al. reported a survival rate of 89% at the follow-up of 10 years [47]. Despite the long-term results, Buechel-Pappas prosthesis is currently not available.

26.4.2 Agility Ankle Prosthesis

The Agility prosthesis is a semi-constrained two-component design. The prosthesis is composed of titanium tibial and cobalt-chromium talar components (Fig. 26.3). Bone resection was minimized and the syndesmosis fused to increase the surface area for the tibial component and limit subsidence.

Fig. 26.4 Hintegra prosthesis

The Agility prosthesis was most commonly used in the United States, and has the longest follow-up [27, 59, 60]. However, high revision and reoperation rates were reported [61, 62]. As a result, the prosthesis has been replaced by other implants [63].

26.4.3 HINTEGRA Total Ankle Prosthesis

The HINTEGRA total ankle design is an unconstrained, three-component system that provides inversion-eversion mobility (Fig. 26.4). The tibial component has a flat, 4-mm-thick plate with 6 pyramidal peaks. The talar component is conically shaped, with a smaller radius on the medial side. It has 2.5-mm-high rims on each side that ensure stable positioning and guide the anteroposterior translation of the mobile bearing. The anterior shield of this component increases primary bone support [64].

Barg et al. [65] analyzed the survivorship of 722 ankle arthroplasty. The overall survival rates were 94% and 84% after 5 and 10 years. The midterm survivorship of the HINTEGRA implant was comparable with that of other third-generation total ankle replacements.

26.4.4 STAR (Scandinavian Total Ankle Replacement) Total Ankle Arthroplasty

The STAR prosthesis is a mobile-bearing prosthesis and has one of the longest histories in ankle replacement surgery (Fig. 26.5) [66]. The tibial and talar components are made of a cobalt–chromium alloy with coated surfaces allowing bone ingrowth.

Fig. 26.5 STAR prosthesis

Nunley et al. [67] evaluated 82 consecutive patients and reported that TAA with STAR prosthesis was associated with significant improvement in terms of pain, function, and quality of life.

Daniels et al. [68] reported that TAA with the STAR design led to good clinical outcomes at intermediate to long-term follow-up, but 29% of the ankles required polyethylene bearing exchange and/or metal component revision.

26.4.5 Salto Total Ankle Prosthesis

Salto prosthesis is mobile-bearing implant (Fig. 26.6). The tibial component has the flat and smooth surface toward the mobile bearing. It allows translation and rotation. The 3-mm medial rim protects the polyethylene from impingement with the medial malleolus. The tibial component has a fixation peg. The shape of talar component is similar to the natural talar anatomy. The anterior width is wider than the posterior, and the lateral flange has a larger curvature radius than the medial.

Wan et al. [69] reviewed 59 ankles operated by Salto prosthesis and reported that the short-term prosthesis survival was 94.9%. Hofmann et al. [70] reported that in a study of 81 ankles, the implant survival rate was 97.5% in the mean

Fig. 26.6 Salto prosthesis

follow-up of 5.2 years. Stewart et al. [71] found a survival rate of 95.8% in at least 5-year follow-up and significant improvements in the VAS and AOFAS score.

26.4.6 INBONE Total Ankle System

The tibial and talar components are made of cobalt–chromium with a titanium plasma spray coating. INBONE system has been changed in design to reduce component failure. The INBONE total ankle has a talar component with a central sulcus, providing additional coronal stability (Fig. 26.7).

In contrast to all other total ankle system, the INBONE total ankle system uses intramedullary referencing for placement of the tibial component and requires more fluoroscopy time than other prosthesis.

Fig. 26.7 INBONE prosthesis

26.4.7 Mobility Ankle System

The Mobility prosthesis is unconstrained three-component systems composed of cobalt-chromium porous coated tibial and talar components and a mobile-bearing polyethylene inlay (Fig. 26.8). The tibial component has a flat articular surface and a short, conical stem. The talar component is designed to leave the malleolar surface intact, and has a central longitudinal sulcus. The stability of the talar component is enhanced by two pegs on its non-articulating aspect. The non-articulating surfaces are porous coated to provide bone ingrowth [72]. The PE insert creates a conforming, congruent interface with a deep sulcus on the talar component, and has a flat surface on the tibial side to minimize shear stresses.

Muir et al. [73] performed 178 total TAA. They had satisfactory results over 85% of all patients at the average 4-year follow-up, but there is a significant incidence of persistent pain, particularly on the medial side, for which we were unable to establish a cause.

Fig. 26.8 Mobility prosthesis

26.4.8 TNK Total Ankle Prosthesis

TNK ankle is semi-constrained, two-component system (Fig. 26.9). It is made of alumina ceramic and its interface with bone is coated with alumina beads. This prosthesis combines biocompatibility of alumina ceramics with a design that facilitates fixation to bone.

The third-generation implants have made improvements in the high incidence of aseptic loosening of the first- and second-generation implants. Studies by the designer reported favorable results using the third-generation TNK prosthesis [74].

26.5 Surgical Techniques for Ligament Balancing and Malalignment

In most end-stage arthritic ankles, some degree of instability, deformity, contracture, or combination of these elements is present. Contracture

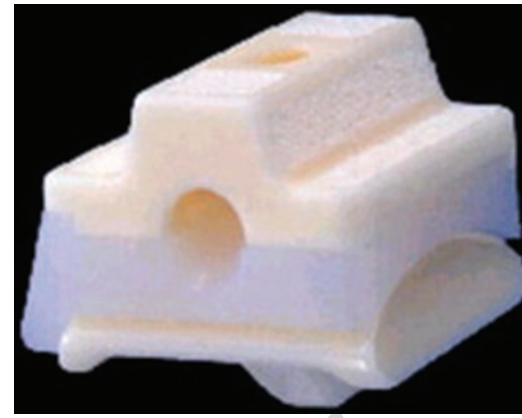

Fig. 26.9 TNK prosthesis

of soft tissues is a secondary transformation that generally arises as a consequence of trauma or long-standing angular malalignment. In general, medial-lateral soft tissue balancing requires release of contracted soft tissue on the concave side of the deformed ankle. Release of contracted medial soft tissue in varus ankles is, for the most part, quite different from the release of contracted lateral structures in valgus ankles.

Even if bone cuts can be made to establish anatomic alignment, proper soft tissue balance is required to maintain alignment throughout the range of motion. It is important, therefore, that surgeons be provided with a rationale and predictable techniques to perform soft tissue release, as well as indications regarding additional procedures commonly performed in balancing varus and valgus ankle in primary TAA.

26.5.1 Anterior Approach

Most implants are inserted using the anterior approach. A 10–15 cm incision is made at the anterior portion of the ankle joint, laterally to the tibialis anterior tendon and along the extensor hallucis longus (EHL) tendon. The superficial medial branch of the peroneal nerve is discovered

and retracted laterally. Next, the incision of the extensor retinaculum in line with the EHL tendon and the medial retraction of the EHL are performed. On applying this approach, attention should be taken not to damage the neurovascular structure, which is located behind the EHL. After the joint capsule is exposed, the longitudinal incision is applied. The incision must be sufficient to ensure the ankle joint is exposed. Following the identification of the lateral and medial gutters, the osteophytes at the tibia and the talar neck are excised. Implant stability must be secured by proper bone cutting, and soft tissue balancing. After the implantation, insertion of the drainage tube, followed by layered closure, is performed.

26.5.2 Operative Procedure for Varus Ankle

If the deformity of the ankle is located at the joint level, the algorithmic approach to soft tissue balancing in varus ankle is recommended (Fig. 26.10).

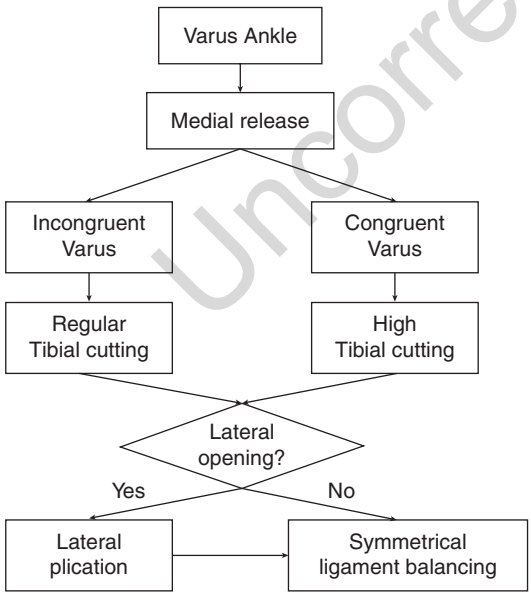

Fig. 26.10 Treatment algorithm in varus ankle osteoarthritis

26.5.2.1 Medial Release and Gap Balancing

Ligament balance is achieved by progressively releasing medial soft tissue until the length of the lateral ligamentous structures is reached. The extent of the release can be monitored by periodically inserting lamina spreaders or using a ligamentous tension meter to gauge alignment. Alternatively, trial components can be inserted, guiding the ankle through by applying varus and valgus stress to the ankle.

Following the surgical procedure and ankle joint exposure, removal of the periarticular osteophytes from the distal tibia and talus can be performed to effectively lengthen the medial capsuloligamentous tissue. Posterior osteophytes of the distal tibia should be carefully removed because they can lead to heterotopic ossification [75] or restrict the sagittal plane range of motion of the ankle. Next, soft tissue balancing corrects any talar tilt before proceeding to making cuts in bone. Gradual release of the deltoid ligament should be performed at its distal insertion using a curved osteotome (Fig. 26.11). It is important to release all components of the deep deltoid ligament, including the anterior tibiotalar, tibionavicular, and posterior tibiotalar ligaments. This gradual release technique was developed to alleviate the risk of

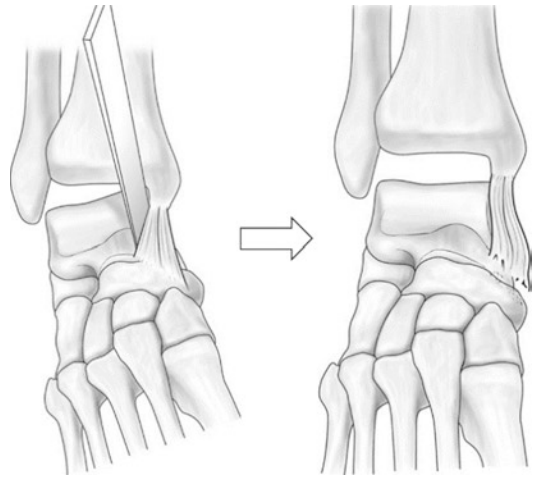

Fig. 26.11 Medial release by using an osteotome

medial ligamentous instability (or osteonecrosis of the talus) after extensive stripping from the talus, and to optimize ligamentous balancing. After bone preparation, trial components are then positioned and varus and valgus stress is applied to the ankle to assess balancing. The ankle is inspected for residual medial tightness or lateral gapping in a neutral position. In ankles with moderate to severe varus, the medial compartment of the ankle commonly remains tighter than the lateral compartment. A more definitive medial release should be performed at this time to balance the ankle. Once all the extra-articular deformities are noted, such as tightness in the posterior tibial tendon, patients may require a further incision to release the relevant contracture.

26.5.2.2 Lateral Plication-Peroneus Longus Transfer to Peroneus Brevis

Any residual imbalance in the supine position can result in subluxation or dislocation of the UHMWPE insert component on weight-bearing movements.

The modified Brostrom procedure [76] is preferred when the lateral ligamentous complex is spared. In patients with long-standing varus ankle arthrosis, however, varus deformity is commonly associated, to some extent, with chronic lateral ankle instability. Varus deformity is frequently associated with loss of the anterior talofibular ligament and calcaneofibular ligament, as well as anteriorly displaced talus. In such cases, several nonanatomic reconstruction procedures can be performed. Satisfactory results have been achieved with a peroneus longus tendon transfer to the base of the fifth metatarsal, [54] as described by Kilger et al. [77]. This procedure effectively enhances lateral ankle stability and weakens plantar flexion force over the first metatarsal. In addition, this procedure is easily combined with TAA.

To perform this procedure, a small, longitudinal incision is made over the base of the fifth metatarsal. The sural nerve and small saphenous vein courses follow posterior to the tendon and are subcutaneous at this level. The peroneus brevis insertion at the base of the fifth metatarsal is observed and the peroneus longus is identified adjacent to the peroneus brevis tendon. The peroneus longus tendon is transected at its most distal portion in full plantar flexion and eversion of the ankle is performed to allow sufficient harvesting of the tendon. A suture anchor can be used at the base of the fifth metatarsal, immediately plantar and lateral to the insertion of the peroneus brevis tendon. The peroneus longus tendon is sutured under moderate tension while the foot is held in a slightly plantarflexed and everted position. This provides sufficient eversion power postoperatively. A side-to-side tenodesis is then performed between the residual peroneus longus and brevis tendons. Degeneration or attritional rupture of the peroneal tendon is often present, which may be associated with an extended varus deformity of the ankle; if such is the case, all abnormal-appearing tendon should be debrided and tubularized.

26.5.2.3 Calcaneal Osteotomy

After the ligamentous imbalance has been managed, the alignment of the hindfoot should indicate whether calcaneal osteotomy should be performed. Frequently, varus deformity of the hindfoot is associated with varus ankle osteoarthrosis. Correcting the hindfoot deformity before or simultaneously with the TAA is essential to achieve optimal long-term results. Numerous calcaneal osteotomies have been reported with good clinical results, such as lateral displacement osteotomy, which translates the posterior fragment 5–10 mm laterally, and triplanar osteotomy [78], which corrects all 3 planes of the cavovarus deformity by lateral translation of the tuberosity fragment coupled with lateral closing of the wedge osteotomy to correct varus and proximal sliding of the tuberosity fragment and to adjust the calcaneal posture of the hindfoot and subtalar arthrodesis. The lateral closing wedge osteotomy was introduced by Dwyer [79]. It has commonly been used for the correction of the heel varus in combination with TAA, because it is technically easy and requires only a few additional minutes.

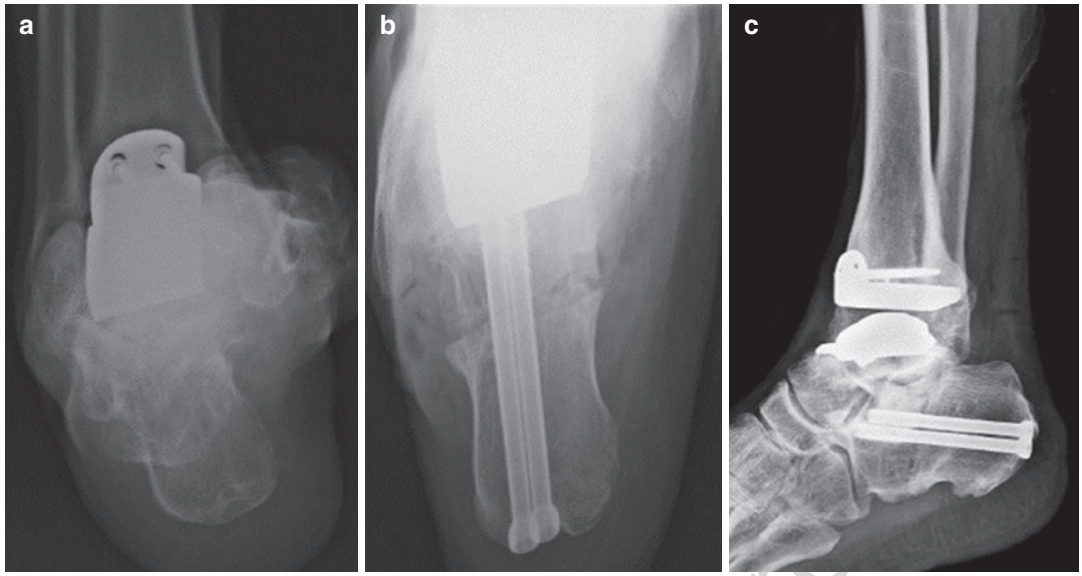

Fig. 26.12 Hindfoot varus after TAA was noticed. Calcaneal closing wedge osteotomy should be considered. Intraoperative heel varus (**a**), postoperative heel alignment view (**b**) and lateral view (**c**)

The procedure involves making a short, lateral, oblique incision directly posterior to the peroneus tendons, performing a lateral-based wedge osteotomy and tapering the wedge to, but not through, the medial cortex. After closing the gap, the correction of the varus deformity is ensured. While holding the osteotomy in the desired position, 2 guide pins are inserted to determine the correct position for the insertion of the cannulated cancellous screws. When the first pin engages the proximal fragment, a bone hook can help to pull the guide pin laterally to minimize the gap and to compress the bony surfaces together. Two 6.5-mm cancellous screws with partial threads are inserted perpendicular to the osteotomy site and positioned slightly posterior and lateral on the tuberosity segment, angled anteriorly and slightly medially (Fig. 26.12).

26.5.2.4 Dorsiflexion Osteotomy of the First Metatarsal

Once the ankle and hindfoot alignment is corrected, the surgeon should inspect the level of the metatarsal heads by holding the foot in a neutral position. Correction of the hindfoot and ankle varus can drive plantarflexion of the first ray. As the plantarflexed first ray forces the heel and ankle into varus [80], a dorsal closing wedge osteotomy should be performed on the first metatarsal by TAA. Through a small incision on the dorsum of the first metatarsal base, approximately 1 cm distal to the first tarsal-metatarsal joint, a dorsal-based wedge of the bone is removed using a sagittal saw. Cuts in the first metatarsal should be angled obliquely to allow for easier screw insertion. The most troublesome complication to date has been transfer metatarsalgia, attributable to an excess dorsiflexion of the distal fragment, resulting from too much bone resection. Once the osteotomy is created, it is important to preserve enough of the proximal fragment for screw placement by avoiding the region at the beginning of the osteotomy, which is too close to the first tarsal-metatarsal joint. After the metatarsal is elevated, 2 guide pins can be inserted from the proximal-dorsal to the plantar-distal aspect of the metatarsal. Two headless compression screws are inserted over the guide wires to engage both cortices for maximal compression (Fig. 26.13).

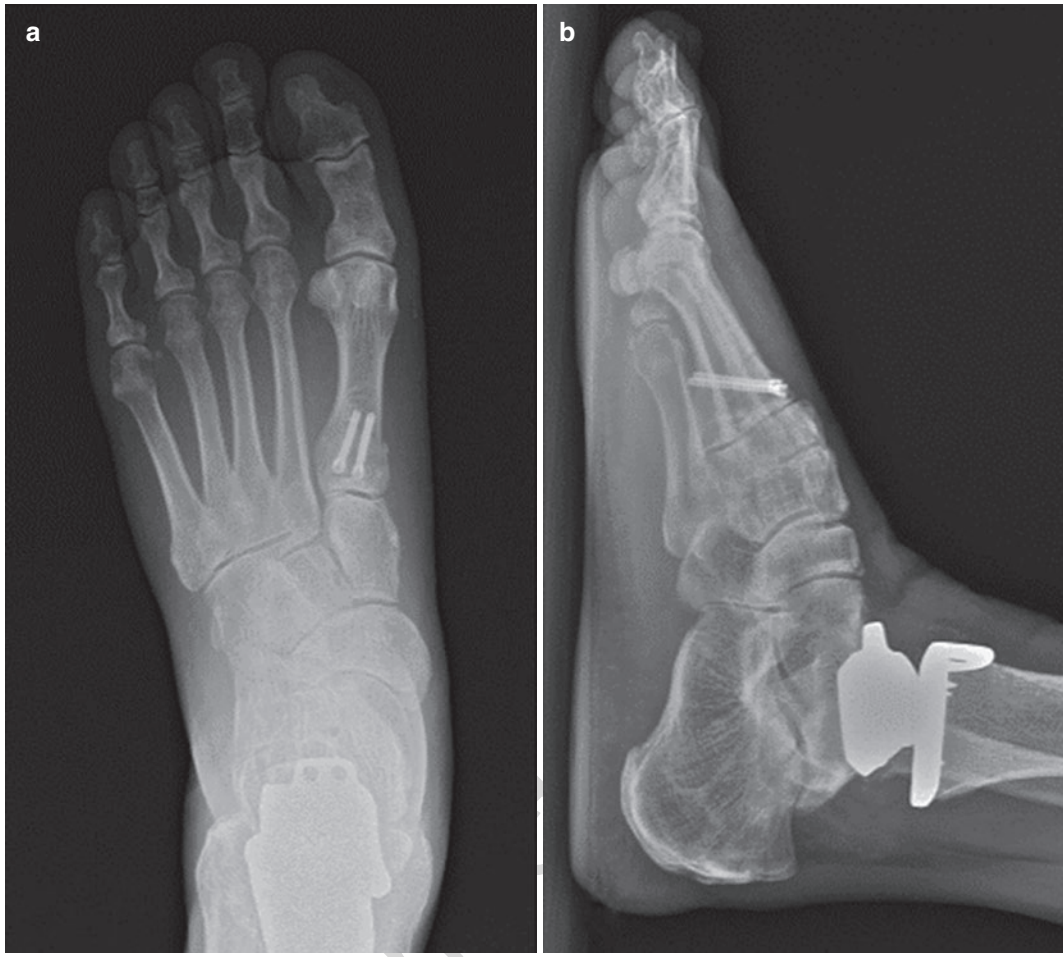

Fig. 26.13 Dorsiflexion osteotomy of the first metatarsal bone. Postoperative foot anteriorposterior view (**a**) and lateral view (**b**)

26.5.3 Operative Procedure for Valgus Ankle

Valgus ankle deformity is rare and often associated with malunion after ankle fractures and with posterior tibial tendon dysfunction. The most common scenario of malunion after ankle factures is the shortening and external rotation of the fibula, which can develop if the fixation of the fibula is inadequate [81]. To correct valgus ankle deformity, a transverse osteotomy is made above the level of the syndesmosis using a lateral transmalleolar approach. The syndesmosis should then be opened and pulled down using a bone reduction clamp to distract the lateral malleolus distally. An autologous iliac crest bone graft or structural allograft bone graft is interposed into the osteotomy site, whereas the distal segment is distracted. It is positioned firmly using a plate and screws. The amount of lengthening and the rotational correction of the fibula necessary can be difficult to determine. Comparison radiographs of the contralateral ankle or the articular contact between the fibula and the lateral edge of the talus may be helpful to determine the appropriate amount of correction to be performed.

The majority of valgus deformities occur secondarily to an advanced posterior tibial tendon dysfunction. The progressive deformity results in forefoot supination with medial column instability and eventually pes planovalgus. The foot deformity must be managed before addressing the ankle to obtain a stable plantigrade foot. Procedures to be performed to correct posterior tibial tendon dysfunction include medial displacement calcaneal osteotomy, lateral column lengthening, soft tissue procedures (e.g., flexor digitorum longus tendon transfer, repair of the deltoid and spring ligament), and/or plantarflexion osteotomy of the first ray. In patients with rigid fixed deformity of the hindfoot, multiple arthrodeses are considered the procedures of choice, including isolated subtalar arthrodesis, isolated talonavicular arthrodesis, talonavicular and calcaneocuboid arthrodesis, and triple arthrodesis [82]. These procedures can shift the heel contact point laterally to obtain a plantigrade, stable foot, and thus reduce stress on the lateral tibiotalar joint. The algorithm that is suggested for the treatment of a valgus ankle is shown in Fig. 26.14.

26.5.4 Additional Procedures

26.5.4.1 Heel Cord Lengthening

Patients with end-stage ankle arthrosis who undergo TAA often have a contracture of the gastrocnemius-soleus complex. Recognition of tight heel cord is also possible by observing the limitation of ankle dorsiflexion. If a minimal dorsiflexion of 10 ° cannot be achieved through TAA, heel cord lengthening can be considered as an option. It can be achieved by either gastrocnemius recession or percutaneous tendo-Achilles tendon lengthening, depending on the results of the Silfverskiold test.

Gastrocnemius recession Also known as the Strayer procedure, is a treatment option for patients who have heel cord tightness in which the chief cause of contracture is in the gastrocnemius alone. A posterior longitudinal incision is made over the middle of the calf at the level of the musculotendinous junction. After exposing the gastrocnemius aponeurosis, a transverse incision is made through it. The surgeon can control tension by dorsiflexing the ankle to the desired angle (>10 °). The paratenon and deep fascia are

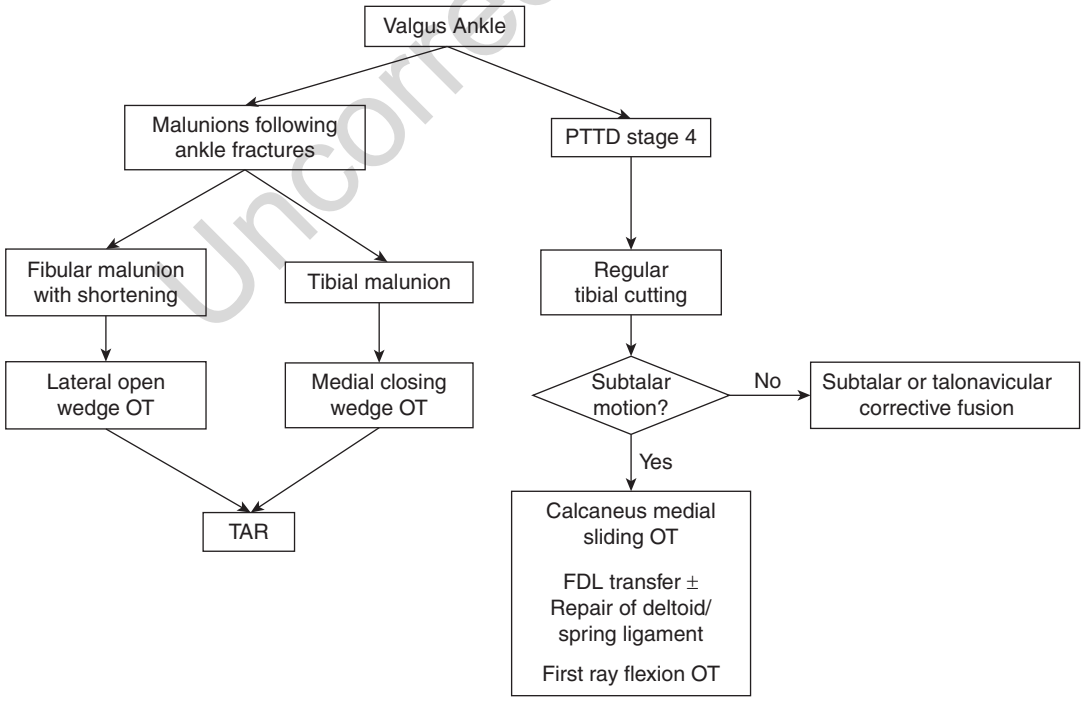

Fig. 26.14 Treatment algorithm in valgus ankle osteoarthritis. TAA must be followed by correction of PTTD deformity

then carefully repaired to prevent adhesion to the overlying skin.

Percutaneous lengthening of the Achilles tendon Tendo-Achilles tendon lengthening is indicated when both the gastrocnemius and soleus contribute to heel cord tightness through an open or percutaneous approach. Percutaneous teno-Achilles tendon lengthening is usually performed using the triple hemisection technique, described in detail by Hatt and Lamphier [83]. Percutaneous tendo-Achilles lengthening is preferable to open procedures because the former is quick and free of complications [84] and is easy to combine with TAA. Regardless of approach, particular attention should be taken to avoid complete rupture of the Achilles tendon that can occur during overzealous dorsiflexion of the ankle.

26.5.4.2 Hindfoot Fusion

Patients with end-stage arthrosis of the ankle joint frequently present with malalignment in the coronal plane, but also with degenerative change or deformity affecting the adjacent joints [51, 85, 86]. For these reasons, TAA occasionally requires adjunctive procedures to the hindfoot along with aforementioned procedures in order to obtain a plantigrade foot. Poor results for TAA have been reported in younger and patients with higher demanding movement requirements and hindfoot arthrodesis [87]. Performing various hindfoot fusions simultaneously with TAA or as a staged procedure before TAA, Kim et al. [88] recently reported good midterm outcomes in their attempt to address the challenges of hindfoot arthritis and deformity in TAA.

Subtalar fusion and/or talonavicular fusion are most frequently combined with TAA [72, 88], and, if necessary, these can also be performed with triple arthrodesis to create a plantigrade foot in TAA. The calcaneocuboid joint is usually spared if there is no evidence of arthrosis, because sparing of this joint can reduce nonunion [89, 90] and further adjacent joint arthritis [91, 92].

Hindfoot procedures can be performed either as simultaneous or staged operations; however, arthrodesis of the hindfoot combined with TAA would be too extensive a procedure for the patient's limb to tolerate in a single setting. Therefore, the patient's condition and the surgeon's skill should be considered when combining these procedures with TAA simultaneously.

26.6 Complications

26.6.1 Surgical Wound Problems

Along with medial malleolar fractures, postoperative complications related to surgical wounds are the most common complications after surgery, with an incidence rate of about 10% [93, 94]. Negative pressure wound therapy is effective for wounds with large dehiscence and preserved extension zone. When the wound is connected into the ankle joint, methods such as implant removal, free flap, or arthrodesis are necessary (Fig. 26.15).

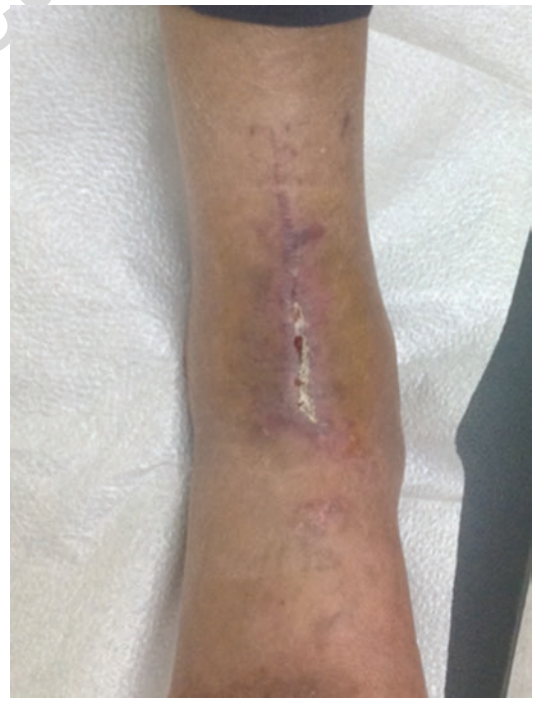

Fig. 26.15 Wound dehiscence along the incision after primary TAA

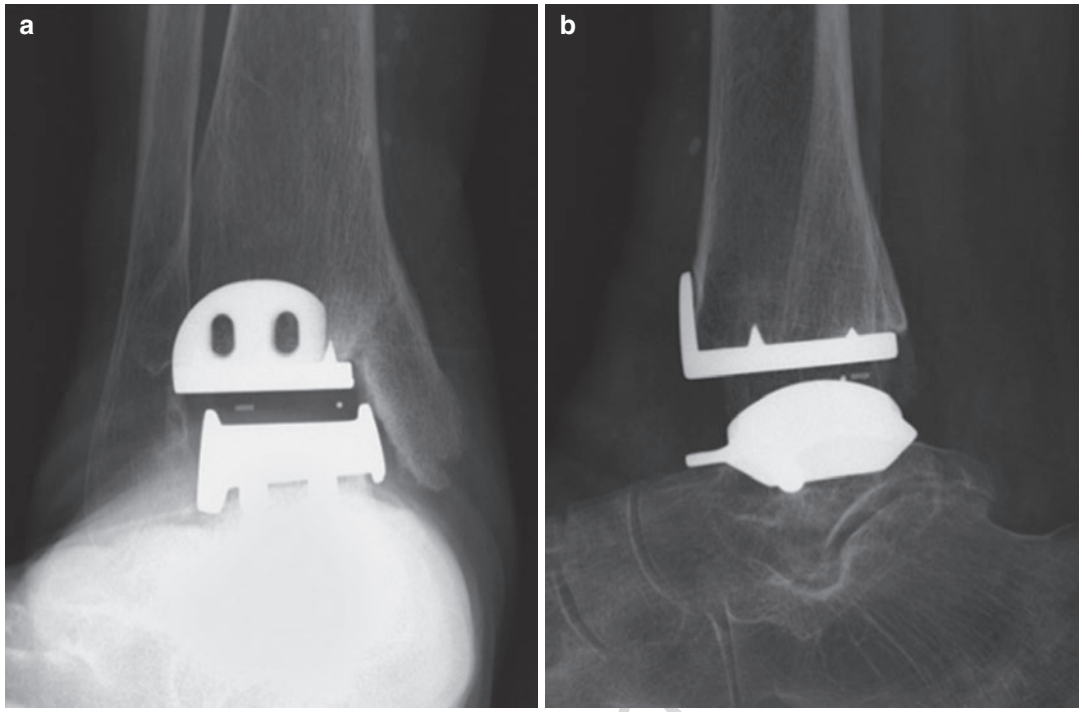

Fig. 26.16 Ankle AP (**a**) and Lateral (**b**) view at the follow-up of 5 months. Medial malleolar stress fracture was seen on radiographs

26.6.2 Medial Malleolar Fractures

Medial malleolar fractures occur in 20% of all cases [47, 93] and causes include careless saw use, excess traction of the medial malleolus, improper positioning, and size of the tibial implant. Since medial malleolar fractures may be discovered subsequently in postoperative plain films, attentive intraoperative observation is needed (Fig. 26.16).

26.6.3 Malalignment

Malalignment is reported in 4–45% of all cases [6, 47]. For prevention of malalignment, intraoperative confirmation of the alignment of the cutting guide in both coronal and sagittal planes are necessary. Malalignment that existed preoperatively must be corrected gradually or immediately during TAA using surgical techniques such as osteotomy or tendon transfer.

26.6.4 Postoperative Infection

Infection after TAA occurs in 0–2% of all cases, which is rare and is similar to that of total hip arthroplasty or total knee arthroplasty [6, 47, 94]. Adequate patient selection is crucial for prevention of infection. Thorough investigation for current infection is necessary in patients with a prior history of ankle joint infection or osteomyelitis. Assessment of vascular problems, skin disease, and long-term corticosteroid or immunodepressant use is needed, since such patients are at higher risk of postoperative infections after TAA. Adequate hemostasis throughout the surgery, handling the soft tissue with minimal damage can lower the risk of postoperative infections. Protocols for treating infection after TAA are similar to those of TKA or THA. Cellulitis or superficial infection may be easily controlled by irrigation, debridement, and antibiotic use if the intra-articular infection is not present and the wound is closed properly layer by layer. Acute

pyogenic infection requires irrigation, debridement, replacement of the polyethylene bearing, and antibiotic use. Subacute or chronic infection requires removal of the implant, antibiotic-eluting cement, antibiotic coverage, staged revision arthroplasty, or arthrodesis.

26.6.5 Subsidence and Migration

Subsidence is generally a result of deficient bone ingrowth or inadequate component support during weight-bearing activities. Strenuous exercise or being overweight may bear stress on the implant and may trigger subsidence. Severe destruction of the talus body or patients with rheumatoid arthritis has higher incidence rates [95]. Progressive subsidence is also associated with small-sized tibial implants, or >10 ° of preoperative deformity [6, 47]. However, since stabilization of the uncemented implant requires 6 months of time for stabilization, moderate implant subsidence and migration can occur during the early postoperative phases.

26.6.6 Aseptic Loosening and Osteolysis

While migration of implant is associated with early failure of implant stabilization, osteolysis is triggered by osteolytic reactions or bone cyst reactions caused by polyethylene wear particles. The primary cause of osteolysis is the malalignment of the implant and incongruent articular surface between the implant and the polyethylene bearing, which leads to edge loading. For radiological evaluation of the implant positioning, Hintermann et al. [96] have suggested the following classification: the α angle is the angle between the axis of the tibia and the tibial implant on AP film, the β angle is the angle between the axis of the tibia and the tibial implant on the lateral film, and the γ angle is the angle for the talar bone on the lateral film.

Hintermann et al. [96] defined loosening of the tibial implant as a difference in the α or β angle of over 2 °, or a radiolucent line of over 2 mm, and the loosening of the talar implant was defined as the difference in the γ angle of over 5 ° or a difference in the length of c or d of over 5 mm (Fig. 26.17). However, plain films can falsely minimize the degree and extent of osteolysis, true AP and lateral films are hard to obtain at each follow-up, and measuring the distance using Picture Archiving Communication System is inaccurate. Thus, the Hintermann's method possesses several limitations. Hanna et al. [97] insisted that CT scans can aid early detection of osteolysis, and can measure the extent of osteolysis in a more precise matter.

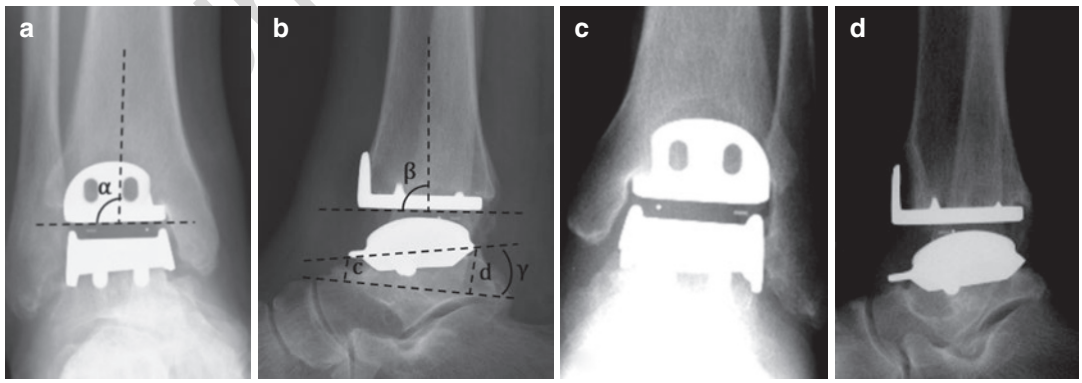

Fig. 26.17 The reference lines and angles on the AP view (**a**), and lateral view (**b**). Follow-up AP and lateral view shows the osteolysis around the tibial and talar components (**c**, **d**)

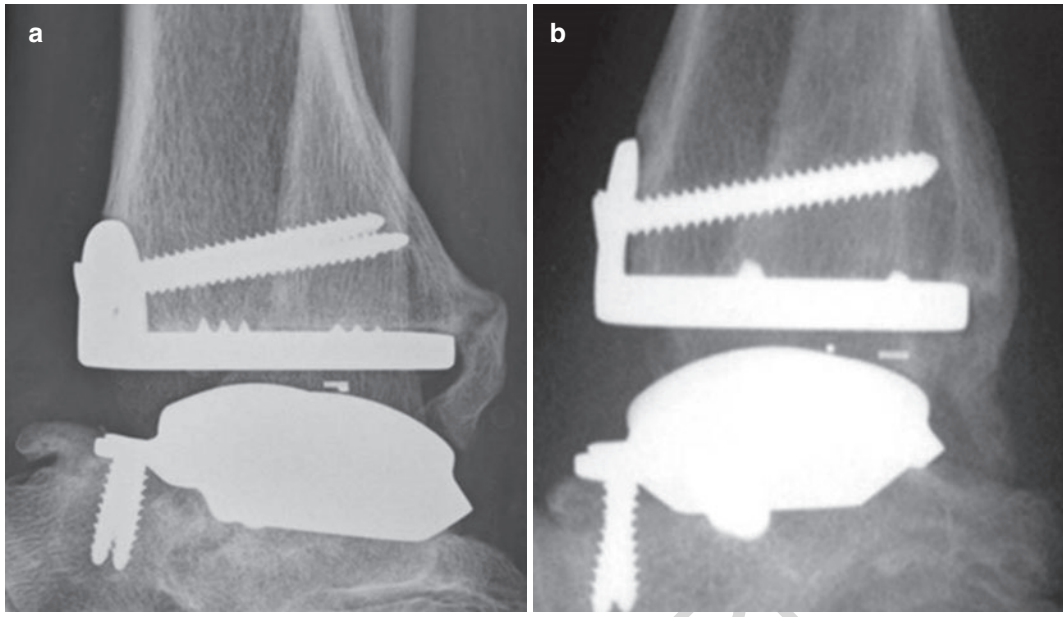

Fig. 26.18 Lateral view shows heterotrophic ossification which occurred after TAA. Postoperative ankle lateral view ((**a**), (**b**), different cases)

26.6.7 Impingement and Heterotrophic Ossification

Impingement and heterotrophic ossification after TAA is very frequently observed, and some studies have reported an incidence rate of collision of approximately 63% [87, 98]. Exposure of the cancellous bone at the resection margin may cause heterotrophic ossification (Fig. 26.18). In order to prevent such events, bone wax may be spread to the resection margin, or high-pressure washing may be performed. Selection of an adequately sized implant and excision of osteophytes may reduce the incidence of impingement after TAA. If impingement is present, the use of smaller-sized talar implants is recommend. Heterotrophic ossification after TAA is reported to occur in 7–64% of cases, and Bai et al. [99] have reported that heterotrophic ossification may be related to pain and contraction [93, 100].

26.6.8 Instability

Ligament balancing during TAA must be performed. Varus deformity after TAA requires release of the deltoid ligament, with additional modified Brostrom techniques, the modified Evans technique, autologous or allogenic ligament transfer using the peroneus brevis or hamstrings, depending on the degree of instability. Cases with valgus deformity may require reconstruction of the deltoid ligament. However, the surgical technique is difficult, and results have not been verified to date.

26.6.9 Adjacent Joint Arthritis

Pain may occur in nearby joints after TAA. If osteoarthritis of the hindfoot is thought to be the cause of pain, preoperative CT scans or injection of local anesthetics, concurrent or staged arthrodesis after TAA must be considered. Though the incidence rate of hindfoot arthritis in TAA is lower than in AA, it cannot be completely prevented. In a 9-year follow-up study in patients who underwent TAA using Agility implants, talonavicular arthritis occurred in 15% of cases, and subtalar arthritis occurred in 19% of cases [101].

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Part IV

Tendons and Biology

27 Biologics in the Foot and Ankle

Kimberly Allen, Enrique Feria-Arias, Christopher Kreulen, and Eric Giza

27.1 Epidemiology

Tendon injuries are common in sports and can be difficult to treat. Tendon injuries account for 30% of the $30 billion spent every year on musculoskeletal injuries in the USA alone [1, 2]. While observing U.S. Division I collegiate athletes participating in 37 sports, Raikin, Garras, and Krapchev found that foot and ankle injuries accounted for 27% of all musculoskeletal injuries [3]. Basketball, in particular, has a relatively high incidence of tendon injuries compared to other sports [4]. Lievers et al. examined the rate of foot and ankle injuries in collegiate American football and found that the rate of foot and ankle injuries was 15 per 10,000 athletic sessions [5]. In tendon healing, primary healing is prolonged and recurrence rates as high as 30% have been reported [6–8]. Given the high cost of foot and ankle injuries, it is important to understand the current challenges facing the treatment of tendon injuries, as well as identify optimal treatment strategies for rehabilitating athletes effectively.

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27.2 Tendon Biology

Tendon is connective tissue that connects muscle to bone. A tendon produces motion by transferring muscle contraction to the skeletal structure [9]. Tendons insert into bone via four transitional tissues of increasing modulus: (1) tendon, (2) uncalcified fibrocartilage, (3) calcified fibrocartilage, and (4) bone [2]. A tendon has a highly organized hierarchical structure. Fibrils are the small unit, and they are formed by triple helical collagen molecules [10]. Bundles of fibrils form fibers and are then organized together with tenocytes to form fascicles [2]. Fascicles are bundled together with endotenon in between and epitenon surrounding the bundles [11]. Endotenon and epitenon are cellular, loose connective tissues, but the endotenon also contains fibroblasts, which produce mostly type I collagen and are also responsible for the healing process [2]. Finally, surrounding the outer layer of epitenon is the paratenon which, together with the epitenon form the peritenon, is the most external sheath of the tendon [2, 10]. Tendons are comprised of 90% collagen, with collagen type I being the most abundant (Figs. 27.1 and 27.2) [10].

27.3 Tendon Healing

There are three main phases of tendon healing: (1) inflammation, (2) proliferation, and (3) remodeling [9]. These phases are distinct but can overlap

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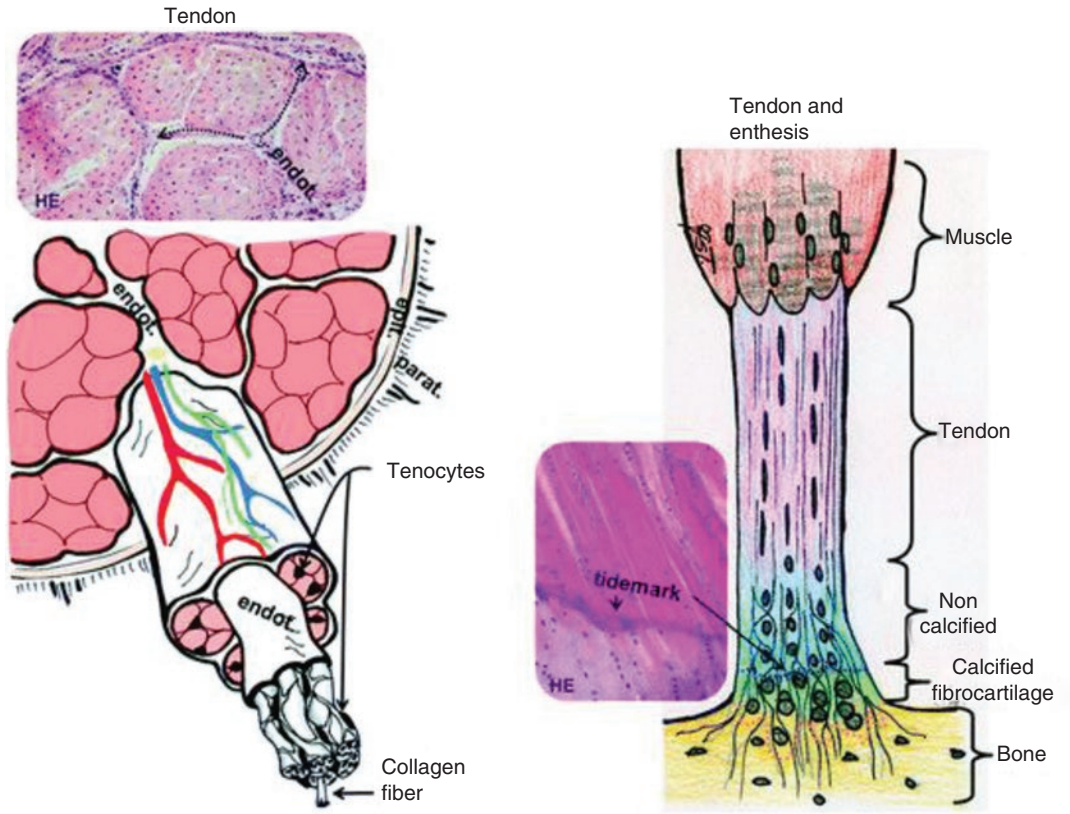
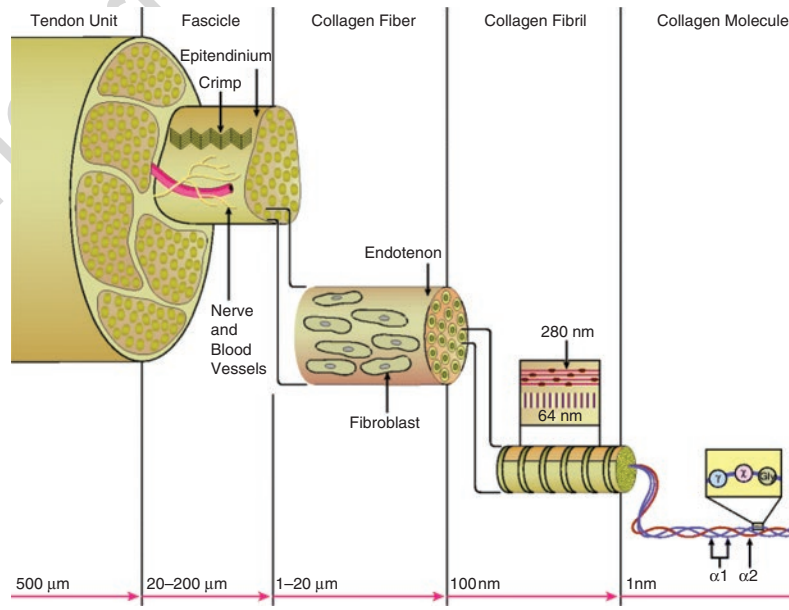


Fig. 27.1 Basic tendon morphology (*Drez and Delee Orthopaedic Sports Medicine Chapter 1 Basic Science and Injury of Muscle, Tendon, and Ligament*)

Fig. 27.2 Tendon insertion histology



with timing depending on the injury [9]. During the initial phase of inflammation, cytokines are released from the damaged site that attract extrinsic cells of the innate immune system (e.g., neutrophils, monocytes, and macrophages) [6]. These cells invade the injured tissue to clean up

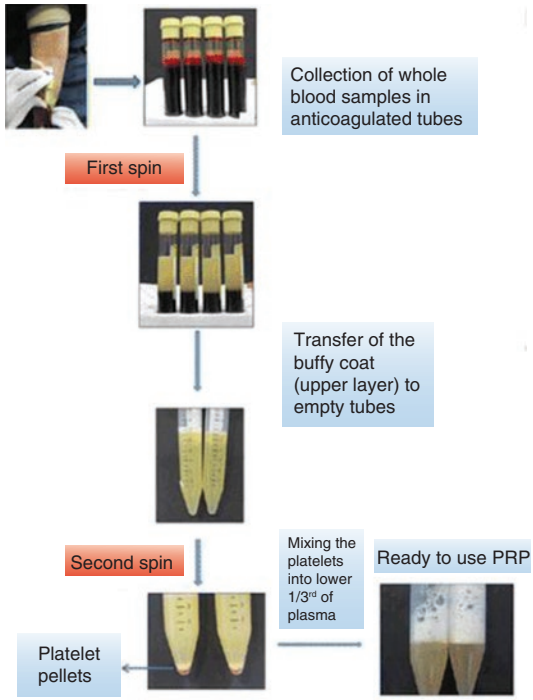


Fig. 27.3 Platelet-rich plasma preparation

the damaged area by engulfing cellular debris via phagocytosis [6]. Fibroblasts are also recruited to the injury site and begin to synthesize collagen type III and other components of the extracellular matrix (ECM) [9]. This fibroblast recruitment and production of collagen and ECM components is a key step in tendon healing [6, 11].

The proliferation stage then begins and can last several weeks [12]. It is characterized by continued type III collagen deposition by recruited fibroblasts, along with other ECM components, to form multicellular layers of epitenon cells to replace injured or torn tendon [10]. The fibroblasts also produce and secrete growth factors, such as TGFB, BFGF, IGF-1, and VEGF, that stimulate angiogenesis in the repair site [10]. The lineage of these fibroblasts and the various growth factors that work with them are key areas of research to understand tendon repair [6].

The third stage is the remodeling stage, which begins about 6–8 weeks AFTER injury [12]. During the remodeling phase, type III collagen is replaced by type I collagen and there is a decrease in cellularity and matrix production [13]. It is thought that there are two different mechanisms, extrinsic healing and intrinsic healing, that work together to proliferate tendon cells and ECM [9]. Extrinsic healing involves fibroblasts and inflammatory cells from peripheral tissue that infiltrate the injury site to stimulate repair and remodeling [13, 14]. The intrinsic healing, by contrast, pulls cells from the endotenon and epitenon of the tendon itself [13]. Collagen fibers then start to organize along the longitudinal axis of the tendon, which restores tendon stiffness and tensile strength [9, 14]. After approximately 10 weeks, the maturation stage starts, which includes an increase in collagen fibril crosslinking and the production of more mature tendinous tissue [9].

Research has focused on the different stages and mechanisms of tendon healing to determine the process for optimal regeneration and to augment biomechanical performance after injury. Growth factors, stem cells, and the potential sources of both, such as platelet-rich plasma, are discussed in this review, as well as the delivery technologies, scaffolds, and tissue engineering strategies comprised of multiple biologics.

27.4 Platelet-Rich Plasma

Platelet-rich plasma (PRP) is an approved biologic currently in use for tissue regeneration in bones, cartilages, ligaments, and tendons [9]. PRP injections deliver a concentrated amount of activated platelets capable of releasing growth factors to the damaged tendon site [7]. PRP made the sports news headlines in June of 2018 when it was released that NBA's first-round draft pick Lonzo Ball of the Los Angeles Lakers underwent PRP therapy for a left knee contusion that sidelined him for the final eight games of the 2017–2018 regular season [15]. However, many studies on the efficacy of PRP therapy for various tendon injuries have returned mixed results [7, 16]. Activated PRP releases growth factors almost immediately after being added to the tendon site and have a short half-life of only minutes to hours making the timing of injection critical [7]. It has been found that changing the balance of native biologics by using PRP too early in the tendon healing process may have a detrimental effect [7]. In contrast, adding platelet-derived growth factors on day 7 post-surgery in animals models show improved results in cellular maturation and tensile strength compared to application during surgery (Fig. 27.3) [17].

Meta-review of PRP experiments and clinical trials suggest it can effectively increase the rate of healing, but may not improve the end outcome results [8, 18]. Seijas at el. found that the use of PRP accelerated remodeling of tendon grafts used in anterior cruciate ligament repairs by 48% compared to the control group but did not improve the overall outcome after 1 year [18]. Another prospective controlled study found that the use of a PRP matrix that delivered a slow release of growth factors to posterosuperior rotator cuff tears decreased the re-tear rate to 14% compared to 50% in the control group at 13 months postop [19]. While the re-tear rate was improved, overall long-term function remained unchanged [19].

27.5 Growth Factors

Growth factors are small peptide signaling molecules controlling many aspects of tendon repair, including local recruitment of inflammatory and stem cells to the site of injury, cell proliferation and differentiation, and ECM synthesis [20]. Growth factors bind to cell surface receptors initiating intracellular signaling cascades that result in DNA transcription or regulation [12]. Tendon injury stimulates the production of a variety of growth factors including bFGF, BMPs, CTGF (connective tissue growth factor), IGF-1, PDGF, TGFβ1 -β2 -β3, and VEGF [9]. Growth factors are upregulated following injury and active during multiple stages of the healing process [20]. The effect of growth factors on tendon healing has been extensively studied in vitro using tenocyte and stem cell cultures, and in vivo with tendon injury animal models. Results are promising, but no human clinical studies investigating recombinant growth factors in tendon healing have occurred [9]. A deeper understanding of the synergies and antagonisms among growth factors and with other molecules, along with improved techniques for the temporal and spatial delivery of growth factor therapy are necessary for clinical application [20].

27.5.1 PDGF

PDGF (Platelet-derived Growth Factor) strongly influences healing immediately after injury by stimulating the synthesis of other growth factors, such as IGF-1 and TGF-B, and promoting general angiogenic, chemotactic, and mitogenic activity in the tissue [2]. PDGF persists for over 6 months at the site of injury and plays an important role in the remodeling stage through the synthesis of proteoglycans, collagen, noncollagenous protein, and DNA [21, 22]. Recombinant human PDGF promotes tendon repair in animal models [2]. Hildebrand et al. found that PDGF-BB (the highest affinity ligand) significantly increased the quality of healing when injected into the injury site of the MCL of rabbits on the basis of mechanical testing [23]. Tokunaga et al. found the use of a PDGF-BB impregnated hydrogel sheet in a rat rotator cuff injury model improved collagen fiber orientation, ultimate failure loads, stiffness, and stress to failure at 12 weeks relative to controls [24]. Clinical trials have been conducted on the efficacy of PDGF therapy and shown to improve the healing of periodontal osseous defects post-surgery [25].

27.5.2 TGF-β

Transforming growth factor beta (TGF-β1, β2, β3) is active in almost all stages of tendon healing [20]. Other members of the TGF superfamily, including bone morphogenetic proteins (BMPs) and growth differentiation factors (GDFs), have been studied extensively for their role in tendon healing [6, 9]. TGF-β1 is one of the main growth factors involved in tendon development and is responsible for lineage specific differentiation in most mesenchymal-derived cell-lines, including tenocytes [6, 12]. TGF-β1 is profuse in healing and scar formation and has been shown to improve tendon healing in animal models [26]. However, the positive effects of TGF-β1 are dose-dependent and supra-physiologic levels of

TGF-β1 in tendons are associated with adhesions, as well as fibrous and chondroid tissue deposition [6, 20].

Consistent with these activities, attenuation of TGF-β1 signaling by either antibodies or antisense oligonucleotides reduces scarring and adhesion formation in animal models during healing [27–29]. This is also supported by studies of TGF-β1 in the healing of fetal tendon tissue [6]. In the bovine model of scarless tendon healing it has been shown that TGF-β1 expression and inflammatory cell infiltration are significantly higher in adult healing tendons than in fetal tendons, while the fetal isoforms (TGF-β2 and -β3) contribute to regenerative healing without scar tissue formation [6]. Balancing the expression of the different TGF-β isoforms, by inhibiting TGF-β1 and exogenous administration of β2 and β3, holds promise as a strategy to promote regenerative tendon healing with less scar tissue formation [6, 12, 20].

27.5.3 IGF-1

Insulin-like growth factor-1 (IGF-1) is prominent in the early stages of tendon healing [9]. The primary effects of IGF-1 on tendon healing are mitogenesis, the stimulation of fibroblasts and tenocyte proliferation at the site of injury during inflammation, and collagen and ECM production during remodeling [20]. In rabbit flexor tendons, IGF-1 stimulated tenocyte proliferation with associated increases in collagen and proteoglycan synthesis [6]. In an equine superficial digital flexor tendinitis model, intralesional injections of IGF-I increased cell proliferation and collagen synthesis, reduced overall lesion size, and increased mechanical strength compared to control tendons [30]. There are no reports of IGF-1 application in human flexor tendon conditions, but clinical application of recombinant IGF-1 in flexor tendon disruption in racehorses has improved the rate of return to sustained athletic activity [12]. As one of the main components in the inflammatory cascade, it is thought that high concentrations of IGF-1 may act in a negative feedback loop to switch off early gene expression involved in inflammation [20].

27.5.4 bFGF

Basic fibroblast growth factor (bFGF) is one of the main growth factors involved in tendon development and upregulated in mature tenocytes, fibroblasts, and inflammatory cells in the early stages of healing [9]. Fukui et al. treated MCL injuries in rabbits with varying does of recombinant bFGF carried by a fibrin gel, and recorded early formation of repair tissue relative to controls [52]. Kobayashi et al. found similar results investigating bFGF-enhanced repair in canine ACL injuries [31]. Defects were introduced into the anteromedial bundle, a region with low healing potential, and treated with bFGF-impregnated pellets. The early stages of healing were positively influenced by bFGF, with improved neovascularization, histology, and orientation of collagen fibers relative to controls [31]. More research is necessary to characterize the therapeutic value of bFGF treatment in modulating tendon healing [6].

27.5.5 VEGF

Vascular endothelial growth factor (VEGF) promotes angiogenesis and is active in all stages of tendon healing [9]. VEGF is expressed in tendon sheet fibroblasts and VEGF mRNA peaks 10 days after surgery [6, 9]. Zhang et al. used VEGF injections in a murine model of Achilles tendinopathy and found significant increases in the tensile strength of healing tendons compared to control tendons [32]. Local injections of VEGF into the healing site of patellar tendons in rats increased load to failure [9]. VEGF increases TGF-β1 expression; whether benefit is derived from VEGF directly or from its secondary signaling activity is still undefined [6, 9].

The synergistic effect of growth factors in tendon healing has created interest in combination

therapies. However, the results of experiments involving more than one growth factor have been mixed [9, 20]. This highlights the need for more research on the expression patterns, concentrations, and kinetics necessary for optimal growth factor application [33]. Due to their limited half-life in vivo, the direct local delivery of growth factors has limited use, and more advanced strategies for sustained, safe, and reproducible delivery are necessary [2]. Much research has been done on the development of smart scaffolds, microcapsules, coated sutures, porous sutures, fibrin-heparin delivery systems, etc. that will provide controlled release of factors at suitable doses for an appropriate measure of time [2, 9]. Stem cells transduced with growth factors and direct modulation by way of gene therapy are other promising technologies being explored [2, 6]. While no consensus exists on the best methods for in vivo growth-factor therapy, further research will elucidate measures for controlled spatiotemporal release of the factors, improve long-term stability and expression patterns and ultimately optimize the healing outcome for tendon injuries [34].

27.6 Scaffolds for Delivery Method

Scaffolds are one technique for achieving tighter control over the delivery rate of growth factors as well as providing mechanical augmentation and structural support [35]. Scaffolds serve as space-filling substrate for new tissue to grow and can be seeded with stem cells or exogenous growth factors [33]. Scaffolds made from devitalized tissues retain numerous endogenous morphogens and have been available on the commercial market for years [33, 35]. Demineralized bone matrixes (DBM), first introduced almost 50 years ago, are an early example of scaffolding technology [33].

Scaffolds can be natural, synthetic, or a combination, each offering advantages and disadvantages [34]. Tissue remodeling does not typically occur extensively during natural tendon healing and could offer improved outcomes [36]. Natural scaffolds are made of decellularized tendon scaffolds (DTS) containing native tendon ECM [36]. These biologic ECM scaffolds have been found to show improved tendon repair. Healing closely resembles original tenocyte activity regarding collagen arrangement, growth factors, biocompatibility, and biomechanical characteristics [36]. One advantage of DTSs is their biodegradability in vivo, which combined with the production of new ECM by the host cells, can aid in tissue remodeling and complete repair [36].

Synthetic scaffolds offer stronger mechanical properties and more controlled growth factor delivery options compared to biologic scaffolds [34]. Wang at el. found synthetic scaffolds seeded with growth factors such as autologous or allogeneic fibroblasts, in rabbit Achilles tendons, improved overall strength, load bearing, and Young's modulus ratings [37]. Autologous-seeded scaffolds tested the closest to normal uninjured Achilles tendons in mechanical analysis relative to cell-free or allogeneic-seeded scaffolds at 7 and 13 months postop [37]. Cell-seeded scaffolds also help increase ECM production by directing the orientation of new cell growth along the direction of the fiber [34].

Further advancements such as "smart" artificial scaffolds and tissue engineering can custom deliver various growth factors and/or stem cells at a dynamic rate that responds to the surrounding environment [33, 34]. Scaffolds seeded with stem cells could deliver growth factors optimally to stimulate cell differentiation and maturation [35, 38]. The combination of mesenchymal stem cells (MSCs), specifically bone marrow derived mesenchymal stem cell (BMSC), used in conjunction with scaffolds has been largely studied evaluating potential improvement to the biomechanics of tendons post-injury [35]. Tissue engineering seeks to create biological tissue replacements using autologous cells that will avoid rejection [39]. Tissue engineering could offer an appealing delivery strategy; however, researchers have yet to pinpoint which specific biologics, in what exact amounts, and at what precise point along the healing timeline provides the optimal outcome [33].

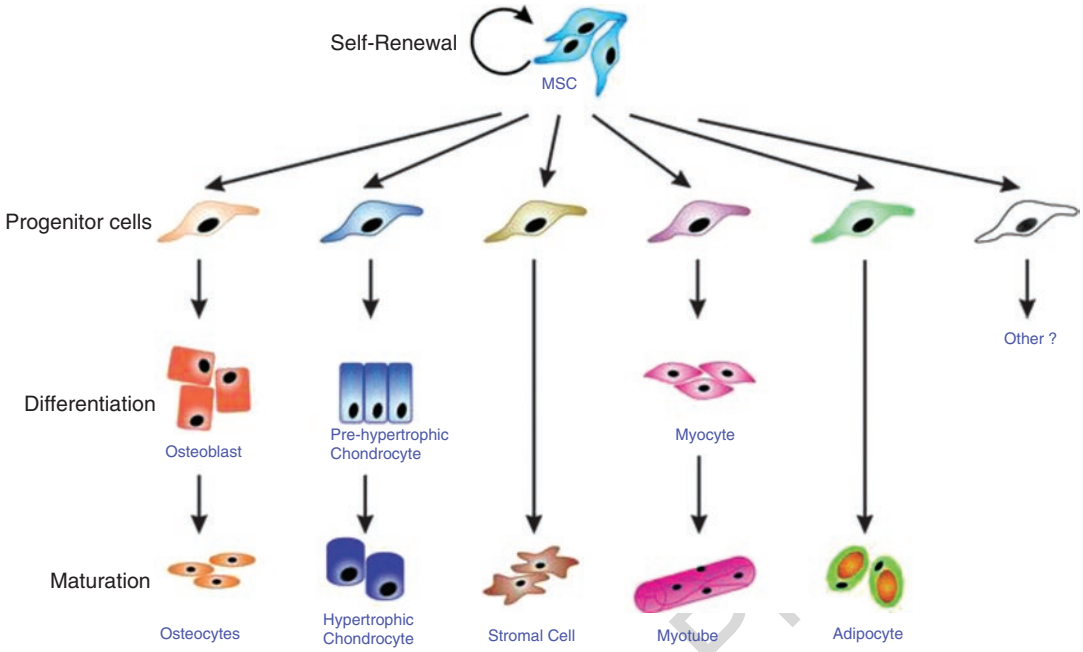


Fig. 27.4 Mesenchymal stem cell differentiation (*Human Bone Marrow Mesenchymal Stem Cells: A Systematic Reappraisal* Via *the Genostem Experience*)

27.7 Mesenchymal Stem Cells

Mesenchymal stem cells (MSCs) are multipotent cells that have the potential to become fibroblasts, tenocytes, chondrocytes, osteoblasts, myocytes, and adipocytes as well as generate multiple growth factors [35]. MSCs also have paracrine functions favorable to angiogenesis and improved healing [40, 41]. In early research, MSCs were thought to be beneficial because of their multipotent ability; however, more recently it has been found that the paracrine functions that stimulate and support the regenerative state of the healing tissue is the dominant beneficial characteristic of MSCs for tendon healing [40]. Research suggests that the healing potential of MSCs could be dose-dependent [40]. Chamberlain at el. used rat medial collateral ligaments and found that with low dosage (1×10^6), compared to a higher dosage (4×10^6), MSCs exhibited less of an inflammatory response driven by M1 macrophages and their inflammatory-inducing cytokines [40]. Other research suggest that the healing benefits are time and duration dependent. Working with a rat tissue model, Kraus et al. found MSCs improved tendon repair in regard to load bearing and stiffness when used during the first 14 days of early healing stages relative to the later 14–28 day period [42]. Later-stage MSC therapy may have contributed to detrimental results compared to the control (Fig. 27.4) [42].

Multipotent MSCs can be chemically and physically directed to selectively differentiate into tenocytes [43, 44]. Hoffman et al. found that by transfecting Smad8, a signaling-mediating agent of bone morphogenetic protein (BMP), MSCs differentiated favorably into tenocytes while inhibiting the osteogenesis pathway [44]. Scleraxis (Scx) is a transcription factor in the basic helix-loop-helix family (bHLH) that controls embryonic tendon formation [43, 45]. Studies show that MSCs expressing the transcription factor scleraxis causes MSCs to preferentially differentiate into tenocytes over other lineages [43, 45]. The addition of Scx to MSCs increases the expression of target genes by binding to specific promoters

during transcription that directs MSCs to tenocyte differentiation [43, 45]. This guided increase of tenocyte expression improved cellular organization and maturation of the injured tendon compared to the use of MSCs alone [43, 45].

The use of viral vectors to deliver engineered growth factors into stem cells has also showed favorable results. Gene-altered growth factors that have been transfected into stem cells using viral vectors, notably adenovirus and adeno-associated virus (AAV), showed improved tendon repair compared to naïve stem cells [12]. Specifically, MSCs that were transfected with VEGF using AAV showed an increase of the beneficial anabolic growth factor TGF-β [12]. Another study evaluating equine tendonitis showed that MSCs transfected with IGF-1 using adenovirus exhibited improved tenocyte morphology and biomechanical parameters compared to naïve MSCs [12]. These gene-enhanced stem cells with transfected growth factors can be injected directly in the repair site or be built into scaffolds [12]. In rat rotator cuff models, fibroblasts that were transduced with PDGF-β and IGF-1 and integrated into synthetic scaffolds showed improved tenocyte proliferation, cellular repair, and collagen formation [12, 46]. These selective tenocyte differentiation methods using genetically modified MSCs show a promising direction for further study towards optimal tendon repair [44, 47].

27.8 Adipose-Derived Stem Cells

While MSCs, specifically BMSCs (bone-marrow stem cells), were the choice in early studies for stem cell tendon therapy, adipose-derived stem cells (ASCs) have recently been shown as effective and even faster in proliferation and tenocyte differentiation compared to MSCs [39, 48]. Cultivating MSCs ex vivo prior to implantation is labor and time consuming [39]. ASCs, in contrast, have potential for tendon regeneration in vivo with low donor site morbidity [39, 48]. Using ASCs for cellular therapy allows for the potential of a one-step procedure where ASCs could be harvested and delivered back to the tendon repair site during the same surgery [48]. The use of ASCs for tendon repair treatment could provide a less expensive and time-consuming option providing similar, if not improved results compared to MSCs [48].

Studies have been done to separate ASCs into various subpopulations with differing differentiation potential with some populations favoring tenocyte generation [49]. Gonçalves et al. showed that by identifying and only using the ASC populations that expresses tenomodulin (TNMD), a marker for tendons and ligaments, an increase of upregulation for tenocyte generation as well as collagen I and collagen III can be achieved compared to general, unsorted ASCs [49]. Researchers used the TNMD+ ACSs with growth factor supplementation to achieve increased tenocyte generation [49]. However, even without any growth factors, TNMD+ cells still expressed tendon markers in high amount [49]. This novel research identifying and implementing tenocyte-driven subpopulations of ASCs opens new areas of study for improved tendon tissue engineering.

27.9 Directed Tenocyte Differentiation

There has been continued research to further identify new ways of only generating new tenocytes from other stem cells. One of the more recent studies successfully demonstrated tenogenic induction using human embryonic stem cells (hESC) [50]. Researchers delivered BMP12, BMP13, and ascorbic acid to hESCs for 40 days and achieved new tenocyte growth similar to native tenocytes in morphology [50].

Neural crest stem cells (NCSCs) derived from induced pluripotent stem cells (iPSCs) are multipotent and also have the ability of forming tenocytes among other mesenchymal lineages [51]. One study found that iPSC-derived NCSCs improved tendon repair at 4 weeks and generated fetal tendon-related matrix proteins, tenogenic differentiation factors and increased the rate of endogenous repair [51]. These selective tenocyte differentiation methods could pave the way for novel tendon engineering with improved results [50].

27.10 Summary

When it comes to tendon healing, balance and timing is key. Today, much of the promising research and newer tendon therapies utilized several of the aforementioned innovations in combination. Engineered growth factors, stem cells and various delivery methods of scaffolds and viral vectors provide many variables showing encouraging results. In addition to these techniques, more research is being done to examine selective tenocyte differentiation from multipotent and pluripotent stem cells, such as NCSCs and hESCs, to yield improved regenerated tendon tissue [50, 51]. At this time, no clinical research has been found to regenerate injured tendon tissue that will mimic native tendons. However, these advancements along with improved tissue engineering show encouraging progress that has laid the groundwork for future breakthroughs towards optimal tendon repair.

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Peroneal Tendon Injuries

28

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28.1 Introduction

Improved knowledge based on recent literature confirmed that peroneal tendon injuries are a serious cause of posterolateral ankle symptoms following acute or chronic lateral ankle sprains [1, 2]. These injuries, however, are often misdiagnosed as lateral ankle ligament pathology, resulting in suboptimal management, which may lead to long-term sequelae and more chronic pathology requiring invasive treatment. Therefore, accurate diagnosis and prompt treatment in an early stage is important [2, 3]. Adequate knowledge of the peroneal tendons' anatomy and clinical presentation of the associated pathologies is essential to optimize management of peroneal tendon injuries. This chapter provides an overview of the anatomy of the peroneal tendons and the clinical presentation, diagnostics, and management of associated injuries. Moreover, it provides a step-by-step description of the peroneal tendoscopy procedure.

28.2 Anatomy and Function of the Peroneal Tendons

There are two peroneal muscles: the peroneus brevis (PB) and the peroneus longus (PL) muscle. Together they form the lateral compartment of the lower leg, or "the peroneal compartment." The peroneal tendons act as the primary evertors and abductors of the foot. In this manner, they play an important role in providing the active lateral ankle stability, in the foot's eversion strength, and stabilization of the lateral column of the foot during stance. It remains unclear if one of the tendons has greater contractile strength than the other. While early research found that the force generating capacity of the PL was twice as high as the PB, a more recent study suggested that the PB was the more effective foot evertor [4].

The PL originates at the lateral condyle of the tibia, the lateral aspect of proximal fibular head, the intramuscular septa, and the adjacent fascia.

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The PB originates more distally on the fibular shaft and inter-osseous membrane. Where the PL muscle becomes tendinous 3–4 cm proximal to the distal fibular tip, the PB muscle usually runs up to 2 cm more distally [5]. In some cases, the PB musculotendinous junction occurs beyond the fibular tip, better known as a low-lying muscle belly [6, 7]. There is no consensus in literature whether this variation predisposes the tendons to pathology [7].

Around the fibular tip both tendons share a common fibro-osseous tunnel in which the PB is anteromedially located from the PL and flattened against the fibula. This tunnel is formed by the superior peroneal retinaculum (SPR), the deep posterior compartment fascia, and the retromalleolar groove of the fibula which is buttressed by a fibrocartilagenous ridge [8]. The SPR provides stability of the tendons within the groove and is therefore critical in preventing the tendons to dislocate.

Distal to the fibular tip, the tendons are separated by the calcaneal peroneal tubercle. Here, each tendon enters an individual fibrous tunnel, secured by the inferior peroneal retinaculum. A cadaveric study found the peroneal tubercle to be considered prominent in 29% of specimens, which may lead to pain and damage to the tendons [7].

After curling around the fibular tip, the tendons course posteroinferolaterally; the PB inserts at the base of the fifth metatarsal while the PL tendon runs more distally and after turning plantarly at the cuboid groove, it inserts at the plantar side of the medial cuneiform and the first metatarsal base (Fig. 28.1). At the level where the PL curls around the cuboid bone, an os peroneum (OP) is found in up to 4–30% of specimen [9, 10]. The OP protects the PL tendon from damage at the location where it redirects from the lateral to the medial aspect of the foot, but has also been associated with peroneal tendon pathology [9, 10].

The superficial peroneal nerve innervates both tendons and blood is supplied by the peroneal artery and branches of the anterior tibial artery. Branches run through a common vincula formed by the distal fibers of the PB muscle belly and penetrates the tendons over their entire length [11, 12] (Fig. 28.2). Historically it has been assumed that the peroneal tendons have critical avascular zones around the distal fibular tip and the cuboid bone, playing a role in the development of pathologies [13]. Recent research, however, found no evidence to support these avascular zones [12].

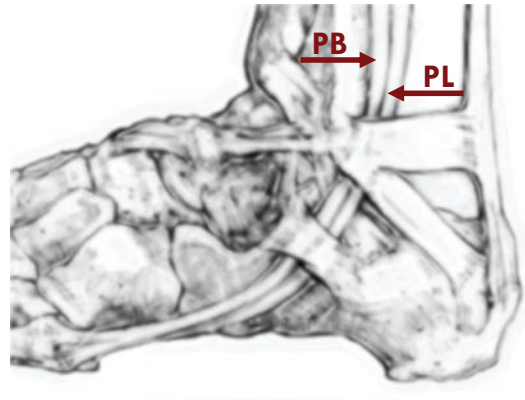

Fig. 28.1 After curling around the fibular tip, the tendons course posteroinferolaterally. The peroneus brevis (PB) tendon inserts at the base of the fifth metatarsal, the peroneus longus (PL) tendon runs more distally and after turns plantarly at the cuboid groove, it inserts at the plantar side of the medial cuneiform and the first metatarsal base

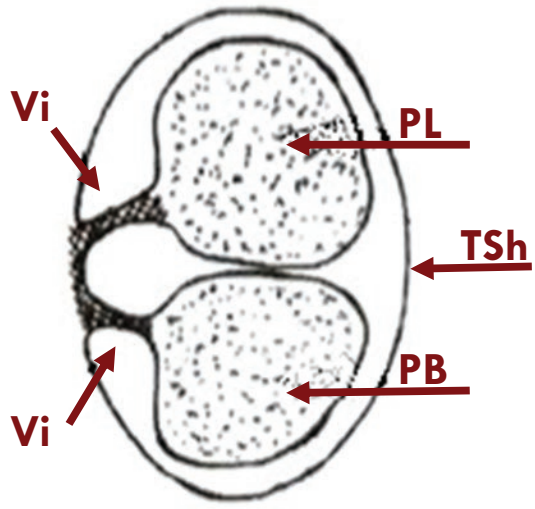

Fig. 28.2 Axial view of the common vincula (Vi). Vascular branches run through the vincula formed by the distal fibers of the peroneus brevis (PB) muscle belly and penetrate the tendons over their entire length. **Ths* Tendonsheath, *PL* Peroneus longus

28.3 Peroneal Tendon Injuries

During inversion of the ankle, the peroneal tendons are exposed to high mechanical loads and remain under significant pressure within the retromalleolar groove [1, 14, 15]. Recurrent ankle sprains amplify these loads, chronically squeezing the PB in between the PL and the retromalleolar groove [15]. In this way, the PB is predisposed to hypertrophic tendinopathy, recurrent stenosis, tearing, or rupturing of the tendon [1]. As discussed in the "Anatomy and Function of the Peroneal Tendons" (Sect. 28.2), several anatomical variabilities may predispose the tendons to pathology. Other predisposing factors include rheumatoid arthritis, psoriatic arthritis, diabetic neuropathy, calcaneal fractures, fluoroquinolone use, and local steroid injections [16–22].

Pathology of the peroneal tendons may occur anywhere along the course of the tendons, but is most often found within the areas where the tendons are exposed to the greatest stress: around the lateral malleolus (PB), the peroneal tubercle (PB and PL), or within the cuboid groove (PL). In general, pathology linked to the peroneal tendons is categorized into three types: (1) tendinopathy (tendinitis, tenosynovitis, tendinosis, and stenosis), (2) partial or complete ("rupture") peroneal tendon tears, and (3) subluxation or dislocation [23]. Other pathologies causing posterolateral ankle symptoms include chronic lateral ankle instability, posterior ankle impingement, avulsion or calcification of the posterior talofibular ligament (PFTL), bony spurs, rheumatoid arthritis, and disorders of the posterior compartment of the subtalar joint [24].

28.3.1 Tendinopathy: Tendinitis, Tenosynovitis, Tendinosis, and Stenosis

Chronic peroneal tendinitis and tenosynovitis may lead to degeneration of each tendon's collagen fibrils, also known as tendinosis. In recent literature, however, it is preferred to only use the term tendinopathy. Microscopically, tendinosis is characterized by increase of mucoid ground substance, loss of collagen continuity, hyperplasia of the tenocytes or fibroblasts, hypervascularization, and necrosis [15, 25]. Macroscopically, the tendon's surface changes to dull, predominantly brown and/or gray, and irregular thickening. If left unaddressed, chronic tendinopathy can eventually lead to fibrosis, synovial proliferation, hypertrophy and stenosis of the tendon within its tendon sheath [26].

28.3.2 Tears and Ruptures

The recent peroneal tendon consensus statement of the ESSKA-AFAS defined tears as partial (either simple or complex) longitudinal tendon tears that do not result in complete discontinuity of the muscle tendon unit. Ruptures were defined as transverse discontinuity, resulting in complete dissociation of the muscle and tendon at that level [23].

The prevalence of peroneal tendon tears in general population remains unclear, but cadaveric studies found tears in 11–38% of specimens [27, 28]. With the PB tendon being squeezed in between the PL tendon and the bony fibular groove, it is most prone to tear at that level [29, 30]. A cadaveric study found a PB tendon tear in 87.5% of the specimen, while a PL tendon tear was found in only 12.5% [31]. Another study found concomitant tears in both tendons in 38% of patients treated operatively for peroneal tendon tears [3].

28.3.3 Subluxation and Dislocation of the Peroneal Tendons

Peroneal tendon dislocation has been reported in 0.3–0.5% of all traumatic ankle injuries and is most prevalent in the athletic population performing sports that require short cutting movement, such as soccer, gymnastics, and skiing [32].

Dislocation occurs when one or both tendons are displaced from the retromalleolar groove, typically provoked by sudden eccentric contraction of the peroneal muscles against acute plantarflexion of the inverted foot or forced dorsiflexion during eversion. The PL tendon is more prone to dislocate than the PB tendon, due its anatomical location in between the PB tendon and the SPR.

Peroneal tendon dislocation is generally classified in four grades [23, 33, 34]. Grade one, found in over 50% of the cases, includes cases where the SPR is subperiosteally elevated from the fibula. In grade two, around 33%, the SPR is elevated together with the fibrocartilagenous ridge. In grade three, approximately 13%, the SPR is completely ruptured off the fibula together with a cortical fragment [33]. Grade four, which is rarely diagnosed, includes cases with a ruptured posterior part of the retinaculum [34]. More recent, Raikin added an extra classification of intrasheath subluxation, with the SPR remaining intact while the peroneal tendon change from their natural position within the retromalleolar groove [35]. In type A, the PL lies deep in relation to the PB, and in type B, the PL subluxates through a tear within the PB [35].

28.3.4 (Painful) Os Peroneum Syndrome

The (painful) os peroneus syndrome (POPS) is a relatively uncommon condition and forms an umbrella term for different types of pathology associated with the OP [9]: (1) entrapment of the OP and PL tendon as a result of an hypertrophic peroneal tubercle, (2) (partial) PL tear, (3) rupture of the PL, (4) acute fracture of the OP or diastasis of a multipartite OP, and (5) chronic fracture of the OP associated with PL stenosing tenosynovitis.

28.4 Patient History and Clinical Examination

In the opinion of the authors, careful patient history and clinical examination is the most important key to proper diagnosis of peroneal tendon injuries. Acute injuries are often reported as "an ankle sprain that never resolved," while chronic disorders occur after a gross ankle inversion trauma in the medical history or in patients with chronic lateral ankle ligament instability. Patients typically present with lateral ankle pain or pain along the course of the peroneal tendons that worsens with activity. Other symptoms reported include swelling, tenderness, giving way and lateral ankle instability. Differentiation between peroneal tendinopathy and tearing of the tendon during physical examination is difficult; a tendon tear may appear with less pain but more weakness and swelling. In case of dislocation, the patient may report a popping or snapping sensation.

Findings during physical examination include a recognizable tenderness over the peroneal tendons, crepitus, and swelling. Active dorsiflexion and eversion often exacerbate pain, and muscle strength can be weaker when compared to the contralateral side. In tears, pain may be exacerbated on acute loosening of resistance during the provocation test [29, 36]. Possible dislocation of the tendons sometimes can often be provoked during physical examination by combined active dorsiflexion and eversion [37].

28.5 Additional Diagnostics

While thorough patient history and physical examination is key to pinpoint the exact diagnosis, in most cases additional diagnostics are required to rule out other pathologies and to create an optimal treatment strategy.

To rule out acute and chronic osseous pathologies such as fractures, spurs, or calcifications, weight-bearing radiographs in anteroposterior and lateral direction are recommended. Moreover, in case of type 3 peroneal tendon dislocation, a small avulsion fracture of the lateral malleolus or "fleck sign" may be visible on the anteroposterior view (Fig. 28.3) [38].

For evaluation of the peroneal tendons and surrounding structures, MRI remains the standard diagnostic test [29] with a reported sensitivity and specificity of 84–90% and 72–75%, respectively [39, 40]. Normal peroneal tendons appear with homogenous signal intensity on T1- and T2-weighted images. Abnormalities include a C-shaped tendon, clefts, irregularity of the tendon contour, and increased signal intensity due to fluid within the tendon sheath (Fig. 28.4) [41,

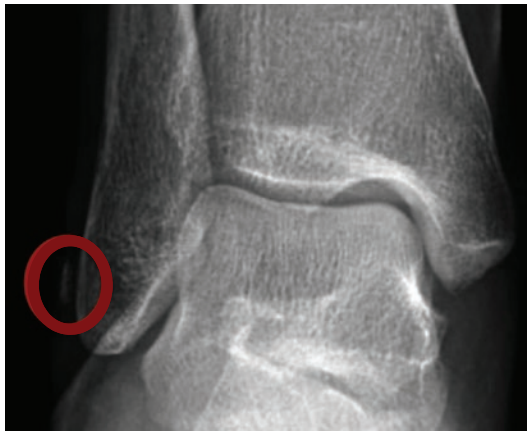

Fig. 28.3 In case of type 3 peroneal tendon dislocation, a small avulsion fracture of the lateral malleolus or "fleck sign" may be visible on the anteroposterior view

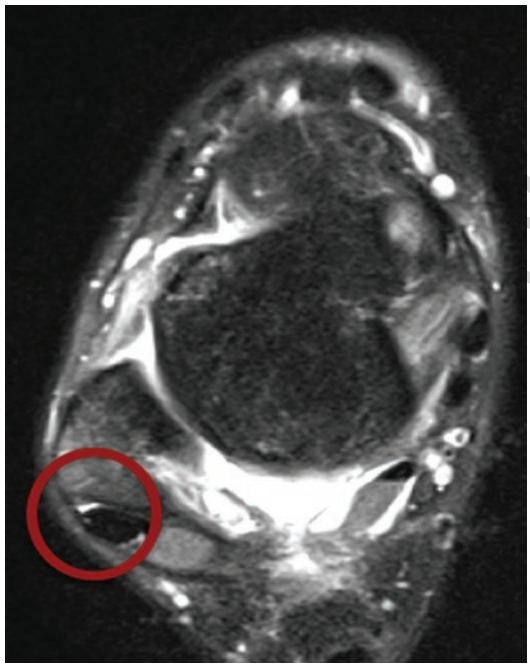

Fig. 28.4 A C-shaped and irregular peroneus brevis tendon with increased signal intensity due to fluid within the tendon sheath; suggestive for a peroneal tendon tear

42]. An increased signal intensity, however, can also be seen in asymptomatic patients due to the so-called magic angle effect [26]. While this effect only appears on T1-weighted images, in tears these signal abnormalities are found on both T1- and T2-weighted images. This underscores the importance of evaluating the tendons in both settings.

Ultrasound (US) has several advantages in comparison to MRI; it is less expensive, can be employed in the outpatient clinic, and has the ability of dynamic evaluation of the tendons. The last matter makes it easier to diagnose dynamic injuries such as (episodic) subluxation, dislocation, and tears that are not seen on MRI. It must be taken into account, however, that the quality of the US is strongly correlated with the quality of the observer. Abnormalities visible on US include tendon thickening, peritendinous fluid within the tendon sheath, ruptures, and luxation of the tendon(s) over the fibular tip.

Peroneal tendoscopy should be reserved for patients with a high clinical suspicion of peroneal pathology, but absence of positive findings or inconclusive abnormalities on imaging [40, 43]. It is highly sensitive and specific for both static and dynamic injuries and provides an easy transition to (minimally invasive) treatment [40, 44].

28.6 Treatment

While there is only limited evidence, conservative management is the first step in treatment of peroneal tendon injuries, including a period of rest, immobilization to reduce symptoms, or activity modification [23]. Physical therapy is recommended in order to strengthen the peroneal and surrounding muscles. When symptoms persist longer than 3 months, there exists mounting evidence for the use of shockwave therapy [23].

If conservative treatment fails, surgical treatment should be considered. Especially in tears and dislocation, surgery is required in most cases since these pathologies rarely heal themselves [3, 31, 45]. According to the recent peroneal tendon consensus statement of the ESSKA-AFAS, the first choice of operative treatment of peroneal tendon tears includes debridement and tubularization of one or both tendons. Only in cases this is not feasible, single stage autograft with the hamstrings or side-to-side tenodesis is recommended. If one of the tendons is deemed irreparable, it is recommended to perform debridement

and tubularization on the reparable tendon and an autograft or tenodesis procedure on the irreparable tendon. If neither of the tendons can be repaired and the proximal muscle tissue is healthy, single stage autograft is recommended [2, 3, 23].

When treating dislocation operatively, multiple operative techniques have been described, all with the primary purpose to restabilize the tendons back into the retromalleolar groove by restoring the anatomy of the superior peroneal tunnel. The different techniques can generally be divided into four groups: (1) repair or replacement of the SPR, (2) deepening of the retromalleolar groove, (3) bone-block procedures, and (4) enhancement of the SPR by rerouting of other soft tissue structures. The latter two are associated with relatively high complication rates, and therefore over the last years attention is drawn to the first two categories. Studies looking at repair of the SPR, with or without concomitant groove deepening, show promising outcomes, high satisfaction, and a 83–100% rate of return to sports [35, 46]. Evidence showed that the combination of SPR repair and retromalleolar groove deepening provides significant higher return to sports rates as compared to SPR repair alone ($p = 0.022$) [47], and therefore the combination of (endoscopic) groove deepening and retinaculum repair is recommended in athletes [23].

Over the last year, peroneal tendoscopy has become more appreciated as a treatment modality [11, 43, 48, 49]. Not only does it accommodate an accurate diagnostic tool as noted in Sect. 28.5, it is also associated with functional improvements in patients with peroneal tendon injuries. The primary indication for peroneal tendoscopy is posterolateral pain due to tenosynovitis, subluxation or dislocation, partial tears or postoperative adhesion [11]. Recent studies report a relatively low rate of complications with reduced costs and earlier recovery when compared with traditional open procedures [40, 50–53].

Inadequate management of anatomical abnormalities may lead to persistent pain and dysfunction on the longer term. Therefore, during operative treatment of peroneal tendon injuries, additional predisposing factors should also be assessed [54, 55]. Additional procedures such as a lateralizing calcaneal osteotomy may be necessary in case of hindfoot varus [14].

28.7 Peroneal Tendoscopy: A Step-by-Step Description of the Procedure

A peroneal tendoscopic procedure can be performed in the outpatient clinic under local, regional, epidural, or general anesthesia. Optimal portal access is achieved in lateral decubitus position with the foot supine, allowing access to both the anterior and the posterior aspect of the ankle when an open procedure is required. In case an arthroscopic procedure in conjunction with tendoscopy is considered, the patient is best placed in semi lateral position in order to facilitate access to the anterior as well as to the lateral ankle.

Before anesthesia is administered, the patient is asked to actively evert the foot in order to locate the tendons and to draw their course on the skin. Moreover, the portal locations and the course of the superficial peroneal nerve are marked. Next, a tourniquet is placed around the upper leg to optimize visualization and a support is placed under the leg to promote free ankle motion during surgery.

In most cases, the use of two portals is sufficient. First, the distal portal is created 2–3 cm distal to the posterior tip of the lateral malleolus (Fig. 28.5). An incision is made through the skin, followed by penetration of the tendon sheath by a mosquito clamp. A 2.7 mm 30° arthroscope is introduced and the tendon sheath is filled with saline using a low pressure, low flow pump of 50–70 mmHg. Some surgeons prefer a 4 mm scope, which produces an increased flow under a lower pressure [44]. Passing the larger diameter scope through the retinaculum, however, can be challenging [48]. The second portal is made under direct vision of the scope by introducing a spinal needle, approximately 2–3 cm proximal to the posterior edge of the lateral malleolus (Fig. 28.5).

Inspection of the tendons starts around 6 cm proximal to the posterior edge of the lateral malleolus. At this level, a thin membrane splits the tendon compartment into two separate chambers. Running more distally, the tendons share one compartment. Rotating the scope over and in between the tendons and within the tendonsheath, the complete course of the tendons can be evaluated. In case of significant tenosynovitis, complete tenosynovectomy using a shaver can be performed in order to allow better visualization of possible pathologies including tears, ruptures, dislocation, and stenosis [44].

In patients with subluxating or dislocating tendons, fibular groove deepening can be performed using the tendoscopic technique. In should be taken into account, however, that the limited workspace around the fibular tip makes this procedure time consuming and challenging. When performing a tendoscopic groove deepening, it is therefore preferred to create an extra portal 4 cm proximal to the posterolateral portal [56]. To minimize the risk of iatrogenic tendon damage, two Kirschner wires are used to keep the peroneal tendons out of the working area. A concavity in the retromalleolar groove can be created using a 3.5 mm burr. To prevent the tendons from damage, the surface of the groove is smoothened and possible sharp edges are rounded. After finishing the procedure, the stability of the tendons within the groove can be tested. Only in case of persistent instability after the groove deepening procedure, a ruptured SPR is sutured with the use of suture anchors [23]. The authors prefer the endoscopic groove deepening technique by means of the 2 portal hindfoot approach since it provides a better overview of both tendons and the groove itself. Moreover, it allows better judgment on the amount of deepening both in width and in depth [56].

When a tendon tear is found, a mini-open approach is required for optimal debridement of the degenerative tissue. Depending on the amount of tissue removed, the tendon is tubularized using the buried sutures knot and a running technique (Fig. 28.6).

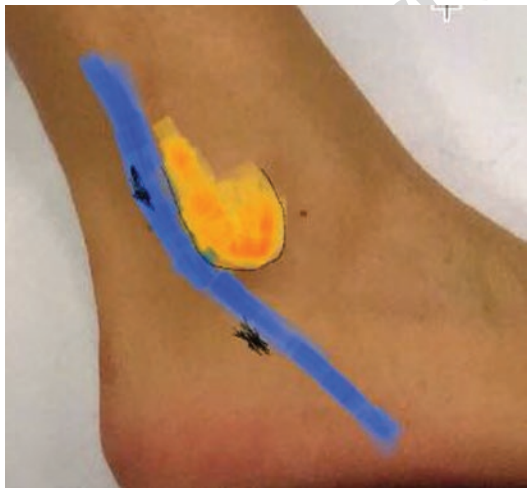

Fig. 28.5 The location of the portals (marked in black) in relation to the lateral malleolus (yellow): (**a**) The distal portal is created 2–3 cm distal to the posterior tip of the lateral malleolus. (**b**) The proximal portal is located 2–3 cm proximal to the posterior edge of the lateral malleolus

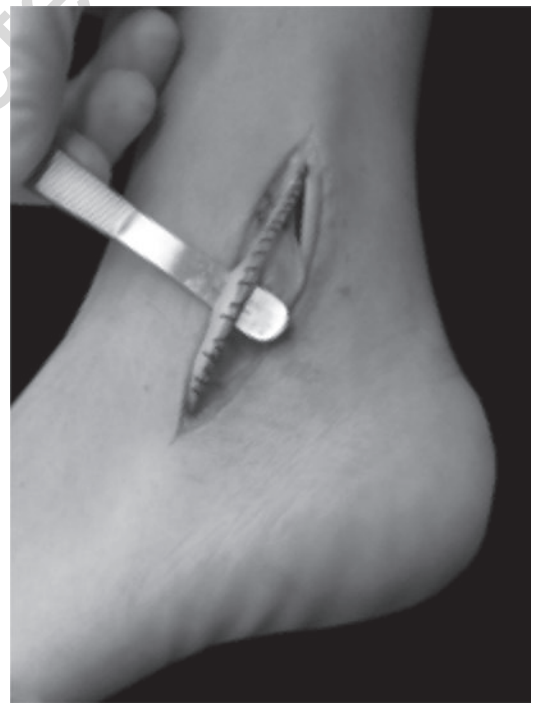

Fig. 28.6 In case of a peroneal tendon tear, a mini-open approach is required for optimal debridement of the degenerative tissue followed by tubularization of the remaining tendon

At the end of the procedure, all portals are closed by sutures to prevent sinus formation.

28.8 Rehabilitation

Adequate rehabilitation is an important factor for optimal management of peroneal tendon injuries and should be individualized for each patient [57]. For optimal rehabilitation the surgeon must distinguish whether or not the SPR is repaired.

In cases the SPR is not repaired, rehabilitation should be goal-based with the promotion of early mobilization, rather than time-based. If surgery included repair of the retinaculum, rehabilitation should start with 2 weeks of non-weight bearing in a lower leg cast, followed by 4 weeks of weight bearing in a cast or a walker boot. 2 weeks after the procedure, active range of motion can be started. It is important that the tendons are not loaded until 6 weeks after repair of the SPR [23].

28.9 Conclusions

Recent literature confirmed that peroneal tendon injuries are a serious cause of posterolateral ankle symptoms following acute or chronic lateral ankle sprains, and can be very debilitating. To prevent the tendons from chronic damage and deterioration, early diagnosis and treatment is important. While MRI and US are both helpful tools in diagnosing peroneal tendon injuries, accurate patient history and clinical examination is the key to adequate diagnosis and management. Conservative management remains the first choice of treatment, but most cases of peroneal tendon tears, ruptures and dislocation require surgical intervention. With an advantageous character compared to open treatment, peroneal tendoscopy has become a popular tool for both diagnosis and treatment of peroneal tendon injuries. Not only does it provide a minimal invasive technique with a low complication risk, it is also associated with a high satisfaction of patients.

28.10 Pearls and Pitfalls

1. While MRI and US can be helpful tools in the management of peroneal tendon injuries, accurate patient history and clinical examination is the key to adequate diagnosis and treatment.
2. Peroneal tendoscopy should be reserved for patients with a high clinical suspicion of peroneal pathology, but absence of positive findings on imaging. It is highly sensitive and specific for both static and dynamic injuries and provides an easy transition to (minimally invasive) treatment.
3. In peroneal tendoscopy, identify the location of the peroneal tendons by asking the patient to actively evert the foot and draw the course of the tendons on the skin before starting a surgical procedure. Moreover, localize the maximal pain spot and mark this on the skin. In this way, a clear reference for your portals and intraoperative reference point is created.
4. Identify the posterior talofibular ligament and the calcaneofibular ligament before initiating the work on the posterior distal fibular surface during a groove deepening procedure to prevent iatrogenic damage.
5. Introduction of the surgical instruments must be performed smoothly without any resistance to prevent iatrogenic tendon damage. Increase of fluid pressure during the tendoscopy allows for more working space, thereby preventing iatrogenic damage.
6. Don't include retinacular tissue during closure of the portals in order to prevent adhesions.

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29 Concept of the Hindfoot Endoscopy

Jin Woo Lee and Bom Soo Kim

29.1 Introduction

Posterior ankle pain is a frequently observed problem, especially in athletics or active patients. Various pathologies can be associated with pain or discomfort in the posterior aspect of the ankle. Intra-articularly, osteochondral lesions located in the posterior aspect of the ankle joint or in the posterior facet of the subtalar joint, as well as loose bodies, osteophytes, and synovitis, can cause hindfoot pain. Extra-articular pathologies include tenosynovitis of the flexor hallucis longus (FHL) tendon, posterior impingement due to os trigonum, inflammatory tissue, or hypertrophic posterior capsule or intermalleolar ligaments.

Due to the anatomic complexity and deep location, open approaches provide limited visualization and cause difficulties in the surgical procedures despite large skin incisions and extensive soft tissue dissections. Open approaches accompany greater postoperative pain and require prolonged rehabilitation.

Minimal invasive approach to the posterior aspect of the ankle using endoscopy provides good visualization with less morbidity compared to the open approach. Since its first introduction by van Dijk et al. [1] in 2000, the hindfoot endoscopy using two posterior portals has been rapidly popularized due to its ease of procedure and low complication rate [2–6].

The concept of lateral hindfoot endoscopy includes posterior ankle arthroscopy, subtalar arthroscopy, and hindfoot endoscopy (Fig. 29.1). When the arthroscope is introduced in the extra-articular space, "arthroscopy" becomes a misnomer and hence "endoscopy" is more appropriate. Practically, however, distinguishing between the two terminologies is less important due to the similarity of the procedures. Therefore, "hindfoot endoscopy" can be used as a generalized terminology when endoscopy is used to treat intra- and extra-articular lesions in the posterior aspect of the ankle.

29.2 Surgical Technique

29.2.1 Patient Position and Setup

The patient is placed on the operating table in a prone position. A pneumatic tourniquet is applied around the upper thigh. A small bump is placed under the ankle joint (Fig. 29.2).

To perform endoscopy for the lesions located in the extra-articular space, traction of the joint is not necessary. For most of the lesions located in the posterior aspect of the talus, dorsiflexion of the ankle is usually enough to expose the lesion. In such cases, relaxing the gastrocnemius muscle by slightly flexing the knee joint is helpful to ease the ankle dorsiflexion.

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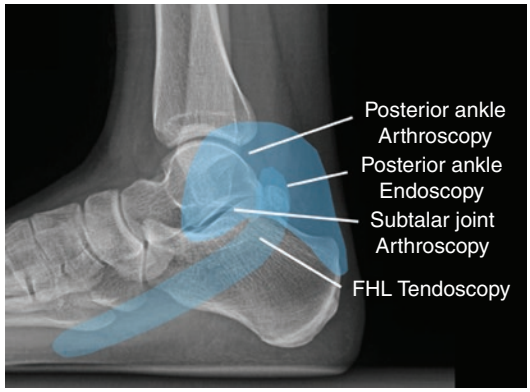

Fig. 29.1 Hindfoot endoscopy is a comprehensive terminology encompassing the posterior ankle and subtalar arthroscopy, posterior endoscopy, and FHL tendoscopy

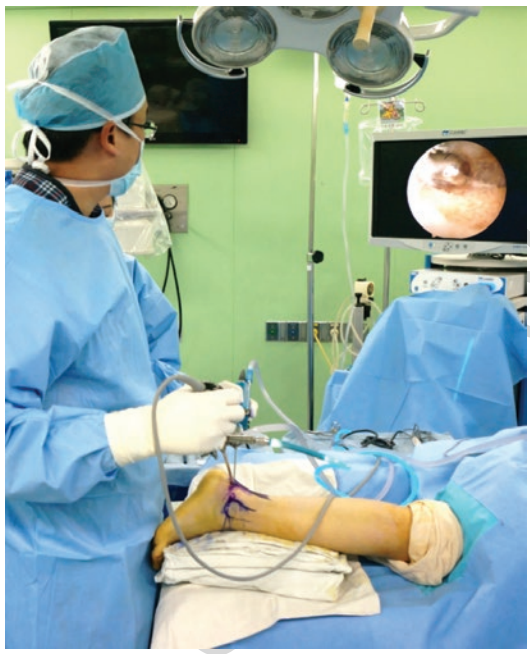

Fig. 29.2 Patient position and the arthroscopic setup. The patient is placed on the operating table in prone position. A pneumatic tourniquet is applied around the upper thigh. A small bump is placed under the ankle joint. Most of the hindfoot endoscopic procedures can be performed without joint distraction

29.2.2 Portal Establishment and Approach

A standard hindfoot endoscopy [1] utilizes two portals, posterolateral and posteromedial. The portals are placed in a same level, at the tip of the lateral malleolus, both about 5 mm away from the

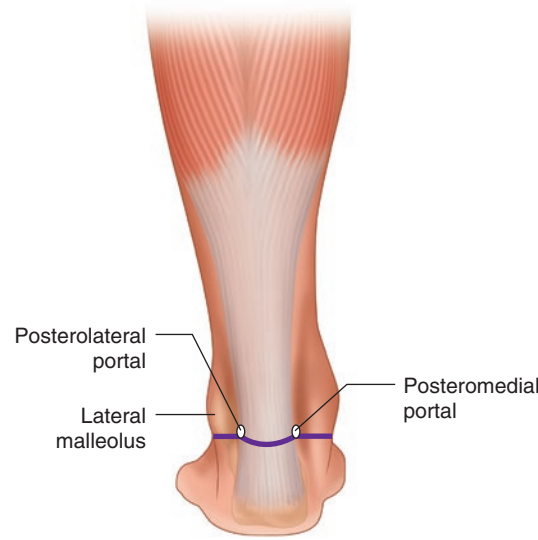

Fig. 29.3 Portal placement. The portals are placed in a same level, at the tip of the lateral malleolus, both about 5 mm away from the lateral and medial border of the Achilles tendon

lateral and medial border of the Achilles tendon (Fig. 29.3). Care should be paid not to place the portals too close to the Achilles tendon because injury of the Achilles tendon by arthroscopic instruments can result in focal enlargement of the tendon and chronic pain. At the same time, injury of the sural nerve must be avoided when making the posterolateral portal.

The posterolateral portal is always made first. A small vertical stab wound is made to incise the skin. A straight mosquito clamp is inserted to split the subcutaneous tissue. The mosquito clamp is then directed towards the first webspace until the tip touches the bony structure (Fig. 29.4). The clamp is then exchanged for an arthroscopic cannula with a blunt trocha. A 4 mm arthroscope is inserted through the cannula [1].

The posteromedial portal is made in the same way on the medial aspect of the Achilles tendon. A shaver is inserted through the posteromedial portal directing laterally towards the proximal shaft of the arthroscope. Once the tip of the shaver touches the shaft of the arthroscope, the shaver tip is moved anteriorly towards the ankle using the arthroscope shaft as a guide. When the shaver tip reaches the bone, the arthroscope is slightly withdrawn to visualize the tip of the shaver. Once the shaver tip is

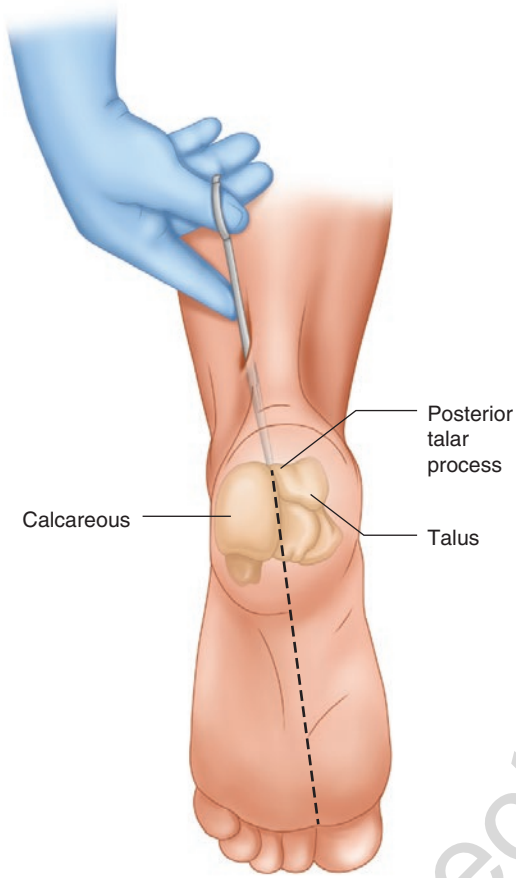

Fig. 29.4 Direction of the mosquito clamp. A straight mosquito inserted into the posterolateral portal is moved deep anteriorly towards the first webspace until the tip touches the bony structure

visualized, working space is created by shaving the extra-articular soft tissue in front of the tip of the arthroscope [1].

29.2.3 Tips and Pitfalls

An alternative method that the authors found to be useful is to use two straight mosquito clamps to make the initial working space (Fig. 29.5). Insert one mosquito clamp through the posterolateral portal, directed towards the first webspace. The tip of the mosquito clamp usually lands on the posterolateral tubercle of the talus or os trigonum.

A second mosquito clamp is inserted through the posteromedial portal directing towards the tip of the first mosquito clamp. Once the tips of the two mosquito clamps are touched in a triangulation fashion, the mosquito clamps are spread a few times to make a working space at the posterior aspect of the talus. Care should be paid not to proceed the clamps too deep medially. Once the working space is made, the mosquito clamps are exchanged with the arthroscope laterally and shaver medially.

This way, less shaving of the soft tissue is required and faster approach to visualize the posterolateral tubercle of the talus is possible.

Blind insertion of the two mosquito clamps directed towards the posterior talar tuberosity can be safely performed because the neurovascular bundle is away from the initial working space. A cadaveric study reported that introducing arthroscopic instruments into the posterior aspect of the ankle with the patient in prone position can be safely performed without gross injury to the posterior neurovascular structures. The average distance between the cannula and adjacent anatomic structures after dissection was 3.2 mm (range, 0–8.9 mm) to the sural nerve, 4.8 mm (range, 0–11.0 mm) to the small saphenous vein, 6.4 mm (range, 0–16.2 mm) to the tibial nerve, 9.6 mm (range, 2.4–20.1 mm) to the posterior tibial artery, 17 mm (range, 19–31 mm) to the medial calcaneal nerve, and 2.7 mm (range, 0–11.2 mm) to the flexor hallucis longus tendon [7].

Once the endoscopy is introduced, identifying the flexor hallucis longus, medial to the os trigonum or the Stieda process is the first step in the hindfoot endoscopy. Flexion and extension movement of the great toe can help identifying the FHL tendon.

Flexor hallucis longus tendon is an important landmark separating the safe zone lateral to the tendon from the danger zone medial to the tendon. Since the neurovascular bundle is in close approximation with the FHL, a special attention should be paid not to use the shaver medial to the FHL tendon.

29.3 Os trigonum and Hindfoot Endoscopy

Os trigonum is one of the most common indication for hindfoot endoscopy [2]. Os trigonum is an accessory bone or separated Stieda process at

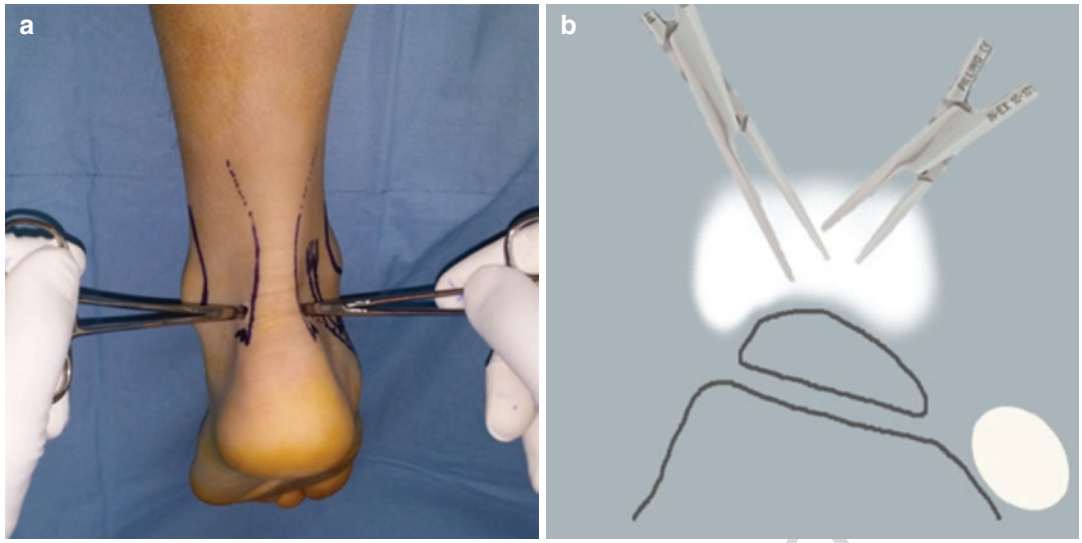

Fig. 29.5 Authors' preferred technique using two straight mosquito clamps. (**a**) The first mosquito clamp is inserted from the posterolateral portal, directed towards the first webspace until the tip lands on bony structure. The second mosquito clamp is inserted from the posteromedial portal, towards the tip of the previously inserted mosquito clamp, in a triangulation fashion. (**b**) Spreading the mosquito clamps right behind the bony structure helps creating the working space with less shaving of the soft tissue

the posterior aspect of the talus. Direct impingement of the os trigonum between tibia and calcaneus during ankle plantar flexion can cause pain. It can also be associated with inflammation causing fluid collection and soft tissue impingement.

When the arthroscope is first introduced at the posterior aspect of the ankle, poor visualization makes it difficult to find the os trigonum. However, since os trigonum is at or near the usual landing site of the arthroscopic instruments directed towards the first webspace, it can be easily palpated with the shaver tip. Shaving of the soft tissue around the hard round bony structure will reveal the os trigonum.

In order to remove the os trigonum, the ligamentous structures, the posterior talofibular ligament laterally and the flexor hallucis longus retinaculum medially, should be released using arthroscopic scissors. If the os trigonum is completely separated from the talus, it can be easily removed with a grasper. If os trigonum is partially attached with the talar body, an arthroscopic burr can be used (Fig. 29.6).

Weiss et al. [8] reported the satisfactory outcomes of endoscopic excision of a symptomatic os trigonum performed in 24 patients. Return to full activity was achieved at an average of 1.5 months with no limitations at an average of 7.8 months after surgery. Out of 24 cases, one transient posterior tibial nerve calcaneal branch neurapraxia has occurred. López Valerio et al. [9] reported similar outcomes after endoscopic excision of os trigonum in 20 soccer players. The mean time until the players' return to previous level of sports was 46.9 days (SD = 25.96).

29.4 Osteochondral Lesion of the Talus and Posterior Ankle Arthroscopy

Most of the osteochondral lesions are located in the anterior 2/3 of the talar dome [10], easily approached with conventional anterior ankle arthroscopy. Posteriorly located lesions account for 13% of the cases [10]. If the joint space is narrow, such lesions do not allow access from the anterior portals.

Posteriorly located osteochondral lesions are more easily approached through the posterior portals with the patient in prone position. The standard posterolateral and posteromedial portals

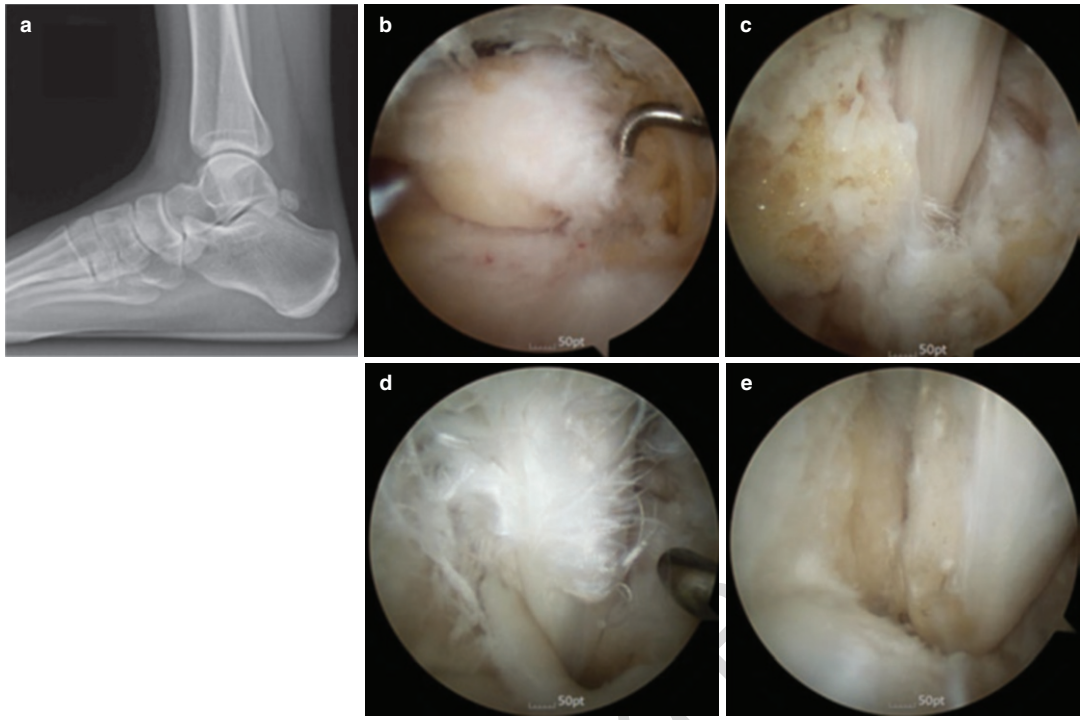

Fig. 29.6 Os trigonum associated with tear of the flexor hallucis longus tendon. (**a**) Os trigonum at the posterior aspect of the talus. (**b**) Endoscopic view of the os trigonum. (**c**) The FHL tendon running in close approximation with the os trigonum. (**d**) Tear of the FHL tendon due to repeated impingement by the os trigonum and the fibro-osseous tunnel. (**e**) After removal of the os trigonum and debridement of the FHL tendon

can be used, but about 1/2 in. proximally placed portals gives better access to the ankle joint.

In order to enter the joint space, the posterior joint capsule and the fatty tissue behind the ankle is removed. Care should be paid not to injure the neurovascular tissue medial to the FHL. Once the joint capsule is removed, the posterior-inferior tibiofibular ligament and the ankle joint space are visualized (Fig. 29.7).

A cadaveric study reported that an average of 54% (range, 42–73%) of the talar dome could be visualized from the posterior arthroscopic approach without traction [7]. Ankle dorsiflexion brings more than 1/3 of the posterior talar dome into exposure. Slight knee flexion to release the gastrocnemius can help ankle dorsiflexion in patients with gastrocnemius tightness. Manual traction of the hindfoot or noninvasive traction of the ankle joint using ankle harness can open the tibiotalar joint, allowing access to more anteriorly located lesions.

29.5 Flexor Hallucis Longus Tenosynovitis and Tendoscopy

Inflammation within the FHL tendon sheath is another frequent cause of posterior ankle pain. Repeated great toe flexion movement and overuse of the FHL tendon cause inflammation within the tendon sheath. Stenosis of the fibro-osseous tunnel due to hypertrophic soft tissue can cause wear and tear of the FHL tendon. Impingement with the freely movable os trigonum can also injure the FHL tendon.

FHL tenosynovitis can be easily diagnosed with MRI scans. Increased synovial fluid in T2-weighted images are diagnostic of FHL tenosynovitis. However, mere fluid collection within the FHL tendon sheath due to overflow of the increased joint fluid from the ankle joint or the subtalar joint should be differentiated.

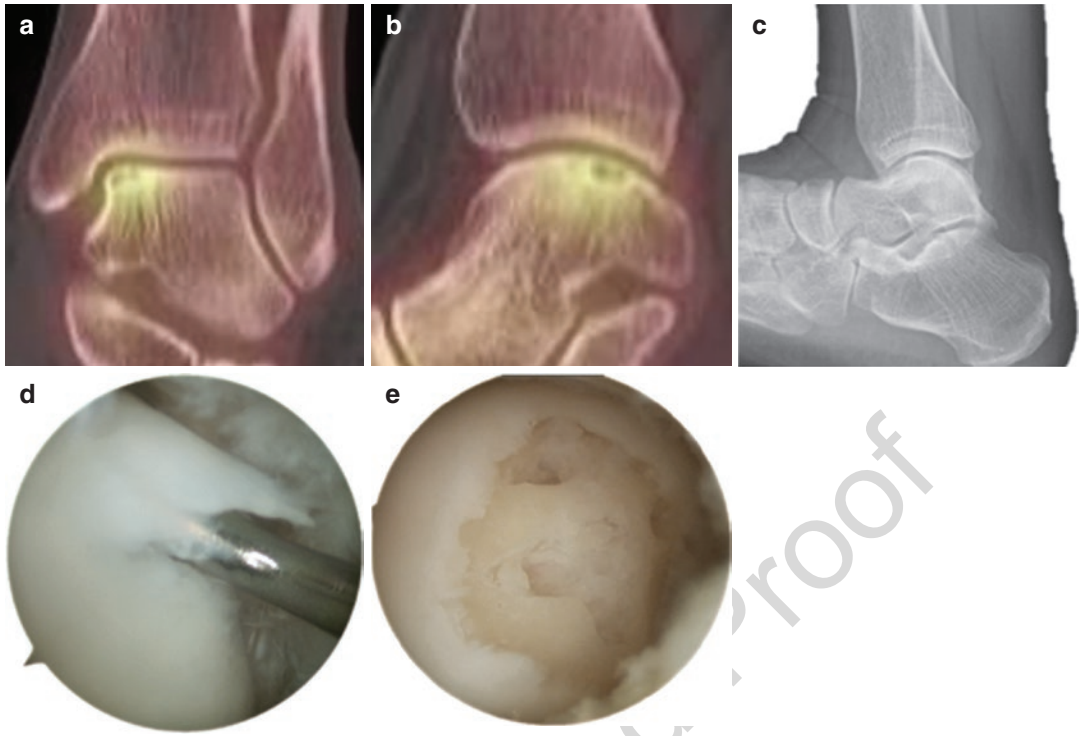

Fig. 29.7 Posteriorly located osteochondral lesion of the talus. (**a**, **b**). The patient was referred to our clinic after failure of anterior arthroscopic approach. Tight joint space, especially with intact ligamentous structures, makes it difficult to approach the deep posteriorly located osteochondral lesion of the talus. (**c**) Ankle dorsiflexion exposes the posterior talar dome out of the tibial coverage. (**d**, **e**) Successful removal of the damaged cartilage and microfracture using the posterior arthroscopic approach

Debridement of the FHL tenosynovitis can be performed using the conventional two posteromedial and posterolateral portals [6] or by utilizing additional plantar portal in the sole [3].

After debridement of the hypertrophic scar tissue at the orifice of the fibro-osseous tunnel, the FHL tendon sheath is entered with the arthroscope. In order to enter the tendon sheath, 2.7 mm or 2.9 mm arthroscope is recommended. Inflammatory synovitis and degenerated vinculae is debrided. Due to the angulation from the posterolateral portal, simultaneous visualization and instrumentation into the tendon sheath is limited to the proximal aspect.

If further instrumentation is required, additional portal is established in the sole by use of a Wissinger rod (Fig. 29.8). With the arthroscope in the posterolateral portal, the Wissinger rod is inserted through the posteromedial portal into the FHL tendon sheath. The rod is passed bluntly, distally to penetrate the plantar muscles in the sole. A stab wound is made in the skin to help exit the sole and create the plantar portal. With the Wissinger rod placed between the plantar portal and the posteromedial portal, the arthroscopic cannula is inserted through the plantar portal using the rod as a guide. This safely places the arthroscopic cannula into the FHL tendon sheath. The rod is then removed and exchanged with arthroscope.

With the arthroscope placed in the FHL tendon sheath through the plantar portal, arthroscopic instruments can be inserted through the posteromedial portal into the FHL tendon sheath. If necessary, the plantar portal and the posteromedial portal can be used interchangeably for visualization and instrumentation.

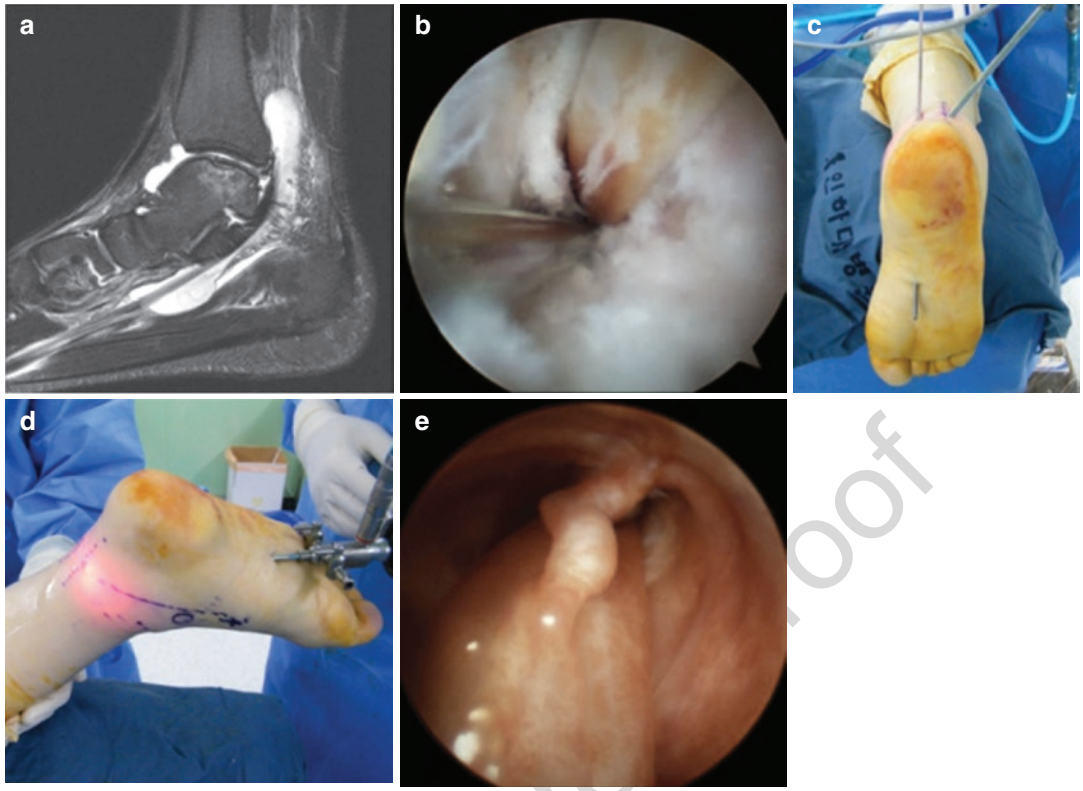

Fig. 29.8 Tenosynovitis of the FHL tendon and tendoscopy utilizing additional plantar portal. (**a**) Increased fluid collection along the FHL tendon sheath. (**b**, **c**) With the arthroscope in the posterolateral portal, a Wissinger rod is inserted in the posteromedial portal, though the FHL tendon sheath, and exited in the plantar aspect of the sole. (**d**) Using the Wissinger rod as a guide, arthroscopic cannular is inserted through the plantar portal. The rod is then exchanged with the arthroscope. (**e**) Tendoscopic view of the FHL tendon showing tenosynovitis and degenerated vinculae

Possible complications associated with the FHL tendoscopy includes injury of the medial and lateral plantar nerve [3, 11–13]. Since the nerve runs in close approximation with the FHL tendon sheath, special caution and gentle manipulation of the instruments is required. Lui et al. [11] performed a cadaveric study and reported that while the proximal half of the tendon sheath is thick and fibrous, the distal half of the sheath is thin and membranous, exposing the nearby nerve at greater risk of injury. Therefore, facing the shaver opening towards the tendon and away from the sheath, as well as minimizing the use of suction, is recommended, especially in the distal half of the FHL tendon [11, 12]. Furthermore, since ankle dorsiflexion brings the nerve closer to the tendon sheath and increase the risk of injury, care should be paid to avoid ankle dorsiflexion [12].

FHL tendoscopy using the conventional two portals is easier and carries less risk of complication compared to the three-portal technique. However, the two-portal technique offers limited inspection and instrumentation. The three-portal technique utilizing additional plantar portal is technically more demanding and carries higher risk of complication, but allows full inspection and better instrumentation. Therefore, the authors recommend starting with the two portals, inspect the proximal aspect of the FHL tendon and depending on the necessity of deeper instrumentation, decide on proceeding with the plantar portal.

29.6 Haglund Deformity and Endoscopic Calcaneoplasty

Enlarged posterior superior calcaneal prominence or the Haglund deformity can cause swelling and pain as a result of repeated mechanical irritation. Formation of a thick skin callosity not only makes shoe wear difficult but can also be a cosmetic problem.

The Haglund deformity can be resected either by open incision or by endoscopic technique [14]. Endoscopic resection of the Haglund deformity has the benefits of minimal invasive surgery including less postoperative pain, faster recovery, and superior cosmetic satisfaction [14–16].

The patient is placed on the operating table in prone position with a bump under the ankle. The first portal can be created on medial or lateral, depending on the surgeon's preference, at the level of the superior aspect of the calcaneus. The portal is placed at the retrocalcaneal space, and fluoroscopic guidance can be helpful. It is recommended to place the first portal as close as possible to the superior edge of the calcaneus and also as far posterior as possible [16]. Care should be paid not to injure the sural nerve when placing the lateral portal. Slight plantar flexion of the ankle allows the instrumentation in the retrocalcaneal space. The second portal is created on the contralateral side using the Wissinger technique. The enlarged posterior superior calcaneal prominence is resected using arthroscopic burr (Fig. 29.9).

When performing endoscopy in the retrocalcaneal space, a special care should be paid to avoid injury of the nearby Achilles tendon. Injury of the Achilles tendon can lead to Achilles tendinopathy or delayed rupture after the surgery. Protection of the Achilles tendon by the hooded side of the burr is recommended. Also, placing the portal too close to the border

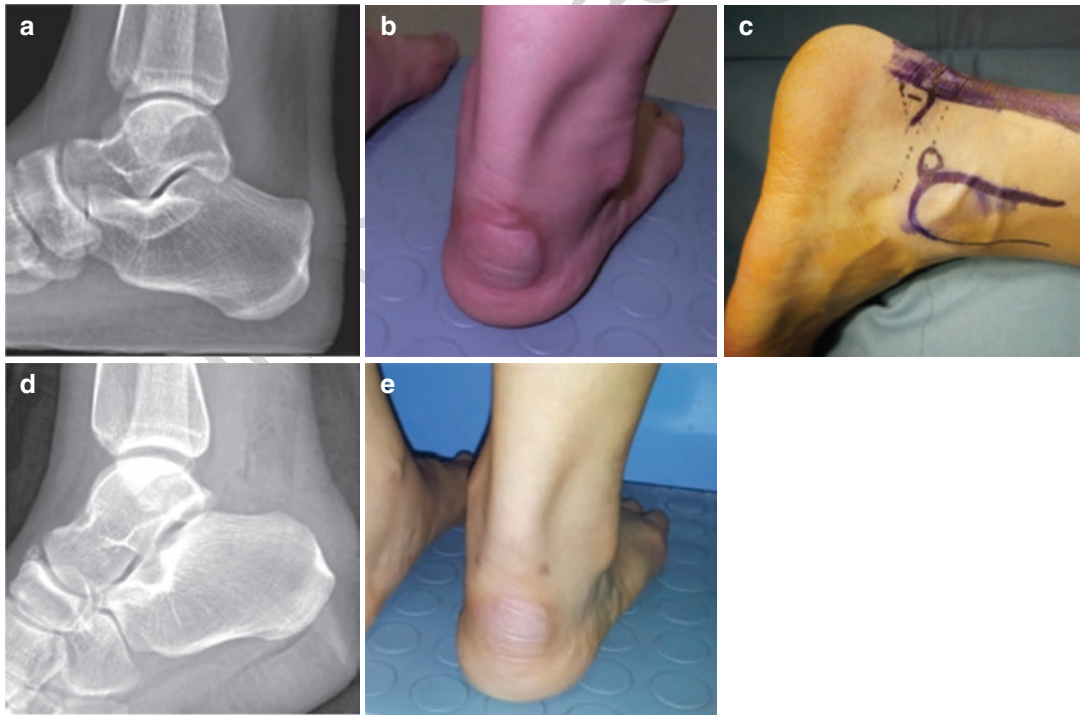

Fig. 29.9 Haglund deformity resected endoscopically. (**a**, **b**) Prominent posterior superior eminence of the calcaneus resulting in painful callosity. (**c**) Portal placement for the endoscopic removal of the Haglund deformity. The portals are placed at the level of the superior border of the calcaneus on each side of the Achilles tendon. (**d**) Successful removal of the bony prominence. (**e**) Reduced callosity at 3 months after the surgery

of the Achilles tendon should be avoided to reduce the risk of tendon injury by the instruments.

29.7 Complications

The endoscopic approach for the treatment of hindfoot and ankle pathology is safe with a low incidence of complications. Donnenwerth and Roukis [17] performed a systematic review study which included a total of 452 ankles that received posterior hindfoot endoscopy. Overall, 17 complications (3.8%) occurred, including 5 wound-healing problems, 4 cases of recurrent symptoms, 3 cases of neuritis of the medial calcaneal nerve, 3 cases of transient incision anesthesia, 1 traumatic sural neuroma, and 1 transient superficial peroneal neuritis. Among these complications, only 8 (1.8%) persisted or required additional treatment or operative intervention: 2 wounds required surgical debridement, 2 reoperations were required for recurrent symptoms, 1 injection was needed for recurrent symptoms, there were 2 cases of persistent medial calcaneal neuritis, and 1 resection of the traumatic sural neuroma was performed.

Kim and Choi [18] reported the outcomes of 10 patients who received flexor hallucis longus tendoscopy using 3-portals. Of these, 9 were satisfactory but 1 experienced ongoing lateral plantar nerve symptom. Lui et al. [12] also reported 2 cases of lateral plantar nerve neuropraxia after FHL tendoscopy using 3-portals. Since ankle dorsiflexion brings the posterior tibial nerve in contact with the arthroscope during tendoscopy, ankle dorsiflexion should be avoided [12].

Lui and Chan [19] performed a cadaveric study and proved that the neurovascular bundle is at risk during instrumentation of the posteromedial ankle through the posteromedial portal but was safe through the posterolateral portal. It is recommended to use the posterolateral portal as the instrumentation portal and the modified or more proximally established posteromedial portal as the visualization portal while dealing with pathology of the posteromedial ankle [19].

Endoscopic calcaneoplasty or resection of the Haglund deformity is a minimally invasive technique with low morbidity. Van Dijk et al. [14] reported generally satisfactory outcomes in 21 cases of endoscopic calcaneoplasty without any surgical complications. Out of 30 cases of endoscopic bony and soft tissue decompression of the retrocalcaneal space, Ortmann and McBryde [15] reported one case of an Achilles tendon rupture 3 weeks after surgery and one case of insufficient resection.

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Foot and Ankle Tendoscopy

30

Phinit Phisitkul, Chris C. Cychosz, and Craig C. Akoh

30.1 Introduction

Tendoscopy was introduced by Wertheimer since 1995 [1]. It was subsequently popularized by van Dijk by extending multiple applications in the foot and ankle [2–4]. The limits of tendoscopy have been challenged by numerous surgeons around the world, especially Dr. Lui who reported versatile use of tendoscopy of both lower and upper extremities [5–8].

Tendoscopy involves minimally invasive surgery with the use of endoscopic instrumentation to visualize and treat tendon pathologies. Its benefits are minimizing surgical morbidities such as pain, scar tissue, infection, and wound complications. Additionally, tendoscopic technique allows for up to 50 times magnification and excellent illumination under endoscopic visualization [9]. The advancement in endoscopic instrumentation such as smaller diameter cameras, more specialized power instrument, and the availability of endoscopic electrocautery probes has facilitated the endoscopic procedures to be performed widely. Surgical indications for tendoscopy in the foot and ankle include diagnostic procedures, decompression, lysis of adhesion, loose body removal, excision, tenotomy, tendon lengthening, and tendon transfers [10–12]. Contraindications for tendoscopy are active overlying infection and extensive postsurgical scarring.

30.2 Principles of Tendoscopy

Most of the tendoscopy are performed under fluid irrigation such as normal saline which can improve distension of the tendon sheath and avoid interference of the view from blood. Occasionally, dry tendoscopy can be used with a slotted cannula such as for gastrocnemius recession [13]. Low-pressure flow up to 40 mmHg or gravity flow provides adequate irrigation while minimizing fluid extravasation [14]. The diameter of the endoscopic camera and its cannula is critical to the success of the procedure. In general, the largest camera that can fit comfortably in the tendon sheath or paratendinous space should be used. A standard 4 mm, 30° camera is ideal for most procedures involving larger structures such as the Achilles and the proximal flexor hallucis longus (FHL) tendons. A 2.7 mm, 30° camera is ideal for smaller structures such as the distal FHL and most other tendoscopy in foot and ankle. It is common

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that the shaft of the camera is used as a retractor as it is placed strategically next to the tendon.

Endoscopic portals are usually made in line with the tendon guided by preoperative markings or direct palpation. Tendon sheath injection with normal saline can facilitate portal placement but it is not routinely necessary. Portals are commonly located proximal and distal to the lesion as determined from preoperative evaluation and imaging studies. Surgeon should keep a minimum of one centimeter of soft tissue tunnel between the skin incision and the location of interest to avoid endoscopic camera dislodgement. The practice of using one finger to stabilize the endoscopic shaft at the portal can be extremely helpful in keeping the camera in place [4]. Portals are mostly created diagonally, so the visualization and instrumentation can be interchanged to cover the length of the lesion. In cases where tendon adhesion is expected, an endoscopic trocar or a hemostat can be used to strip the tendon from surrounding scar tissue prior to camera insertion to improve visualization.

During the learning curve period, the surgeon should not hesitate to convert an endoscopic approach to an open procedure if needed. Patients should be informed and consented for the possibility that an open approach may be required.

30.3 Achilles

30.3.1 Indications

Endoscopy has substantial role in the treatment of Achilles tendon related conditions such as gastrocnemius contracture, Achilles tendinopathy, Achilles tendon rupture within the first 10 to 14 days, and retrocalcaneal bursitis.

30.3.2 Surgical Techniques

30.3.2.1 Gastrocnemius Recession

Positioning: Supine or prone.

Instruments: Slotted cannula and trocar, plane-finder, cotton-tip applicators, 4 mm 30° endoscope, retrograde knife.

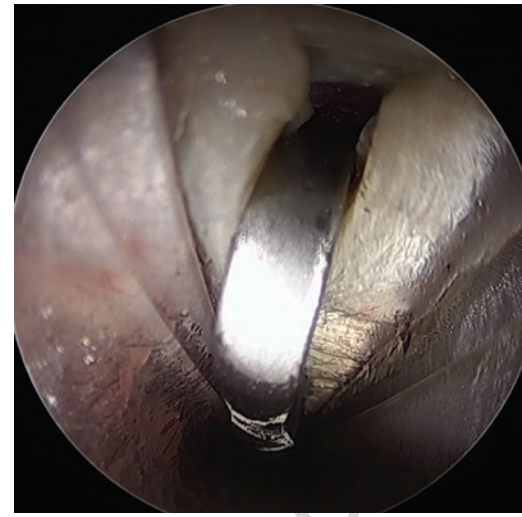

Fig. 30.1 Endoscopic gastrocnemius recession is demonstrated using a slotted cannula and a retrograde knife

A medial portal is created approximately 2 cm distal to the gastrocnemius muscle belly and just dorsal to the palpable gastrocnemius tendon. A slotted cannula and trocar is inserted from medial to lateral along the plane superficial to the gastrocnemius tendon. The lateral portal is created inside out. A 4 mm endoscope is inserted from the medial portal and the gastrocnemius tendon is visualized. The plane of the procedure can be adjusted using a plane-finder and reinsertion of the cannula into the correct plan. The gastrocnemius tendon is released using a retrograde knife from the lateral portal (Fig. 30.1). The camera and the retrograde knife can be switched to complete the release of the tendon medially. The ankle should be able to dorsiflexion past 10° afterwards.

The patient can start progressive weight-bearing in the boot right away. The boot is weaned off at 4–6 weeks.

30.3.2.2 Tenolysis and Longitudinal Tenotomies

Positioning: Prone.

Instruments: 4 mm 30° endoscope, 4.5 mm shaver, electrocautery, retrograde knife, number 11 scalpel.

The tendon enlargement is located by palpation. The proximal medial portal is created 2 cm proxi-

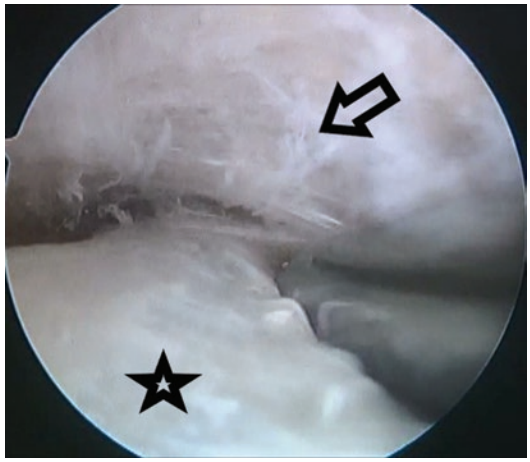

Fig. 30.2 Achilles tendoscopy is shown with a shaver removing adhesion between the Achilles tendon (star) and the overlying fibrous tunnel (arrow)

mal to the tendon enlargement and the distal lateral portal is created 2 cm distally. A hemostat is used to create a plain around the Achilles tendon from both portals. It is common to encounter significant scar adhesion anteriorly. A 4 mm 30° camera is inserted into the anterior aspect of the Achilles tendon. A 4.5 mm shaver is introduced from the other portal for the debridement of scar adhesion (Fig. 30.2). A plantaris tendon may be found in the anteromedial aspect of the Achilles tendon and it may be resected if indicated. If there is a component of stenosing tenosynovitis, the anterior aspect of the fibrous tunnel of the Achilles tendon can be released longitudinally using an electrocautery. Mass occupying lesions such as ganglion cysts, accessory muscle, and low-lying soleus muscle may be excised. If there is significant component of intratendinous lesions, longitudinal tenotomies can be performed using a retrograde knife from one of the portals or if number 11 scalpel percutaneously under direct visualization.

The patient can start progressive weight-bearing in the boot right away. The boot is weaned off at 4–6 weeks.

30.3.2.3 Tendon Repair

Positioning: Prone.

Instruments: 4 mm 30° endoscope, bird-beak suture grasper, number two nonabsorbable sutures.

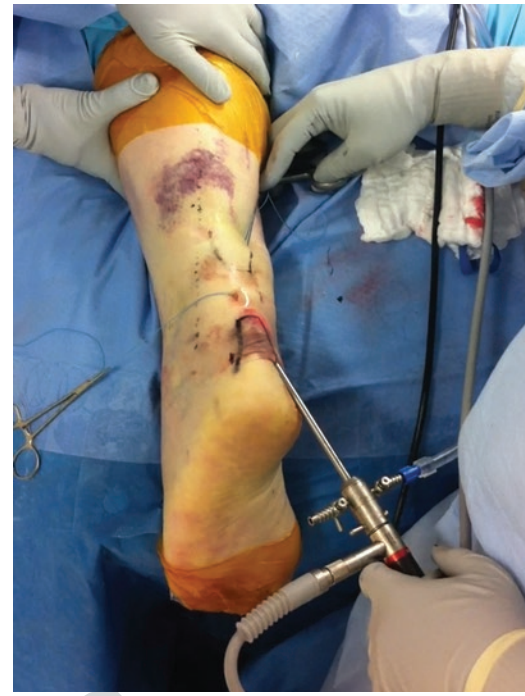

Fig. 30.3 Patient is placed in prone position with the foot just beyond the end of bed. The arthroscope is inserted from the distal-medial portal

The location of Achilles tendon rupture is identified by palpation of a gap on the posterior aspect of the distal hindfoot. Six portals are created on the medial lateral aspects of the Achilles tendon at the level of the rupture, 5 cm proximally, and 5 cm distally (Fig. 30.3). A hemostat is used to create a plain around the ruptured Achilles tendon. With the 4 mm 30° camera from the distal medial portal, a number two nonabsorbable suture is passed across the proximal aspect of the tendon using a bird-beak suture grasper under direct visualization to ensure that the sural nerve is not captured by the suture (Fig. 30.4). This step is also critical as tendinous tissue accounts for only posterior 20% of the stump. Two sets of sutures are placed at the proximal stump to create a box and a crisscross configuration. The camera is removed, and the distal repair is performing the same fashion without the use of an endoscope. The two sets of sutures are tied in the position of maximum plantarflexion after the ankle is moved through range of motion.

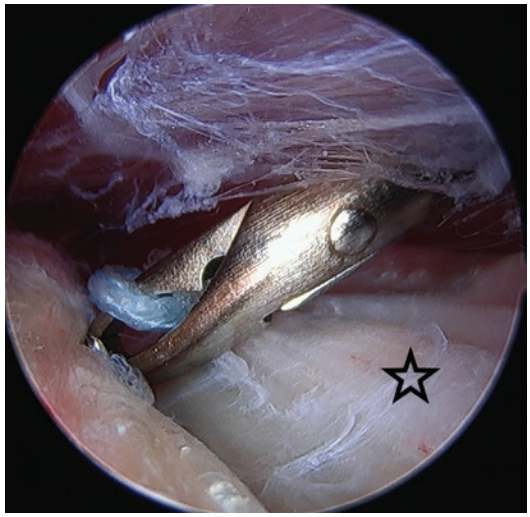

Fig. 30.4 A suture grasper is used to retrieve the end of the nonabsorbable suture as it is passed through the tendinous portion (star) of the proximal tendon stump

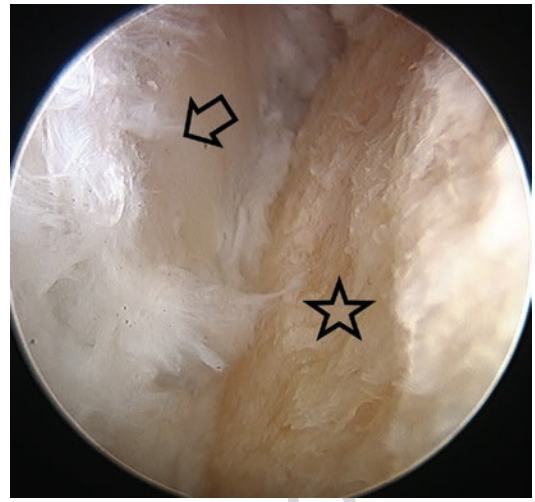

Fig. 30.5 The endoscopic image demonstrates adequate excision of the retrocalcaneal bursa and prominent posterosuperior aspect of the calcaneus (star). Room between the Achilles tendon insertion (arrow) and the calcaneal tuberosity is shown

The patient is immobilized in a boot with the ankle at 20° of plantarflexion for 2 weeks. The patient can start ankle range of motion without dorsiflexion beyond neutral at 2 weeks. At 4 weeks, the boot is adjusted to 10° of plantarflexion and the patient can start progressive weight-bearing. At 6 weeks, the boot is as adjusted to neutral and patient can do full weight-bearing. The boot is discontinued at 10–12 weeks postoperatively.

30.3.2.4 Retrocalcaneal Bursectomy and Decompression

Positioning: Supine or prone.

Instruments: 4 mm 30° endoscope, 5.5 mm shaver, number 11 scalpel.

Two K wires or spinal needles are inserted under fluoroscopic guidance into the calcaneal tuberosity to guide the amount of bone resection. A medial and a lateral portal are made on each side of the Achilles tendon insertion at the level of the superior aspect of the calcaneal tuberosity. A hemostat is used to create a tract into the retrocalcaneal bursa. A camera is inserted, and a shaver is used to remove the inflamed synovial tissue, hypertrophic Achilles tendon, and prominent bone. Bone resection is completed when the two K wires are seen endoscopically. Attention should be paid to remove adequate amount of bone at the most distal aspect where the Achilles tendon inserts and medial and lateral edges of the calcaneal tuberosity (Fig. 30.5). Percutaneous longitudinal tenotomies can be performed under endoscopic guidance using number 11 scalpel if there is associated Achilles tendon degeneration and enlargement.

The patient can start progressive weight-bearing in the boot right away. The boot is weaned off at 2 weeks.

30.3.3 Outcomes of Achilles Tendoscopy

Van Dijk and Scholten published the first case series reporting Achilles tendoscopy outcomes in 1997 [15]. Since that time the Achilles tendon has become one of the most studied tendons using tendoscopy, with most studies comprising level II-V evidence for the indications of tendon rupture repair, peritendinopathy, and midportion Achilles tendinopathy.

Tendoscopy has been utilized to visualize the tendon ends following acute or chronic rupture during percutaneous Achilles tendon repair to grant a more precise re-approximation [16]. Halasi et al. reported a level II comparative study in which a group of 57 patients undergoing percutaneous Achilles tendon repair with the use of endoscopic visualization were compared to a group of 87 patients undergoing percutaneous only technique. They found that the rerupture rate was 1.75% in the endoscopic group compared to 5.74% in the percutaneous only group; however, this difference did not reach statistical significance. A 2009 prospective series by Doral et al. including 62 patients with acute Achilles tendon ruptures repaired percutaneously with endoscopic assistance reported similar results [17]. In their series, 95% of patients returned to previous level of sport at a mean of 11.7 weeks. At mean follow-up of 46 months there were no reruptures, no wounds problems, or other complications. Fortis et al. echoed these results in a 2008 series of 20 patients with acute or chronic Achilles tendon ruptures in which endoscopically assisted repair of the Achilles was performed. All patients were reported to have good to excellent outcomes. However, two patients experienced sural neuralgia in this series. In 2018 Rungprai and Phisitkul reported a series of 23 consecutive patients that underwent endoscopically assisted percutaneous Achilles tendon repair using 4-strand core suture configuration via a 6-portal technique with good results [18]. At a mean follow-up of 54.1 months patients experienced a mean VAS improvement from 7.9 to 0.1, SF-36 improved from a PCS component score 32.5 to 44.7 and MCS from 47.9 to 51.4, and FAAM ADL from 26.1 to 83.0 and sports from 0 to 61.7. Only one superficial portal infection was reported in this series in a diabetic patient.

Pearce et al. reported a retrospective series consisting of 11 patients with noninsertional Achilles tendinopathy who underwent Achilles tendoscopy in conjunction with plantaris tendon release with a minimum follow-up of 2 years [19]. The authors reported that mean AOFAS hindfoot scores improved from 68 (range, 51–82) to 92 (range, 74–100) postoperatively. No complications were reported. Another retrospective series consisting of 24 patients who underwent paratenon debridement with longitudinal tenotomies of the Achilles tendon resulted in 96% of patients be symptom free at mean follow-up of 7.7 years following surgery. Two complications were reported in this series including a keloid scar as well as a seroma with chronic fistula [20]. In 2012, Lui published a small case series of five patients with noninsertional Achilles tendinopathy who underwent endoscopic Achilles tendon debridement with FHL transfer [21]. He demonstrated an increase in Achilles Tendinopathy Scoring System from 29.4 preoperatively to 89 at an average follow-up of 19.8 months with no complications. Vega et al. had previously reported a similar size series of eight patients with chronic Achilles tendinopathy defined as a minimum symptom duration of 3 months [1]. These patients underwent endoscopic debridement and were reported to all be pain free at a mean follow-up of 27.1 months (range, 18–40).

The use of gastrocnemius recession has been gaining popularity recently for expanding indications such as Achilles tendinopathy, diabetic forefoot ulcers, metatarsalgia, and plantar fasciitis [22]. Endoscopic, minimally invasive techniques to release the gastrocnemius have been described in the literature with promising results. Potential advantages over open techniques include decreased wound complications, improved cosmetic result, and diminished postoperative pain [13]. The largest case series (320 patients, 344 feet) of patients that underwent endoscopic gastrocnemius recession for isolated gastrocnemius contracture found a significant increase in mean ankle dorsiflexion, increasing from −0.8 ° to 11 ° at 13 months postoperatively [23]. SF-36 and FFI all increased significantly in these patients, mean VAS decreased from 7/10 to 3/10 postoperatively. However, 3.1% experienced subjective plantarflexion weakness and 3.4% in this series experienced sural nerve dysesthesia. A recent retrospective study comparing open vs. endoscopic release reported significantly lower complication rates following endoscopic release (26.8 vs. 2.6%) [24].

Endoscopic techniques have also been shown to be beneficial in treating insertional disease including posterior ankle impingement, subtalar arthritis, and retrocalcaneal bursitis [14]. Leitze et al. published a prospective series including 33 heels (30 patients) with chronic retrocalcaneal pain who underwent endoscopic decompression and compared results to a group of 17 heels (14 patients) in which an open technique was performed [25]. The endoscopic group reported AOFAS scores of 87.5 postoperatively compared to 79.3 in the open group; however, this did not reach statistical significance ($p = 0.115$). The endoscopic group experienced fewer complications including 3% vs. 12% infection rate, 10% vs. 18% sensory deficits, and 7% vs. 18% scar tenderness.

Based on the above data it appears that tendoscopy is a valuable tool that may be useful in assisting minimally invasive tendon rupture repair, noninsertional Achilles tendinopathy, retrocalcaneal bursitis, and gastrocnemius recession. While there remains a paucity of high-level evidence, current literature has shown good outcomes overall.

30.4 Flexor Hallucis Longus (FHL)

30.4.1 Indications

Tendoscopy has a role in the treatment of FHL related conditions such as stenosing tenosynovitis, tendon contracture, intratendinous ganglion cyst, bacterial tenosynovitis, synovial chondromatosis, loose bodies, and tendon transfer. The FHL tendoscopy is divided into three zones. Zone 1 is located at the posterior ankle just proximal to the opening of the tunnel underneath the sustentaculum tali [10]. Zone 2 is from the tunnel underneath the sustentaculum tali to the knot of Henry. Zone 3 is from the knot of Henry to the tendon insertion on the distal phalanx of the great toe. FHL symptoms related to posterior ankle impingement are usually treated with Zone 1 tendoscopy in conjunction with posterior hindfoot endoscopy, which will be discussed more extensively elsewhere.

30.4.2 Surgical Techniques

Positioning: Prone.

Instruments: 2.7 mm and 4 mm 30° endoscope, 4 mm shaver, Wissinger rod, retrograde knife.

The posterolateral portal is created at the level just distal to the tip of the lateral malleolus and just lateral to the Achilles tendon. The posteromedial portal for FHL tendoscopy is created at the level of intersection between the plantar aspect of the first ray and the medial border of the Achilles tendon. This posteromedial portal is slightly proximal compared to the posterolateral portal. The visualization and examination of the posterior ankle joint is routinely performed for posterior hindfoot endoscopy. The constricting fibrous tunnel at the entrance to the sustentaculum tali or low-lying muscle can be debrided with a shaver or an endoscopic scissors. A probe can be used to retract the tendon together with neurovascular structures medially allowing a part of zone 2 to be accessed from posterior portals.

The entire zone 2 tendoscopy of the FHL tendon is made possible with the establishment of a plantar portal. This portal is created inside out by inserting a Wissinger rod into the FHL tunnel to exit on the medial aspect of the arch of foot. This portal is associated with risks of nerve injuries as it is approximately only 5 mm from the medial plantar nerve [12]. Zone 3 tendoscopy requires a combination of the plantar portal and a plantar toe portal located on the plant aspect of the proximal phalanx of the great toe. This zone requires the use of 2.7 mm endoscope or smaller. A retrograde knife can be used to release tight fascial bands overlying the FHL tendon in cases of stenosing tenosynovitis in the distal aspect of the FHL tendon (Fig. 30.6).

30.4.3 Outcomes of FHL Tendoscopy

Tendoscopy has been studied for several different indications regarding the flexor hallucis longus tendon including FHL autograft harvest, release,

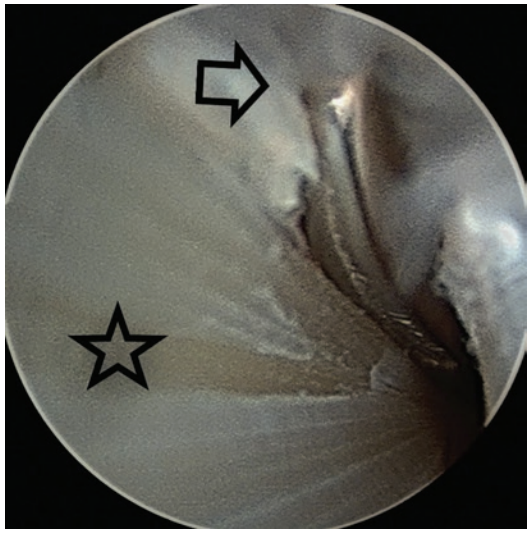

Fig. 30.6 Zone 3 tendoscopy of the FHL tendon (star) is shown with the patient in prone position. Constricting fibrous tunnel (arrow) is released using a retrograde knife

and debridement. However, most studies currently available are level IV and V evidence. The series are often mixed indications and include a variety of concomitant hindfoot procedures. FHL tendoscopy was first described by van Dijk for the treatment of chronic FHL tendonitis in an athlete by means of 2-portal posterior ankle endoscopy [26].

Corte-real et al. in 2012 utilized tendoscopy for FHL release in 27 patients [27]. The authors found good-to-excellent results in 19 out of 27 patients with a mean postoperative AOFAS score of 89. Complications including extensive fibrous tissue proliferation and transient medial calcaneal numbness were noted. Ogut et al. performed FHL tenolysis in a series of 59 patients and found an increase in mean AOFAS-hindfoot score improved from 56.7 to 85.9 with a complication rate of 3.4% including sural nerve irritation and neuroma in two patients [28].

Concomitant procedures commonly performed with FHL release include os trigonum excision, posterior ankle debridement, and possible posterior capsulectomy. Van Dijk published one of the largest series in 2006 including 146 procedures (FHL release, os trigonum, osteochondral drilling, etc.) with the majority of patients having good to excellent results [29, 30]. Only two complications were reported involving nerve irritation. Smith and Berlet in 2009 performed posterior ankle debridement, os trigonum excision, and FHL release in 14 patients with good to excellent results in 12 [31]. Two patients in this series had tibial nerve neuritis postoperatively.

Lui presented a case report including two patients in which FHL tendoscopy was performed for FHL tenosynovitis [32]. In this procedure he included a portal in the arch of the foot allowing access to zone 2 of the tendon. Both patients experienced paresthesia over the lateral sole and plantar fourth and fifth rays. Electromyography studies confirmed the diagnosis of lateral plantar nerve injury. Symptoms resolved in one patient by 5 months, and the other continued to experience symptoms 1 year later. In 2013 Lui published a retrospective series comprising five patients who underwent zone 2 FHL harvest for Achilles tendon augmentation and reported ATSS improved from a mean of 29.4 preoperatively to 89 postoperatively without complications [33].

30.5 Peroneus Brevis and Longus

30.5.1 Indications

Tendoscopy has been more extensively described to assess peroneal tendon conditions. Diagnostic endoscopy is an important tool to evaluate for pathologies such as tendon tears, dislocations, intrasheath subluxation, loose bodies, prominent peroneal tubercle, accessory tendons, and low-lying muscle [6, 9, 18, 23]. Visualization of the tendon conditions allows surgeon to be more precise in the placement of an open incision if needed. The ability of peroneal tendoscopy in the definitive treatment of various pathologies is growing as experienced endoscopists could excise torn tendons, low-lying muscle, peroneus quartus tendon, and groove deepening of the distal fibula.

30.5.2 Surgical Techniques

Positioning: Supine with a bump underneath the ipsilateral buttock.

Instruments: 2.7 mm and 4 mm 30° endoscope, 3.5 mm shaver, 4 mm barrel bur.

A proximal portal is created 2 cm proximal to the tip of the lateral malleolus at the soft spot behind fibula bone. A 2.7 mm trocar is inserted into the peroneal tunnel along the posterior aspect of the distal fibula. The distal portal is created inside out at the location 2 cm distal to the tip of the lateral malleolus and along the line of peroneal tendons. A probe is inserted into the distal portal to assist with evaluation for pathologies. Surgeons should look for tears in the peroneal tendons, especially split tears of the peroneus brevis, contour of the peroneal groove at the distal fibula, integrity of the superior peroneal retinaculum, and low-lying peroneus brevis muscle (Fig. 30.7). Dynamic examination is performed by observing the peroneal tendons during range of motion from inversion to eversion and vice versa. Debridement of partial tendon tears, synovitis, peroneus quartus tendon, and low-lying muscle is performed using a 3.5 mm shaver. Groove deepening procedure is performed preferably with a combination of 4 mm 30° endoscope and a 4 mm barrel bur. The groove should be approximately 10 mm in width, 6 mm in depth, and 15 mm in length but the extent of groove deepening should be individualized depending on the anatomy of each patient. It is critical that the very distal aspect of the groove is contoured aggressively to avoid abrasion of peroneal tendons as they are directed more anteriorly.

Access to the peroneal tubercle is achieved by using the distal portal as described earlier and adding an accessory portal 1.5 cm distal to the peroneal tubercle and along the peroneus longus tendon. To remove the prominent peroneal tubercle, the septum between the peroneus longus and brevis tendons must be detached using a shaver or an endoscopic scissors. The bone is then the compressed using a 4 mm barrel bur until there is no impingement to the tendons. The two portals can be used interchangeably for visualization and instrumentation. Visualization of the distal portion of peroneus longus tendon on the plantar aspect of the foot can be facilitated by using an accessory portal on the lateral aspect of the cuboid.

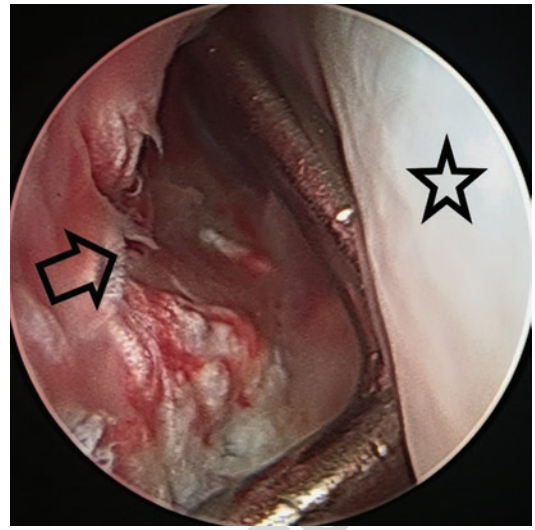

Fig. 30.7 Peroneal tendoscopy of a runner with a severe ankle sprain demonstrates a rupture of the superior peroneal retinaculum. The peroneus brevis tendon (star) is intact

30.5.3 Outcomes of Peroneal Tendoscopy

Tendoscopy procedures allow for minimally invasive treatment of peroneal tendon pathologies with reduced wound complications and scarring. Overall outcome studies for peroneal tendon endoscopic procedures are difficult to generalize given the heterogeneity of pathologies and lack of level I and II evidence [10–12]. Most pathologies fall into three categories: (1) tenosynovitis and tendinitis, (2) subluxation and dislocation, and (3) tendon tear and rupture.

Van Dijk was the first to report on the outcomes of posterior endoscopy for peroneal tendinitis [34]. In his series of nine patients with retromalleolar pain, he found that at a mean follow-up of 19 months, 8 out of 9 patients were symptom free without complications. Jerosch et al. studied 15 patients that underwent peroneal

tendoscopy between 1999 and 2004 for tenosynovitis (seven), low-lying muscle belly of the peroneus brevis (two), and peroneal tendon instability (one), and partial peroneal tears (five). At a mean follow-up of 2.8 years, all patients were asymptomatic and were able to participate in moderate athletic activities [35]. Vega et al. reported the outcomes of 52 patients with heterogenous peroneal tendon pathologies undergoing tendoscopy debridement and peroneal groove deepening from 2008 to 2011 [36]. Their cohort included peroneal tendon ruptures (24 patients), tenosynovitis (13 patients), recurrent peroneal tendon subluxation (7), intrasheath subluxation (6), and adhesions (2). Patients with distal peroneal tendon tears underwent a min-open repair. They found that at a minimum 1 year, intrasheath subluxation patients had 100% excellent results (mean AOFAS score increased from 79 to 99). In the recurrent peroneal tendon subluxation group, 5 out of 7 patients (71.4%) had excellent results (AOFAS score increase from 75 to 93 postoperatively). The peroneal tendon rupture group only had a 62.5% symptom free rate, with 12.5% of patients reporting no change in symptoms.

Outcomes for treatment of isolated tenosynovitis without subluxation has been described in the literature. Scholten and van Dijk assessed 23 patients that underwent peroneal tendoscopy for tenosynovitis with a minimum follow-up of 2 years. Their results showed that there were no complications or recurrence of pathology [3]. More recently, Kennedy et al. reviewed 24 consecutive patients (mean age 34 years) with isolated peroneal pathology that underwent peroneal tendoscopy at a single institution [37]. All cases received platelet-rich plasma as a biologic augment. At a mean follow-up of 33 months, the mean foot and ankle outcome score (FAOS) and short form-12 (SF-12) improved from 57 to 86 and 54 to 81, respectively. Nine patients underwent endoscopic peroneal groove deepening. Two patients with greater than 10 mm tears underwent mini-open tubularization procedures. This series mostly comprised of patients with tenosynovitis and lacked patients with subluxating peroneal tendons. Lui studied seven patients retrospectively with isolated peroneal tenosynovitis that underwent endoscopic peroneal groove deepening for retrofibular pain [38]. At a mean 24 months follow-up, 6 out of 7 patients (86%) returned to sporting or job activities.

Peroneal tendon subluxation represents another subset of individuals with peroneal tendon pathology. Peroneal groove deepening has been advocated to reduce pressure on peroneal tendons to facilitate healing. Edwards et al. showed that there is a wide variation of groove depth, ranging from 0 mm to 3 mm deep [39]. However, he also found that 11% of patients have a flat grove and 7% have a convex groove. A cadaveric study performed by Schon et al. showed that deepening the peroneal groove by 6 mm reduced tendon pressure readings along the middle and distal aspects of the peroneal groove [40]. Vega et al. followed seven patients with chronic peroneal tendon subluxation that underwent tendoscopic deepening of the peroneal groove without superior peroneal retinaculum repair. Four patients had complete disruption of the superior peroneal retinaculum which also underwent repair. At a mean follow-up of 15.4 months, no patients had recurrent subluxation and the mean American Orthopaedic Foot and Ankle Scores (AOFAS) improved from 75 to 93 postoperatively. There was one patient that continued to have subjective clicking without frank subluxation of the tendons.

Intrasheath subluxation represents a group of patients that present with retromalleolar clicking without reproducible tendon dislocation [15]. Raikin et al. reported 14 patients with intrasheath subluxation and intact superior peroneal retinaculum confirmed on ultrasound. Type A intrasheath subluxations occurred when the anatomic position of the peroneus longus and brevis switched at the peroneal groove with resisted dorsiflexion and eversion. Type B intrasheath subluxations occurred when the peroneus longus subluxed through a longitudinal tear in the peroneus brevis tendon. Recently, Guelfi et al. described a new subset of patients with combined intrasheath peroneal tendon subluxation and concomitant superior peroneal retinaculum injury [16]. These patients present with snapping at the peroneal tendon without frank subluxation. The

authors retrospectively followed 18 patients (mean age 29 years) undergoing tendoscopy for a mean follow-up of 45 months. They found that twelve patients had a space occupying lesion and underwent debridement. Six patients were found to have a superior peroneal retinaculum injury and underwent a peroneal groove deepening without superior peroneal retinaculum repair. At the final follow-up, the mean AOFAS scores improved from 76 preoperatively to 97 postoperatively. Additionally, there were no reported recurrences among the cohort.

Lastly, peroneal tendoscopy can be used in conjunction with ankle and subtalar arthroscopy [17]. Bare and Ferkel found that in 30 patients undergoing peroneal tendon procedures, 100% of patients had at least one intra-articular ankle derangement during arthroscopy [19]. In Bojanic's series, 8 out of 13 tendoscopy procedures were performed in conjunction with ankle arthroscopy or open procedures. At 1 year follow-up, all patients did well without pain or clicking [17].

30.6 Other Tendons in the Foot and Ankle

Tibialis posterior tendinitis is commonly seen in early stage planovalgus deformity [20]. Tenosynovitis of the tibialis posterior tendon can present with medial hindfoot pain at the navicular. Over time, the diseased tendon can rupture and lead to attenuation of the spring ligament and medial longitudinal arch collapse. Common nonoperative modalities include orthotic wear and activity modification. When nonoperative treatment fails, tendoscopy debridement can provide pain relief and improved function.

The results of tendoscopy for tibialis posterior tendinitis are favorable. Before endoscopy, Johnson and Teasdall reported 90% good results after open synovectomy of the posterior tibialis tendon [21]. In 1995, Wertheimer was the first to describe tendoscopy treatment of posterior tibial tendon dysfunction [1]. Van Dijk soon reported his technique for tendoscopy debridement of the posterior tibialis tendon [41]. In van Dijk's series of 200 patients that underwent hindfoot arthroscopy, 31 patients undergoing tendoscopy for debridement and vinculum removal had good results [29]. In his series, partial repairs underwent a mini-open procedure. Chow's case series of six patients with stage I posterior tibial tendon dysfunction did well with tendoscopic synovectomy without progression to stage II disease [42]. These patients returned to work at 10 weeks and resumed sports at 6 months postoperatively. Khazen et al. performed tendoscopy for nine patients with stage I posterior tibialis tendon dysfunction, including three open tendon repairs for partial tears [43]. They found that patients with isolated tenosynovitis returned to work by 6 weeks and patients with tendon tears returned to work by 10 weeks. Bernasconi et al. reported 16 patients with stage II posterior tibialis tendon deficiency treated with tendoscopy. At a mean of 25.6 months, VAS pain and SF-36 mental component scores significantly improved [44]. Eighty percent of patients were relieved of symptoms. However, three patients underwent subsequent open calcaneal osteotomy and posterior tibialis tendon augmentation procedures. Most of these patients were found to have severe spring ligament injuries.

Although rarely utilized, tendoscopy debridement of the tibialis anterior tendon and extensor tendons has been reported in the literature [45, 46]. Irritation along the tibialis tendon can arise from overuse, inflammatory conditions, or infection [47–49]. The utilization of tendoscopy around the tibialis tendon and extensor tendon is riskier than other location given the close proximity of various neurovascular structures. Care must be taken not to debride the extensor retinaculum to prevent bowstringing of the extensor tendons [50]. Also, avoiding debriding the posterior aspect of the tibialis anterior tendon will prevent disruption of its blood supply as it enters the paratenon from the medial tarsal artery [51]. There have also been case reports of pseudoaneurysms of the dorsalis pedis artery following tendoscopy of the extensor tendons [49, 52, 53].

Lui reported in 2005 a small case series of three patients that underwent tendoscopic

debridement of the flexor digitorum longus for metatarsalgia and flexor tenosynovitis. One patient presented after a post-infective tenosynovitis from a previous penetrating injury. The other two patients presented with focal idiopathic flexor tenosynovitis. At 2-year follow-up, all three patients had resolution of their metatarsalgia without any complication [54].

30.7 Conclusion

Since the first description of tendoscopy more than two decades ago for the posterior tibial tendon, indications for this novel technique have expanded to other tendons including the Achilles tendon, flexor hallucis longus, peroneal longus and brevis, tibialis anterior, flexor digitorum longus, extensor hallucis longus, and extensor digitorum longus [1]. Techniques have been refined since the original description as well as scope technology. Overall, good outcomes have been reported in the literature for the use of tendoscopy. Advantages of tendoscopy include quicker recovery time and superior cosmetic result compared to open procedures. As with most orthopedic literature, there is a paucity of high-level evidence supporting the use of the procedure, and currently most of evidence has been reported by highly experienced arthroscopists. Therefore, it is important to take endoscopic experience into account when interpreting the evidence for this procedure.

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Insertional Achilles Tendinopathy

31

Gian Luigi Canata and Valentina Casale

31.1 Introduction

Insertional Achilles tendinopathy is a painful and debilitating condition, representing among one-third of all Achilles tendon pathologies [1]. Patients of all ages and activity levels may be affected, especially running athletes [2].

Overuse injuries of the Achilles tendon must be distinguished between insertional (at the calcaneus-Achilles tendon junction) and non-insertional (2–6 cm proximal to the calcaneus-Achilles tendon junction) tendinopathies [3, 4]. Among the insertional Achilles tendinopathies, a clear distinction should be made between the Haglund disease and all the other insertional pathologies. In fact, the term Haglund's exostosis is commonly referred to a clinical assessment characterized by pain and tenderness usually at the postero-lateral side of the calcaneus, where a calcaneal prominence can often be felt [3].

A further classification of the Achilles tendon disorders marks a clear difference between insertional tendinopathies, pre-insertional tendinopathies (or retrocalcaneal bursitis, an example is the Haglund's exostosis), and superficial calcaneal bursitis [5].

The Haglund disease and the other conditions, as the superficial Achilles bursitis, are different in terms of causes, histopathology, prognosis, and treatment [3] (Fig. 31.1).

Insertional Achilles tendinopathy usually occurs in active people, whereas non-insertional injuries are more frequent among older, less active, and overweight people [2]. In a large retrospective study, Kvist et al. reported that 66% of competitive and recreational athletes had non-insertional tendinopathy, while 23% had either retrocalcaneal bursitis or insertional Achilles tendinopathy [6]. A more recent study conducted by Mansur found that 16% of the active people presenting this condition end up quitting sports activity [7].

The insertional Achilles tendinopathy is located at the insertion of the Achilles tendon onto the calcaneus, often associated with the formation of bone spurs and calcifications at the insertion site; the pain is mostly limited to the midportion of the posterior aspect of the calcaneus, where the bone spur may be palpable; the histopathologic process consists in an ossification of the enthesial fibrocartilage, and small tendon tears may occur at the tendon-bone junction [3].

Radiographic findings of the Achilles tendon disorders appear differently according to the various pathologic conditions [3].

In fact, ultrasonography (US) and CT scan are helpful for evaluating patients with insertional Achilles tendinopathy to show the presence of ossification or bone spurs at the tendon insertion, with or without osteophytes along the tendon, calcaneal bony alterations and possible focal lesions

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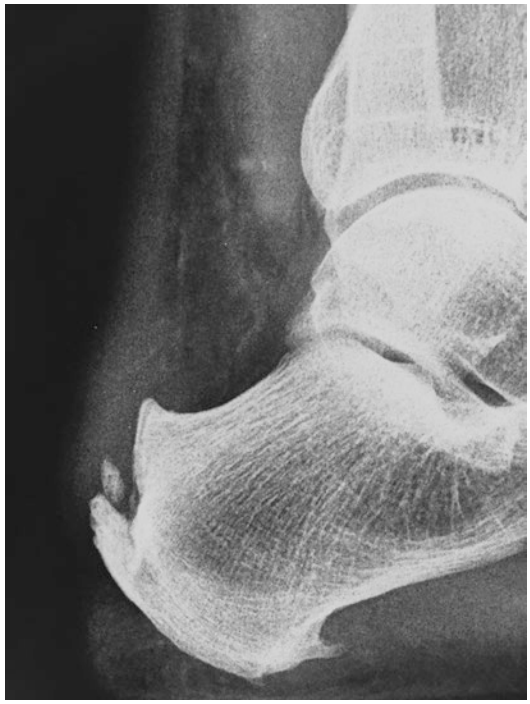

Fig. 31.1 Haglund calcaneal prominence and calcific insertional tendinopathy

within the tendon. CT scan specifically reveals the bone formation and its details, whereas magnetic resonance imaging (MRI) provides hyperintense signals at tendon insertion [3, 8].

Insertional Achilles tendinopathy causes intense heel pain, especially in the morning, and it is exacerbated by activity [9]. Patients classically experience pain and swelling along the distal tendon insertion into the calcaneus [10]. Symptoms are more intense during exercise, when ascending stairs and running on hard surfaces.

Clinically, there may be a palpable point of tenderness and swelling on the posterior aspect of the calcaneal tuberosity, as well as a prominent calcaneal exostosis [11]. Thickening of the Achilles tendon may be present in case of chronic inflammation [12].

31.2 Pathophysiology

The etiology of the insertional Achilles tendinopathy is multifactorial and several predisposing factors have been proposed. Intrinsic risk factors include hyperpronation, pes cavus, leg length discrepancy, limited mobility of the subtalar joint, as well as advancing age, obesity, diabetes, hypertension, and use of steroids, estrogens, and fluoroquinolone antibiotics [13–16]. The extrinsic predisposing factors are associated to changes in training pattern, footwear, and running on smooth, hard, and sloping surfaces [6, 17].

A few studies analyzed the biomechanical causes of this pathology; Maganaris et al. reported that the affected site is usually "stress shielded" [18]. When inflammation is prolonged, the bursa may become fibrotic and reduces its lubricating function [19]. Repetitive traumas determine cartilage-like changes within the Achilles tendon, consequently leading to intra-tendinous bone formation through endochondral ossification [20].

Among the many theories developed over the years, Benjamin et al. reported that the ossification process at the tendon insertion would not depend on previous traumas and inflammations, rather on an adaptive structural change. As the bone-tendon junction surface increases, the tendon stands higher mechanical loads [21].

It has been demonstrated that the site of tendon degeneration is characterized by irregular-sized tenocytes that are likely to develop apoptosis [2, 22]. The result is a chronic mucoid and/or lipid tendon degeneration, with potential fibrocartilaginous metaplasia and calcium hydroxyapatite deposits [23–25].

Tendons are relatively avascular. Thereby, neovascularization becomes the hallmark of a chronic inflammation usually associated with the presence of mechanoreceptors and nerve-related components [26, 27]. A retrocalcaneal bursitis must be suspected if the patient complains of pain and swelling anteromedially and anterolaterally to the Achilles tendon [2].

31.3 Imaging

It is widely assumed that insertional Achilles tendinopathy is clinically diagnosed; nevertheless, radiological imaging may be helpful to better define the clinical assessment, as well as for the preoperative planning if surgery is required [28].

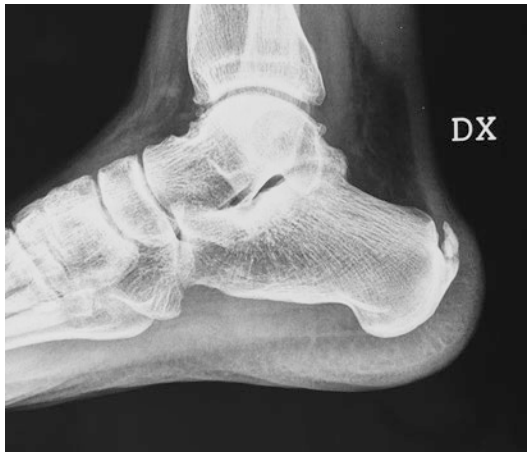

Fig. 31.2 Radiological image of insertional calcific Achilles tendinopathy

Plain weight bearing radiographs of the foot are usually the first exam to be performed [28]: any alteration of the medial longitudinal arch of the foot may be identified through the anteroposterior and the lateral views, and the presence of intra-tendinous calcifications or bony exostosis can be detected [2] (Fig. 31.2).

Both MRI and US provide additional information to distinguish the different structural abnormalities detectable at the Achilles tendon insertion [1]. Nevertheless, it must be considered that the extreme sensitivity of MRI may also identify structural abnormalities that are not strictly related to clinical symptoms [29–31].

31.4 Treatment

31.4.1 Nonoperative Management

In the acute phase, an initial period of rest or immobilization is advisable, associated with modified activity [2].

Other conservative options include stretching exercises, extracorporeal shock wave therapy, the use of non-steroidal anti-inflammatory drugs, orthotics, and shoe modification: in particular, heel lifts contribute to a consistent reduction of the Achilles tendon tension [12, 32].

Eccentric training, through which the tenon is lengthened during simultaneous muscular contraction, has not showed significant results for insertional Achilles tendinopathies, although this option has been proved to be effective in case of non-insertional pain [2, 33]. The key factor resides in the paratendon oxygen saturation: changes in tendon and paratendon microcirculation are well documented in insertional and midportion tendinopathy. The paratendinous postcapillary filling pressure usually increases at the superficial tendon insertional area [34]. The eccentric-training program may reduce both the augmented paratendinous capillary blood flow and the pain [34].

Whereas the eccentric training program for insertional tendinopathies does not guarantee the same good results achievable in other tendon locations, several alternatives have been proposed: infiltrations, electrostimulation, sclerotherapy, and others aim to stimulate healing process to the degenerated tendon [7]. Nevertheless, the low rate of success has led clinicians to promote recovery by angiogenesis enhancing and diffusion of cytokine molecules [7].

Extracorporeal shockwave therapy (ESWT) actually stimulates neovascularization and angiogenesis at the tendon-bone junction, also causing epidermal nerve fibers degeneration and promoting reinnervation [35–38]. Furia et al. reported the effectiveness of this treatment, compared with other conservative management strategies. However, they suggested to avoid a local anesthesia field block before the application of the shock waves, as it could negatively influence treatment effectiveness [13].

Ultrasounds can be also used as a treatment approach: by reducing swelling and pain in the acute phase and increasing function in chronic tendinopathies, they enhance tendon healing process [39, 40]. High quality data are still needed to confirm the efficacy of this treatment option [41].

Some authors propose local injections into the retrocalcaneal space, avoiding the use of corticosteroids and local anesthetics; the risk is to weaken the tendon tissue and to expose the tendon to rupture [42]. It has been also reported by Kleinman that local steroid injections can increase the risk of an acute tendon tear [43].

31.4.2 Surgical Treatment

Insertional Achilles tendinopathy is a distinct clinical entity from the pre-insertional tendinopathy with retrocalcaneal bursitis and both are different from the midsubstance tendinopathy not only concerning etiology and injury mechanism, but also for treatment and rehabilitation ([3, 8, 14, 44]).

Patients who do not respond to conservative management may require surgery. Surgical treatment has been increasingly performed over the last decades.

Most clinicians wait at least from 3 to 6 months before proceeding with surgery [4]. Surgical procedures include tendon debridement, enthesiophyte resection, gastrocnemius elongation, and the posterior superior calcaneal eminence removal in case of concomitant pre-insertional symptoms [2, 8, 44–46].

In a recent review, two main categories of surgical treatment have emerged: debridement alone and debridement combined with tendon augmentation in case of excessive tendon loss [47]. However, there is no specific evidence about which surgical technique may guarantee better postoperative results.

The main goal is to remove the degenerative tissue and its associated ossification and, if needed, the posterior calcaneal eminence, in addition to the retrocalcaneal spur resection [48]. After suturing the detached Achilles tendon fibers, tendon insertion reattachment and tendon augmentation may be subsequently required [49].

Debridement of the degenerative tendon tissue associated with osteotomy is known to be effective for patients younger than 50 years, with moderate tendon involvement [46, 50]. In elderly patients, persistent pain and limited function have been reported as possible postoperative problems, probably due to a limited vascularization and a lower capacity for full recovery [51].

In rare cases, some authors propose the addition of the flexor hallucis longus (FHL) tendon transfer to support and protect the debrided Achilles tendon [51–54]. The result is an improved plantarflexion strength and a more effective healing capacity, especially if the debridement of the Achilles tendon includes a complete tendon detachment [2] that should be avoided, whenever possible. As tendon reattachment may potentially predispose to risks of tendon ruptures [28], this surgical technique is advisable only if more than 50% of the Achilles tendon has been detached [52]. However, a wide consensus among surgeons has not been reached yet.

Some authors have described the surgical management of calcific insertional tendinopathy with a lateral, medial, or midline skin incision [55], as well as J-shaped one [45], eventually followed by a partial or full-thickness Achilles detachment from its insertion; however, no significant clinical differences have been reported between partial or no tendon detachment and a complete tendon detachment [45]. According to some authors, a central tendon splitting incision may be useful to better remove the degenerative tissue and the calcifications [10, 46, 55]. The real extent of calcifications may not be appreciated through medial and/or lateral approaches, because they occur within the middle third of the degenerative tendon insertion in 95% of cases [46].

A further example of skin incision is the transverse Cincinnati approach, described by Maffulli et al. [56]. The retrocalcaneal bursa may be resected to prevent postoperative recurrent pain [12].

The open surgical approach may cause postoperative complications, such as wound dehiscence and infection, sural nerve damage and tendon necrosis scarring, especially if the classic longitudinal extensile approach is performed [57]. In order to reduce the frequency of these events, some authors have proposed less invasive approaches, like the percutaneous technique ([58–63]).

Surgical techniques focused on the treatment of each specific pathology are clearly less invasive and ease rehabilitation processes. Pre-insertional pathologies may be addressed with a mini-open lateral access or endoscopically while insertional calcifications and the debridement of the degenerative tissue can be managed through a direct Achilles tendon splitting (Figs. 31.3a,b, 31.4, 31.5). Moreover, given the blood supply of the tendon is via the paratenon, a lateral or medial incision may disrupt the blood supply [10, 46, 55, 64].

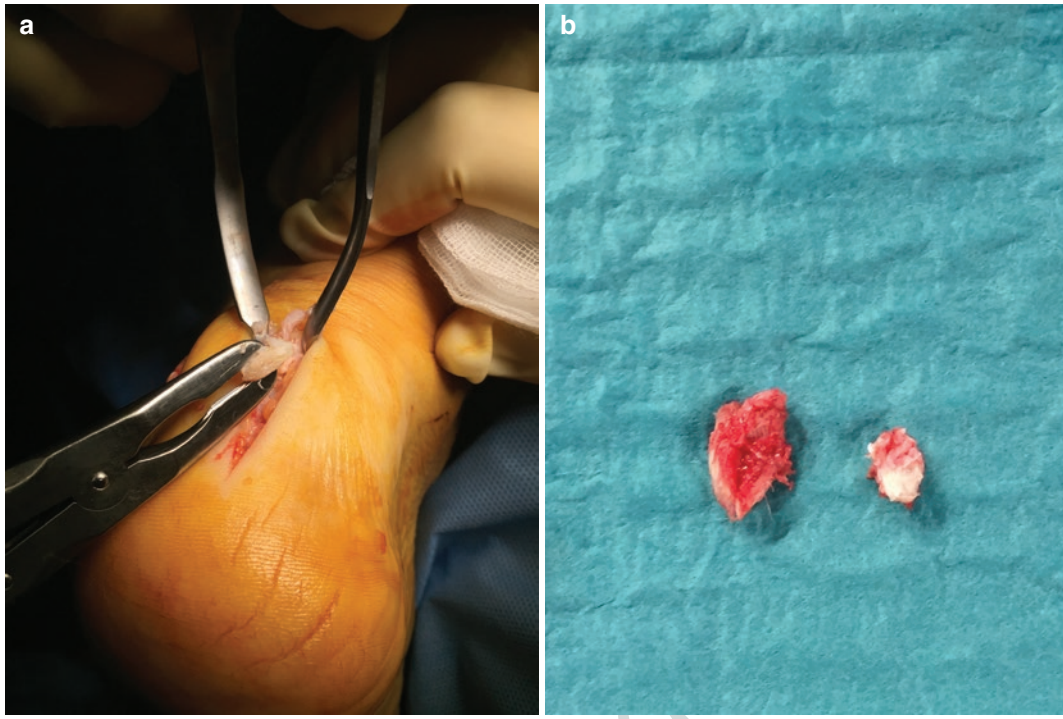

Fig. 31.3 Calcific insertional body excision (**a**); calcific bodies are removed (**b**)

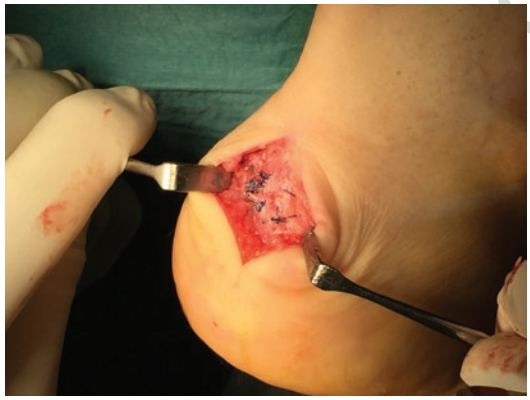

Fig. 31.4 Achilles tendon suture after central tendon splitting for a double calcification

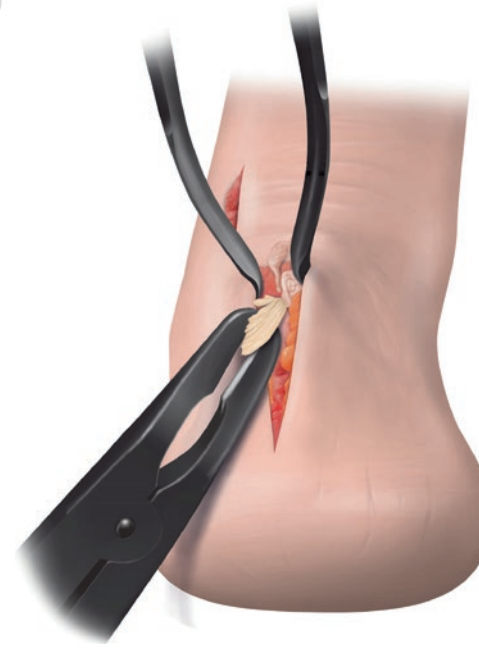

Fig. 31.5 Calcific insertional tendinopathy: Achilles tendon splitting and excision of the bony fragment. At the upper left, the incision for a concomitant Haglund deformity

An accurate preoperative clinical and radiological evaluation is important for the exact localization of the calcific area and allows a direct and less invasive surgical approach.

If an Haglund's deformity is present, the aim is to remove the painful bony prominence of the posterosuperior corner of the calcaneus, as well as

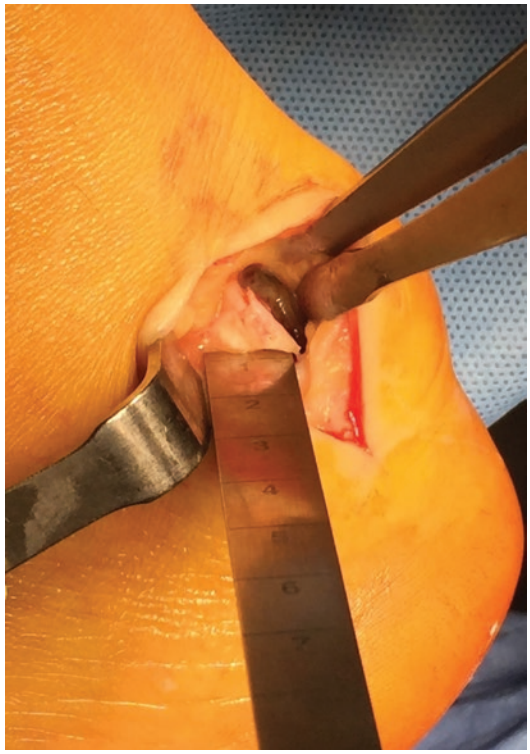

Fig. 31.6 Mininvasive calcaneoplasty for Haglund deformity

to debride the diseased tendon, if necessary, and to excise the inflamed bursal tissue [65]. The surgeon must pay attention not to damage the tendon insertion when removing the bony prominence [66] (Figs. 31.6, 31.7, 31.8). Recently, the endoscopic, the percutaneous, and the mini-open calcaneoplasty are the most described approaches [67–72].

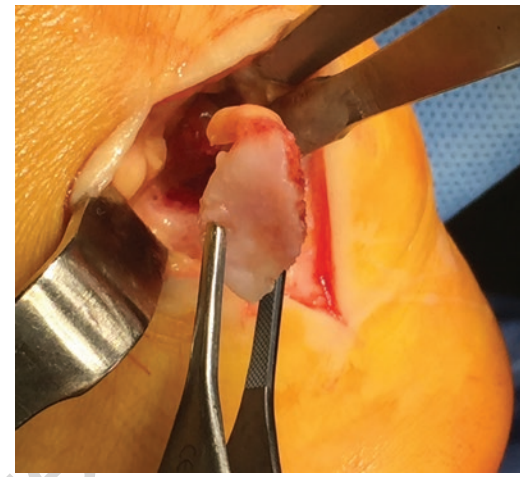

Fig. 31.7 The fragment excised

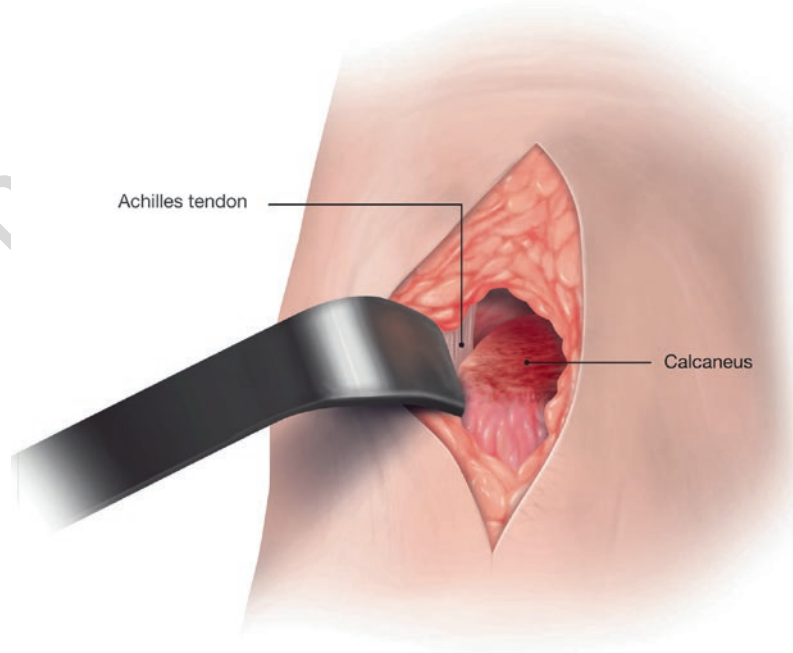

Fig. 31.8 Mini-open technique for Haglund's deformity: bony prominence resection sparing the Achilles insertion

31.5 Postoperative Management and Rehabilitation Program

After surgery, some authors recommend a cast for 6 weeks to guarantee a complete anchoring of the tendon to the bone interface [12]; others suggest an ankle immobilization for 2 weeks or more, followed by a weight-bearing plantarflexed cam walker boot or cast for an additional 3–4 weeks [10]. A removable boot brace is usually recommended for 1 month, starting rehabilitation at the same time, especially to restore an appropriate plantar flexion [55].

The postoperative regimen depends on the degree of the structural involvement and the consequent surgery adopted: in case of paratenonitis, as well as retrocalcaneal bursitis, the range of motion may be immediately instituted and a boot walker is recommended for the first weeks [8]. When the area of tendinosis is extensive, a short-leg cast may be initially placed, and weight-bearing should be limited for the first 4–6 weeks [8].

The postoperative protocol may depend on the physician's confidence in the tendon reattachment, as well as on the portion of the tendon removed: if less than 50% has been excided, an early weight-bearing may be allowed [10].

Physical therapy should be focused on gait training, gradual ankle range of motion recovery, and a progressive gastrocnemius-soleus strengthening program [10].

A full recovery of the range of motion and muscle strength is usually reached after from 6 weeks to 1 year [12].

31.6 Conclusions

Insertional and pre-insertional Achilles tendinopathy are a painful and debilitating condition. When necessary, surgical treatment should be carefully planned, to restore function properly.

Rehabilitation depends on the specific surgical technique performed and on the individual healing time. Less invasive surgery may allow an earlier return to desired daily and sport activities.

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32 Non-insertional Achilles Tendinopathy: State of the Art

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32.1 Anatomy of the Tendon

The Achilles tendon is formed by the confluence of the gastrocnemius and soleus muscles. The soleus lies deep to the gastrocnemius muscle, arising from the posterior surface of the upper tibia. The tendon inserts on the posterior surface of the calcaneus, distal to the postero-superior calcaneal tuberosity. The Achilles tendon is not encased in a true synovial sheath, but is surrounded by paratenon, which is composed of a single layer of cells. The paratenon is highly vascularised, and it is responsible for the tendon's blood supply [1], through a series of transverse vincula which reach the tendon and act as passageways for vessels. The Achilles tendon also receives blood from vessels arising at the musculo-tendinous and osteo-tendinous junctions. Healthy tendons are brilliant white, with a fibroelastic texture. The rotation of the tendon begins about 12–15 cm proximal to its insertion, becoming more marked in the distalmost 5–6 cm. The tendon spirals approximately 90°, with the medial fibres rotating posteriorly, and the posterior fibres rotating laterally. Angiographic injection techniques have demonstrated a zone of hypovascularity 2–7 cm proximal to the tendon insertion.

The number of intra-tendinous vessels, and the relative area occupied by them, is lowest 4 cm from the calcaneal insertion [1].

Within the extracellular matrix network, tenoblasts and tenocytes constitute 90–95% of the cellular elements of tendons. The remaining 5–10% consists of fibrochondrocytes, synovial cells of the tendon sheath, and endothelial cells and smooth muscle cells [2]. Collagen type 1 accounts for 65–80%, while elastin accounts for about 2% of the dry mass of tendons. Tenocytes and tenoblasts lie between the collagen fibres along the long axis of the tendon [2].

Tendon innervation arises from three main sources:

- Cutaneous nerve trunks.
- Muscular nerve trunks.
- Peritendinous nerve trunks.

Nerve fibres cross and enter the endotenon septa at the musculo-tendinous junction. Nerve fibres penetrate the epitenon from plexuses in the paratenon. Most nerve fibres do not actually

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enter the main body of the tendon, but they terminate as nerve endings on its surface. Nerve endings of myelinated fibres function as specialised mechanoreceptors to detect changes in pressure or tension. Unmyelinated nerve endings act as nociceptors, sensing and transmitting pain. Both sympathetic and parasympathetic fibres have been identified in tendons [3]. Autonomic peptides such as neuropeptide Y and vasoactive intestinal peptide, which regulate vasoactivity, act in tendons [3, 4].

Tendons transmit force generated by muscle to bone. They also act as a buffer, by absorbing external forces to limit muscle damage: this function requires mechanical strength, flexibility and elasticity [2]. As collagen fibres deform, they respond linearly to increasing tendon loads [5]. The configuration is initially lost when the stretch exceeds 2%, but is re-gained if the strain placed on the tendon remains at less than 4%. If strain exceeds 8%, macroscopic rupture will occur [6, 7]. The tensile strength of tendons is related to this thickness and collagen content: a tendon with a cross-sectional area of 1 cm^2 is capable of supporting 500–1000 kg. Loading of the Achilles tendon reaches up to 9 kN during running (corresponding to 12.5 times the body weight), 2.6 kN during slow walking and less than 1 kN during cycling [8].

32.2 Introduction

Non-insertional Achilles tendinopathy is the commonest pathological condition, representing between 55% and 65% of disorders [6, 9, 10]. The term 'tendinosis' was used by Puddu et al. [11] in 1976 to describe the histological changes, which include loss of the normal collagenous architecture and replacement with an amorphous mucinous material, hypercellularity, increased glycosaminoglycans and neovascularisation [12–14]. It was previously thought that inflammation was not an important factor in the condition [12, 15]; however, recently the importance of inflammation in the pathological process has been re-evaluated, and the inflammatory process may be a contributory factor to the development of tendinopathy [16, 17].

The term 'tendinopathy' is preferred to the previously used term 'tendinitis' because does not convey the concepts of inflammation or degeneration [18, 19]. In tendinopathy the essential lesion is failed healing response [20].

The incidence of Achilles tendinopathy has been reported to be as high as 37.3 per 100,000 in some European populations [21–23]. This condition is the result of intrinsic and extrinsic factors which contribute to the development of non-insertional Achilles tendinopathy [24]. The first ones are age, body habitus, nutrition, metabolic diseases, genetic, lower limb malalignment, leg length discrepancy [25, 26] and limited ankle dorsiflexion [27, 28]; the others are training errors, drugs including steroids and fluoroquinolones [29, 30], compression, disuse, and excess loading [31].

Among the metabolic diseases, the most important is dyslipidaemia, but measurement of cholesterol in patients presenting Achillodynia does not seem to be justified. The literature nevertheless suggests that it should probably be considered in patients with bilateral extensive disease or those who give a history of intermittent episodes of severe Achilles tendon pain lasting a few days [32].

Another retrospective study also found various statistically significant associations between tendinopathy and diabetes mellitus, obesity and hypertension [33].

32.3 Clinical Presentation

Achilles tendinopathy has been described in association with many different sporting activities, but middle and long-distance runners have the greatest susceptibility to it [10, 24, 34–36]. The annual incidence in high-level club runners was between 7% and 9% [37]. Tendinopathy typically occurs between 2 cm and 6 cm from the insertion of the Achilles tendon into the calcaneus [20].

The major symptom in non-insertional Achilles tendinopathy is pain, which can markedly interfere with function and athletic activity. The pain is common on first moving after a period of rest and it is most intense. A diagnosis of Achilles

tendinopathy is usually clinical on the basis of history and presentation. Patients often present pain and swelling on the posteromedial aspect of the tendon, and tenderness can usually be elicited with palpation over the swelling [31, 38].

The origin of pain in tendinopathy of the main body of the Achilles tendon is, however, controversial [39–41]. Abnormal neoinnervation often accompanies the neovascularisation, and is thought to play a central role in the development of pain [14, 42–44].

32.4 Diagnosis

32.4.1 Clinical Diagnosis

In addition to the swelling on the posteromedial aspect of the tendon and palpation pain, some clinical tests have been described for non-insertional Achilles tendinopathy diagnosis. They can be divided into palpation tests (tendon thickening, crepitus, pain on palpation, the Royal London Hospital (RLH) test, the painful arc sign) and tendon loading tests (pain on passive dorsiflexion, pain on single heel raise and pain on hopping).

The painful arc sign, in which a painful swelling moves with ankle movement, indicates tendinopathy rather than paratendonitis [45]. Less pain of the swelling in ankle dorsiflexion is indicative of tendinopathy with the RLH test [46]. Maffulli et al. studied sensitivity, specificity, reproducibility, and predictive value of palpation of the painful arc sign and of the Royal London Hospital test in 10 patients with Achilles tendinopathy and in 14 asymptomatic subjects, and found no evidence of a difference of the three assessment methods ($p > 0.05$); when the test were combined, the overall sensitivity was 0.586, and the overall specificity was 0.833 [47].

Hutchinson et al. in 2013 studied the ten clinical tests mentioned above and found that two tests (location of pain and pain to palpation) are sufficient and accurate for clinical use [46]. A 2014 meta-analysis concluded that the most appropriate clinical reference standard for diagnosis of Achilles tendinopathy needed further investigation [48].

32.4.2 Instrumental Diagnosis

Imaging techniques, including ultrasound (US) and magnetic resonance imaging (MRI) scans, can occasionally be useful to identify the nature, location and extent of a lesion[49, 50]. US may be particularly useful with the addition of power Doppler, because the pain in Achilles tendinopathy seems to be related to areas of neovascularisation [14, 44, 51]. Neonerves grow into the tendon with the new vessels, and these can transmit the pain of the tendinopathy [52–54]. Treatment modalities which reduce the amount of neovascularisation can lead to a reduction in symptoms [52, 53, 55]. Equally, treatments that have proven to be clinically effective have subsequently been shown to reduce neovascularisation within the tendon, although the evidence for this hypothesis has recently been challenged [39, 56]. Ultrasound may also be used to guide the various injection therapies available.

Few studies have compared ultrasound with MRI in the diagnosis of Achilles tendinopathy. Early studies seem to indicate that MRI scans are better for characterising degeneration in the Achilles tendon [57, 58]. However, later research has shown better accuracy with ultrasound when compared with MRI scans in the detection of tendinopathy [59]. Greyscale ultrasonography was more sensitive, whereas colour Doppler ultrasound had a higher association with patients' symptoms [60].

Newer imaging modalities such as ultrasound tissue characterisation and sono-elastography have yielded promising initial results in improving sensitivity, specificity and accuracy in diagnosis [61, 62]. Further studies may be needed to investigate their role and application in the management of Achilles tendinopathy.

32.5 Treatment

32.5.1 Conservative Management

The first line of management for Achilles tendinopathy is conservative, and different treatments such as nonsteroidal anti-inflammatory drugs,

physical therapy, taping, cryotherapy, shock wave therapy, hyperthermia and various peritendinous injections have been used with varying success [54].

The management of Achilles tendinopathy lacks evidence-based support, because few treatment modalities have been investigated in randomised controlled trials [54], and approximately 25% of patients do not respond to conservative management [63].

Good results have been reported with eccentric exercises [64, 65], but eccentric exercises alone may not work in all patients [66]; however, the mechanism of action is not completely understood [65]. Eccentric exercises are the most effective conservative treatment for non-insertional Achilles tendinopathy. The most commonly used protocol is the Alfredson's protocol: the exercises are performed in three sets of 15 repetitions, twice a day for 12 weeks [67].

This regime was demonstrated to be effective in a 2009 systematic review, and confirmed with a meta-analysis in 2012, which outlined the best pooled data supporting eccentric exercises, with the majority of the studies adopting Alfredson's protocol [68]. Hailing this as "probably the greatest single advance in the management of this condition in the past 20 years" [69], Alfredson and other Scandinavian authors have reported excellent results in prospective RCTs [70, 71]. However, the proportion of good and excellent results in other studies using eccentric exercises is definitely lower [66, 72]; this can result from many factors, and the protocol requires motivated and compliant patients.

Other protocols, such as eccentric–concentric progressing to eccentric (Silbernagel combined) [70] and eccentric–concentric (Stanish and Curwin) [73], have been described. A systematic review showed that combined type exercise have equivalent results to the traditional Alfredson's protocol [74]. Isotonic, isokinetic and concentric loading have also been described, but are inferior to the eccentric-type exercises [75, 76]. In a prospective randomised controlled study, Rompe et al. [77] showed that eccentric strengthening plus repetitive low-energy shock-wave therapy (ESWT) was better than eccentric strengthening alone in terms of Victorian Institute of Sports Assessment—Achilles (VISA-A) scores and pain ratings at 4 months. The proportion of patients who were 'completely recovered' or 'significantly improved' on the Likert scale was also significantly better in the combined therapy group (82%) compared with 56% in the strengthening alone group.

ESWT, when compared with eccentric strengthening in a RCT, showed comparable outcomes, with 60% of the patients completely recovered or significantly improved in both of the treatment groups and significantly better than those in the 'wait and see' control group [72]. The success rate was lower than that seen in other studies, possibly because one-third of the patients in this study were not athletic and results are worse in these individuals [66]. In conclusion, where available, ESWT should probably be the second line treatment.

ESWT works on two aspects of the clinical response, namely tissue healing and pain transmission. Regarding the second, ESWT can change the histological appearance of dorsal root ganglion, modulating both central and peripheral nervous system inducing long-term analgesia [78]. Regarding tendon healing, ESWT can increase the levels of factors involved in tissue healing TGF-β1 and IGF-I expression in a rat tendinopathy model [79] and significantly decrease some interleukins [80] and matrix metalloproteinases (MMPs) on cultured tenocytes [81].

Various injection therapies have been proposed [82]. In a recent systemic review [83], only ultrasound-guided sclerosing polidocanol injections seemed to yield promising results, but these results do not appear to have been duplicated outside Scandinavia [84]. The use of platelet-rich plasma (PRP) seems to be growing exponentially, especially among sports medicine physicians, but the only well-designed RCT published on PRP in Achilles tendinopathy showed no significant difference in pain or activity level between PRP and saline injection at 6, 12 or 24 weeks when combined with an eccentric stretching programme [85].

High volume image guided injections (HVIGI) significantly reduce pain and improve function in patients with resistant Achilles tendinopathy [86].

The effects of HVIGI on neovascularisation and tendon thickness are not known. A prospective study of 2009 [87] assessed the effect of HVIGI (a mixture of 10 mL 0.5% bupivacaine hydrochloride and 25 mg of hydrocortisone acetate, followed by 4 × 10 mL of injectable normal saline) on patients' function, neovascularisation and tendon thickness in a short-term 3-week follow-up. There was a statistically significant difference between baseline and 3-week follow-up in all the outcome measures after HVIGI. In particular, neovascularisation was significantly reduced. The high volume injection may produce local mechanical effects, causing the neovascularity to stretch, break or occlude [87].

32.5.2 Surgical Management

Conservative treatment fails in between one-quarter and one-third of patients, and surgical intervention is required [88]. Open surgery has shown varying success rates between 50% and 100% [89–92] with removal of intra-tendinous lesions, and late-presenting lesions showing significantly fewer good to excellent results [93, 94]. For non-insertional Achilles tendinopathy, surgery has traditionally involved a large incision and excision of all of the pathological tissue, with or without augmentation with a tendon transfer [95].

The main concern with open surgery is the risk of complications. A large series of 432 consecutive patients from a specialist centre reported an overall complication rate of 11% [96]. These may include skin edge necrosis, wound infection, seroma formation, haematoma, fibrotic reactions or excessive scar formation, sural nerve irritation or injury, tendon rupture and thromboembolic disease. The rate of these complications might decrease with the use of minimally invasive techniques [54].

Minimally invasive therapies which strip the paratenon from the tendon, either directly [97] or indirectly with high-volume fluid injection [86], have shown good initial results in relieving the symptoms of non-insertional Achilles tendinopathy [54, 98].

Multiple percutaneous longitudinal tenotomies, which can be performed under ultrasound guidance, produce good results, with the further advantage of being able to perform the procedure under local anaesthesia in an outpatient setting [99, 100].

Minimally invasive open debridement with resection of the plantaris tendon has also shown promising results with minimal complications in elite athletes and regular patients with non-insertional Achilles tendinopathy [38, 101–104]. There are no comparative studies between the different minimally invasive approaches, and therefore it is unclear whether it is necessary to perform longitudinal tenotomies or to excise the plantaris tendon.

Therefore, minimally invasive surgical treatment would appear to be a useful intermediate step between failed conservative treatment and formal open surgery [54].

32.6 Conclusion

Non-insertional Achilles tendinopathy is a painful and debilitating condition arising from a failed healing response that can affect athletes and non-athletes alike. The majority of patients will respond to conservative treatment. For patients who fail conservative treatment, minimally invasive techniques show promising results with low complication rates, and may be a good option before open surgery.

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Achilles Tendon Ruptures

33

Jon Karlsson, Olof Westin, Mike Carmont, and Katarina Nilsson-Helander

33.1 Introduction

Acute Achilles tendon rupture is a common sports-related injury. Most of the patients are middle-aged, participating in recreational activities, such as tennis and badminton.

The rupture occurs in the majority of cases in the midportion of the tendon, approximately 2–6 cm from the bony insertion to the calcaneus. Distal ruptures, in a few cases with avulsion (bony avulsion) from the calcaneus, are less common. The Achilles tendon is the largest and most powerful tendon in the human body. It is formed by the soleus and gastrocnemius muscles and is located in the posterior superficial compartment of the leg. The average length of the Achilles tendon is 15 cm (range 11–26 cm) and mean width is 6.8 cm (4.5–8.6) [1].

The Achilles tendon is important for running and jumping, the reason it can produce such forceful elastic recoil and elongation is due to the spiraling of the tendon. It spirals 90° and in doing so produces an area of concentrated stress, with the fulcrum in the ankle joint [2] (Fig. 33.1). The degree of spiraling depends on the position of the fusion between the two muscles. More distal fusion increases the rotation. The insertion of the tendon in the calcaneus is crescent shaped. This osteo-tendinous junction provides a fulcrum and increases the lever arm. The retrocalcaneal bursa is located between the tendon and the calcaneus and reduces friction during motion [3].

33.2 Biomechanics

In terms of biomechanics, it is important to note that it is the tendon itself that transmits the force, while the muscle-tendon unit, which consists of the tendon as well as its muscle and aponeurosis, creates the whole unit. The Achilles tendon can store energy and release it when necessary. When jumping on one leg 74% of the mechanical energy is stored and 16% of the total mechanical energy comes from the elastic recoil action of the tendon. When force is applied to a tendon it will lengthen, the effect of this is demonstrated in the stress-strain curve. The stress that is placed on the tendon is calculated by dividing the force with the cross-sectional area of the tendon and given as the percentage of change in tendon length during loading; hence a thicker tendon is able to sustain a higher load than a thinner one. To describe the stiffness of the tendon, it is the slope of the linear region of the stress-strain curve. When at rest the fibers of the Achilles tendon are in a

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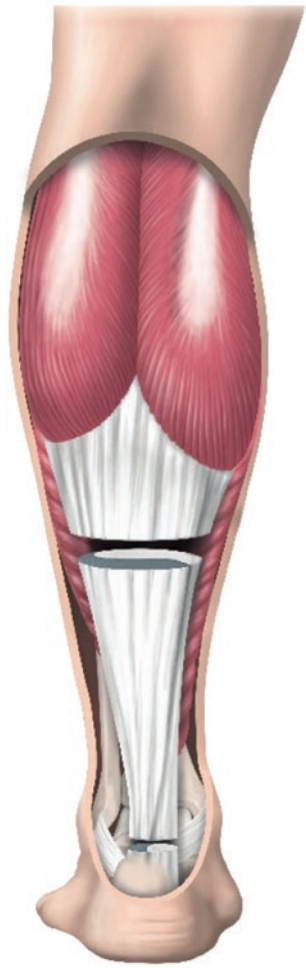

Fig. 33.1 The Achilles tendon anatomy and rotation

curly configuration. As seen in the figure they become stretched at 1–3% and can return to its former length when stretched. At more than 4% the fibers microfailure starts. If further force is applied after this the tendon will eventually break, i.e., macroscopic failure (Fig. 33.2). Eight per cent is often quoted in the literature as when macroscopic failure starts [4].

33.3 Incidence

The incidence of Achilles tendon ruptures has been extensively researched lately. It had been reported to be 18 per 100,000 in the 1990s; however, it is clear that there is a wide variation and that the incidence is increasing with values as much as 55.2 per 100,000 in males. The reason for this increment is that there is an increasing number of ruptures in the over 60 years age group, thought to be due to greater sports participation. Achilles tendon rupture is more common in men than woman with a quote at 5:1 [5]. Two age-related peaks have been reported. One in the early 40s often related to a sporting activity (recreational athletes) and one in the 60–65 years age group, usually more associated with minor trauma [6].

33.4 Injury Mechanism

The mechanism of an Achilles tendon rupture can be classified into three main categories (Fig. 33.3):

- Push-off with the weight-bearing foot while the knee is extended.
- Sudden unexpected powerful dorsiflexion of the foot.
- Forced dorsiflexion of the plantar flexed foot.

33.5 Etiology

The etiology of an Achilles tendon rupture is multifactorial. Degenerative changes may occur in the tendon, which will reduce its strength over time. This may explain the increase in incidence that has been correlated to the increasing participation in sporting activities in the middle aged (around 40 years of age and older). Inflammatory disorders, such as rheumatoid arthritis, as well as chronic renal failure and diabetes have been shown to increase the risk of rupture. The role of corticosteroid injections as a risk factor has been much debated and it is generally thought that the use of corticosteriod may increase the risk of rupture, but the evidence is in fact limited. It is also noted that repeated microtrauma leads to long-standing weakness of the tendon, which over time might lead to rupture.

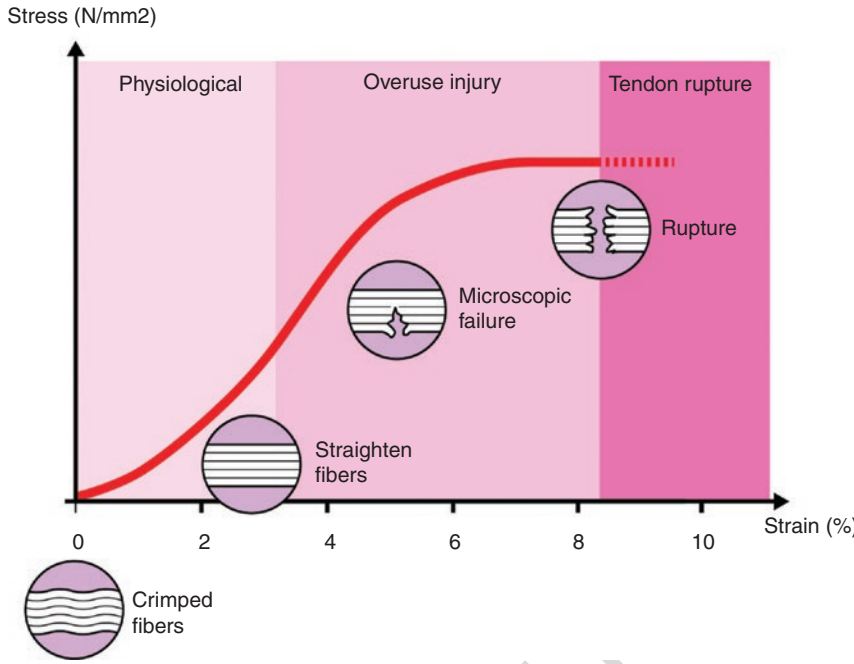

Fig. 33.2 Tendon stress-strain curve

Fig. 33.3 The mechanism of an Achilles tendon rupture

33.6 Clinical Signs

Patients often describe an Achilles tendon rupture as a sudden acute snap in their calf just above the ankle, as if someone had kicked them from behind onto the heel [7]. This is then followed by weakness and difficult to bear weight. Poor balance and altered gait are more common signs for chronic rupture along with passive hyperdorsiflexion of the ankle. Sometimes the clinical presentation can be difficult and it is well known that up to 20% of acute ruptures may be missed in the early phase by patients and physician, and may be mistaken for an ankle sprain.

Physical examination may be a challenge. Sometimes the plantar flexion weakness that one

suspects with a tendon rupture can be masked by the posterior tibial, plantar, and peroneus muscles. It is important to note that patients with a totally ruptured Achilles tendon can still walk on the injured foot, something that may be confusing to the attending clinician. The tendon gap can be difficult to palpate due to swelling and hematoma. It is important to understand this and be able to clinically examine an Achilles tendon in order to reduce the incidence of missed diagnosis. Several specific tests have been described.

In most cases there are no warning symptoms and the injury is related to one distinct ankle trauma. Almost always the rupture is total, and partial Achilles tendon rupture is very rare, especially in cases with a specific and classic symptoms, like "pop" sensation, which is localized to the midportion of the tendon. The diagnosis is clinical in the first place and there is no need to rely on either ultrasonography and/or magnetic resonance imaging (MRI) [8–10]. The diagnosis of mid-substance rupture is clinical with positive Thompson's test (calf squeeze test) [11], an abnormal resting posture, particularly on knee flexion and almost always a palpable gap in the tendon.

The Thompson test, also named the Simmonds test, is performed with the patient in prone position with the ankles hanging of the bed or with the knee flexed and the ankle free in the air (Fig. 33.4). The examiner squeezes the calf which causes a deformation of the triceps surae muscle with synchronized bowstringing of the Achilles tendon away from the tibia. The test is negative if plantar flexion occurs, which indicates that the tendon is intact. If there is no plantar flexion and/or clear difference from the contralateral side, the test is positive.

33.6.1 Surgical or Nonsurgical Treatment

The superiority of surgical or nonsurgical treatment is still debated, together with the timing of rehabilitation, for instance weight-bearing, early or delayed motion and whether functional bracing should be used or not. Treatment decisions depend on a patient's individual requirements, participation in sports activity, acceptance of brace use, and perception of risk.

33.6.2 Nonsurgical Treatment

Traditional methods of nonsurgical treatment include cast immobilization for 3-months followed by physiotherapy referral [BOFAS]. Other established methods of nonsurgical treatment include the use of bespoke braces [12], boots with wedges [13, 14], controlled ankle motion

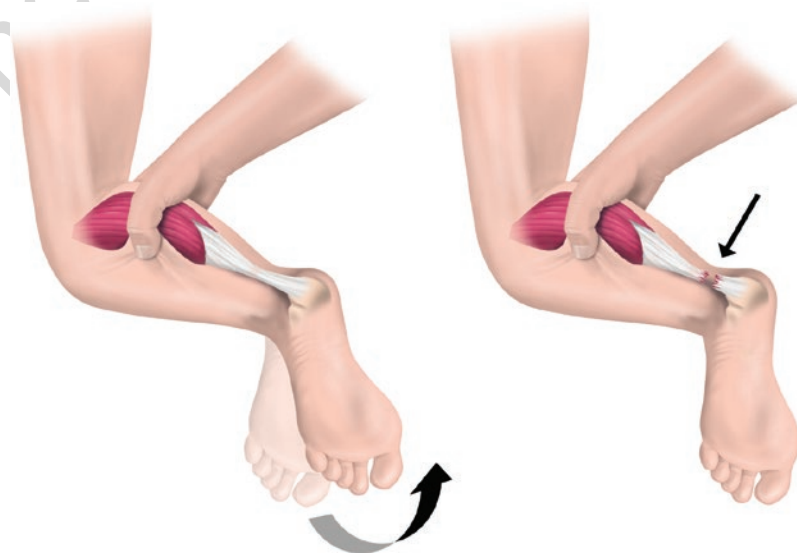

Fig. 33.4 Thompson's test

walkers [15], and conforming vacuum walkers with graduated ankle posture [16]. Meta-analyses have suggested similar re-rupture rates to those of surgical treatment, for nonsurgical management when early weight-bearing and range of motion exercises are adopted [17].

Nonsurgical management with protocols include an assessment after 2 weeks of cast immobilization [16, 18]. If abnormal resting posture to the ankle or a palpable gap is still present surgical repair is recommended. Nonsurgical treatment in the presence of greater than 1 cm separation of the tendon ends has been shown to lead to significant dysfunction [19].

Re-rupture rates in nonoperative treatment may be minimized further by the prolonged wearing of braces for as much as 4 months for vulnerable activities [16, 18].

33.7 Surgical Treatment

Open surgery (end-to-end repair) is considered to be the gold standard surgical procedure and with experience the length of the skin incision can be minimized to only 5–6 cm long [20] [Karlsson]. Studies have not shown any advantage of fascial turn-down flaps [21] (however, this technique can be used for re-ruptures or ruptures with delayed presentation). The end-to-end technique is appositional repair and can be performed without any augmentation up to approximately 3 weeks. The important contraindications are skin conditions (wounds, varicose veins), peripheral vascular diseases, and heavy smoking, where the risk of infection is high.

The open end-to-end repair can be performed in local anesthesia, or regional or general anesthesia. The patient is placed in the prone position and tourniquet is not needed. Prophylactic antibiotics and antithrombotic treatment is recommended. The feet are located over a pillow or hanging over the end of the operating table. It is important to have the opportunity to measure the Achilles Tendon Resting Angle (ATRA) of both ankles to ensure the tendon is repaired 5–7° tighter than on the non-injured [1]. Excessive plantar flexion, possible in flexible females, will increase the risk of overtightening and thereby shortening of the tendon. On the other hand, the risk of elongation is more pronounced and needs to be avoided, as elongation will lead to reduced plantar flexion strength and dysfunction [22].

33.8 Surgical Technique

The skin incision is usually 5–6 cm long. A posteromedial incision is preferred to reduce the risk of iatrogenic sural nerve injury. The paratenon is visualized and incised longitudinally. The frayed ends of the tendon are then visualized and mobilized. Suture placement is placed in the proximal and distal ends. The distal stump is usually around 2–3 cm long and good suture stability can be achieved with locking sutures proximal and distal to the frayed rupture ends. The surrounding tissues must always be handled with great care in order to enable closure and optimize blood flow in the vascular paratenon, enhancing tendon healing and reduce the risk of wound breakdown and infection. The frayed tendon ends are carefully apposed with the foot in plantar flexion. There are several different suture techniques with Kessler, Bunnell, and Krackow sutures being most commonly used [23].

A recently described technique is based on both core and circumferential sutures. This may be termed stable repair [24]. In fact, the purpose is to create such stable construct that immediate weight-bearing (possible without any postoperative immobilization) is achieved. The two core sutures consist of non-absorbable sutures with a modified Kessler technique. The double Kessler locking loop is placed well away from the rupture site in order to increase strength. Care must be taken not to damage the core sutures by needle passage. The ankle is held in 20–30° of plantar flexion, mimicking the other non-injured side and the sutures are tied using four throws (Fig. 33.5). During rehabilitation the tendon may elongate as much as 1 cm and in anticipation of this elongation the tendon is tied in increased tension with the ankle in relating increased plantar flexion or ATRA. When the core sutures are independently tied, a running circumferential suture is added to augment the strength of the repair [25]. Absorbable suture are used, with a continuous interlocking horizontal mattress

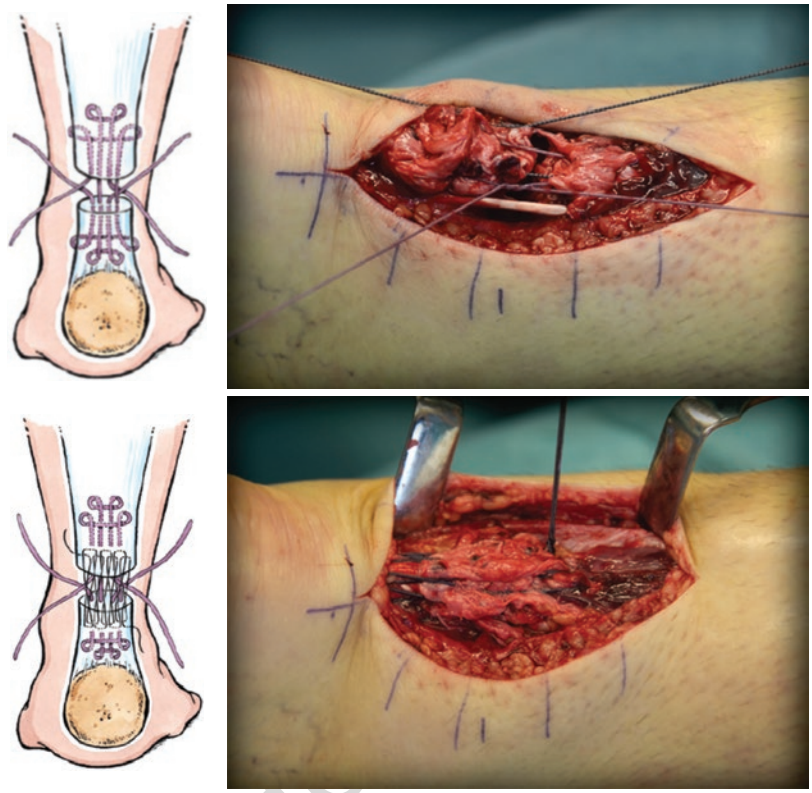

Fig. 33.5 Illustration of core suture

suture [26]. Finally, the paratenon is very carefully repaired and thereafter absorbable subcutaneous sutures and meticulous skin sutures. Interrupted sutures are recommended for optimal skin tension.

33.9 Postoperative Management

The postoperative management is described as accelerated rehabilitation, compared to traditional methods of 3 months cast immobilization [27]. Even though the core sutures are strong and will probably allow full range of motion training already early on, a cast is recommended for the first two weeks in order to rest the wound and reduce the risk of wound breakdown and infection [24, 28]. Weight-bearing is not allowed while the ankle is held in a temporary plaster cast in plantar flexion. A walker brace with 2–3 heel pads is applied after 2 weeks [24] (Fig. 33.6). One heel pad is then removed every other week,

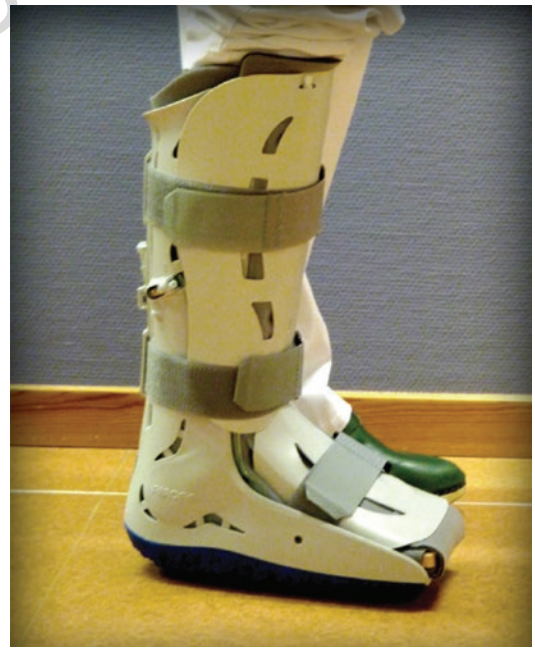

Fig. 33.6 Walker brace

while weight-bearing is gradually increased. Full weight-bearing can be allowed already after 2 weeks. A standard rehabilitation protocol is used. The total rehabilitation time is approximately 6 weeks.

33.10 Summary

Ruptures of the Achilles tendon are increasing.

Treatment should be individualized to a patient's requirements.

Nonsurgical treatment provides good outcome for patients with low physical activity demands.

Surgical repair reduces re-rupture rate, minimizes lengthening for the Achilles tendon, and optimizes plantarflexion strength following injury.

This stable surgical technique allows early range of motion training and early weight-bearing. In a recent study the risk of re-rupture has been shown to be 0%.

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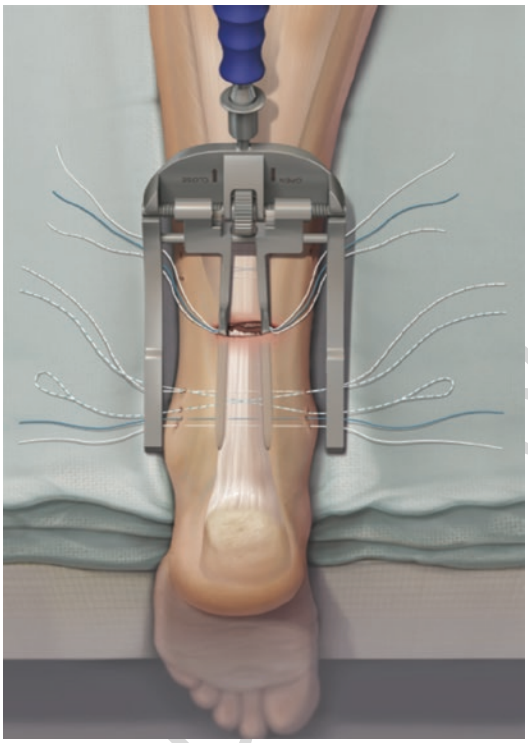
34 Minimally Invasive Repair of Acute Achilles Tendon Ruptures Using the Percutaneous Achilles Repair System (PARS) Arthrex Device

A. Nguyen and J. Calder



34.1 Background

The Achilles tendon is formed from the merger of the gastrocnemius and soleus muscles to insert on the calcaneus. It is enclosed by a thin gliding membrane paratenon, which provides nutrition and vascular supply to the tendon [1].

The tendon is a viscoelastic structure, capable of undergoing elongation and deformation in response to stress. This is true only up to certain levels of strain; if strains are between 4% and 8% the Achilles tendon complex may be damaged, and ruptures can occur if strains exceed 8% [2].

Acute rupture of the Achilles tendon can be associated with a classic history of sudden onset of pain, with almost exclusively no direct trauma to the region. Patients give a typical history of hearing a 'pop' and believing they were hit by something or somebody.

Clinical examination reveals a loss of the physiologic position of the affected ankle, and can be evaluated with the patient prone with both knees flexed. Comparison will reveal a relatively more neutral to dorsiflexed position of the injured side.

Ecchymoses and swelling is common, and a palpable gap can sometimes be felt at the region of injury. A calf squeeze test may show no ankle plantar flexion.

Generally an X-ray is performed as baseline to exclude any bony pathology, but in general no imaging studies are required for the diagnosis of Achilles tendon rupture. Dynamic ultrasound can be useful to aid surgical planning, especially in

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the chronic setting, where tendon mobility and tissue integrity can be questionable and more complex reconstructions may be required.

34.2 Management

There is controversy regarding the optimal management of the acute Achilles rupture. Concern for higher rates of re-rupture in non-operatively managed patients has led to a rise in the popularity of surgical intervention. This is balanced by the risks of infection, wound issues, and other surgical and anaesthetic complications.

The rate of re-rupture has been consistently shown to be high with non-operative cohorts (as high as 10–12% in many recent meta-analyses [3], as opposed to 1–2% for patients treated operatively). These figures have been criticised for including patients not participating in functional rehabilitation in the non-operative cohort but recent studies continue to demonstrate higher rates of re-rupture in non-operative (6.7%) vs. operative (3.7%) Achilles injuries [4].

Another proposed advantage of surgical treatment over non-surgical is the reduced loss of plantar flexion push-off strength. Several studies have demonstrated relative push-off strength is higher following surgical repair compared to non-operative management [5].

Proponents of a surgical management also cite a return to functional activity and sports with surgical treatment. A recent systematic review demonstrated faster rehabilitation, reduced time back to work, and better functional outcome after surgery [6].

One of the major disadvantages of a surgical approach is the complication profile, notable wound healing and infection. Open repair has traditionally been performed with a large longitudinal incision and locking Krakow sutures to approximate the tendon ends. This has been shown to have a higher rate of complications over non-operative treatment, including wound problems [7].

The percutaneous or mini-incision techniques have shown reduced rates of these potentially disastrous complications. A recent study compared the PARS Arthrex system to open repair and found a significant reduction in total complications (5% vs. 10.6%), with improved rates of return to baseline activity [8].

This system also allows for a knotless approach at the repair site when combined with the Speedbridge system; this has been shown to produce excellent results in the elite athlete setting, where faster rehabilitation is made possible by fixation to bone in the distal os calcis, and there is a theoretical reduction in suture bulk and knot slippage [9].

To summarise, management decision-making should be patient focused, with a knowledge of occupation and sporting level, medical comorbidities including smoking and vasculopathy, as well as patient wishes.

34.3 Operative Technique Using PARS®

A general anaesthetic is typically used. The patient is positioned in the semi-prone position with the legs positioned prone, the hips semi-prone and the upper body positioned lateral. Minimal bolster support is required. The uppermost arm is placed in an arm gutter and a sandbag may be placed under the iliac wing to prevent any forward tilt. A preoperative assessment of the patient to exclude significant limitation of hip external rotation or increased tibial torsion should be performed, as this may in rare cases make this positioning difficult. A fully prone position may be used in this case.

A tourniquet is applied around the thigh and inflated to 300 mmHg; this is easiest to apply before the patient is positioned semi-prone. Intravenous antibiotics are administered prior to inflation of the tourniquet. The feet are positioned over the end of the table with a pillow under both tibiae to allow the gastrocnemius to relax slightly.

It is desirable to prepare and drape both legs to allow for comparison of foot position following repair to ensure appropriate repair tension.

Skin sterilisation below the knee is sufficient in the acute repair setting using an alcoholic chlorhexidine preparation solution.

A 2–3 cm incision is placed 1 cm below the end of the proximal tendon stump. The incision can be made either vertically or horizontally depending on preference; the authors have experience with both with no significant complications. If made vertically, the incision is made just to the medial side of the mid-posterior line.

Meticulous skin and tissue handling is imperative throughout this procedure. After the skin is incised, the paratenon, if not already opened as a result of the injury, is incised. Often a gap is then seen with strands of ruptured tendon visible. The tendon ends need to be identified for the PARS device to be passed within the paratenon. Dissection can be performed to identify and free both the proximal and distal tendon ends using a blunt curve Mayo scissor.

The tendon may be stabilised using a tendon clamp, and the inner arms of the PARS jig are now placed within the paratenon on either side of the proximal tendon. Once inside, the inner arms can be opened or closed by rotating the wheel on the jig. The device is then inserted along either side of the tendon. The muscle belly will usually stop the device at an appropriate level (Fig. 34.1).

The jig has corresponding numbered holes on either side to allow for passing of sutures. We typically use suture tape, as it allows for stronger hold, with a flatter profile of suture. There are seven holes for suture options and holes 3 and 4 are obliquely orientated and designed for a looped suture to pass through. This is a locking suture. We typically use holes 1–5 for Achilles fixation. Pass sutures through holes 1–5, to have a configuration as shown. One looped suture is on either side of the jig (Fig. 34.2).

The jig is then slowly withdrawn out of the incision, and once the inner arms are seen, pull the suture loose from the outer arms and remove them from the wound to avoid getting stuck in the holes of the outer jig (Fig. 34.3).

The number 2 hole suture is then passed under suture 3 and 4 and through the loop on each side, and then pulled through to achieve a locking suture on each side (Figs. 34.4 and 34.5).

All steps are now repeated for the distal portion of the tendon, to achieve the following final configuration (Fig. 34.6). We recommend that it

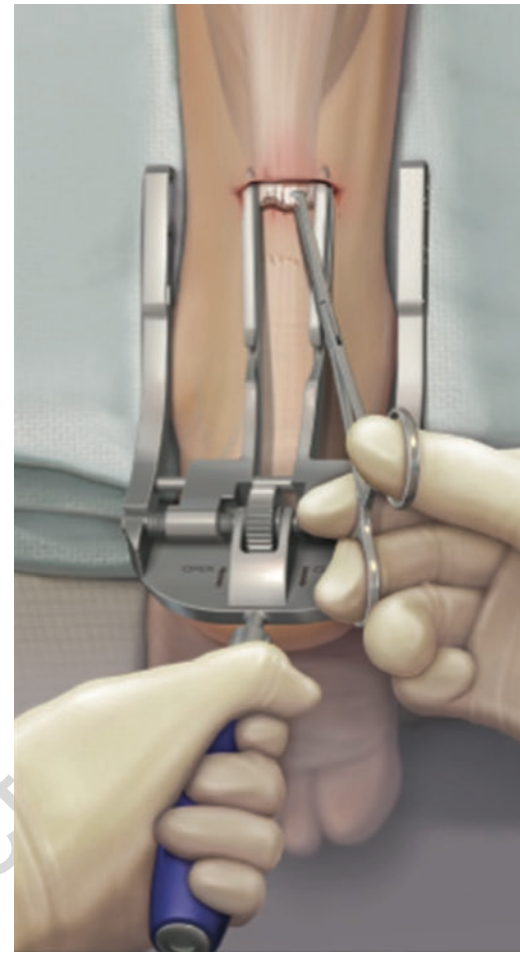


Fig. 34.1 Advancement of the PARS jig inside the paratenon, with careful traction on the proximal tendon

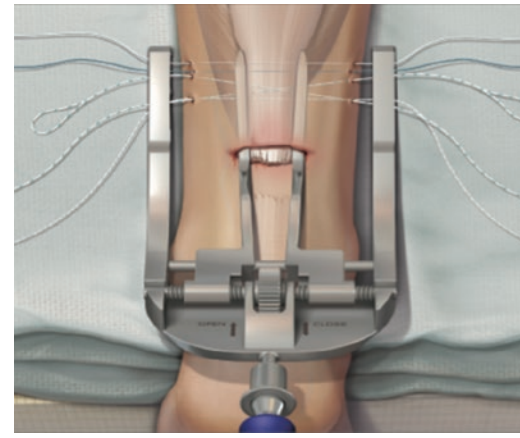


Fig. 34.2 Passage of sutures through the proximal tendon and PARS jig

is essential to check each suture both proximally and distally for pull out strength. If a suture pulls through the tendon on moderate tension, then it needs to be redone. When testing pull out strength, pull the sutures in a direction parallel with the Achilles tendon to avoid strafing the skin and wound edge.

The tendon is now ready to be repaired. With the foot in maximal plantar flexion, tie the suture from hole number 5 with 4 knots on each side. A low sterile table at the end of the bed is useful to lay the foot on in a plantarflexed position, both when exposing the tendon ends and when tying the repair. This is useful to free up the surgical assistant for other tasks. The height of the bed can then be adjusted to obtain the optimal foot position prior to tying the sutures.

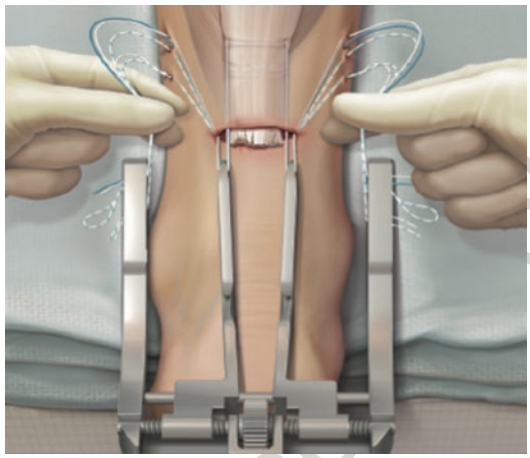


Fig. 34.3 De-tensioning the suture construct to facilitate removal of sutures and PARS jig

This will approximate the tendon and allow for tying of the locking sutures which will not slide. Tie the locking sutures on each side with 5 knots and then the final suture hole number 1 to complete the repair (Fig. 34.7).

A 2/0 vicryl epitendinous suture is then used to augment the repair, with care to place knots on the deep surgical side. The paratenon is then loosely closed with 2.0 vicryl rapide, and the small skin incision with interrupted 3.0 nylon. A front slab is then applied in 20° equinus.

34.4 Alternative Technique Using PARS/Speedbridge

This technique is a modified percutaneous technique that combines the benefits of percutaneous repair with direct bone fixation, bypassing suture

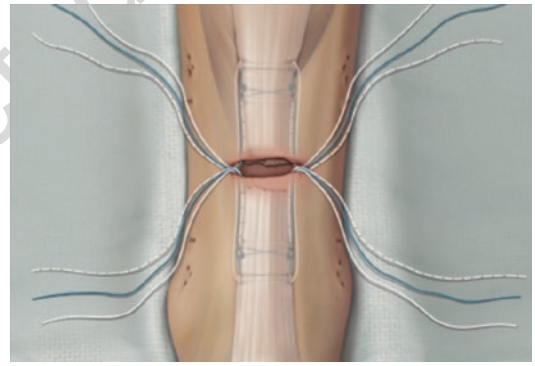
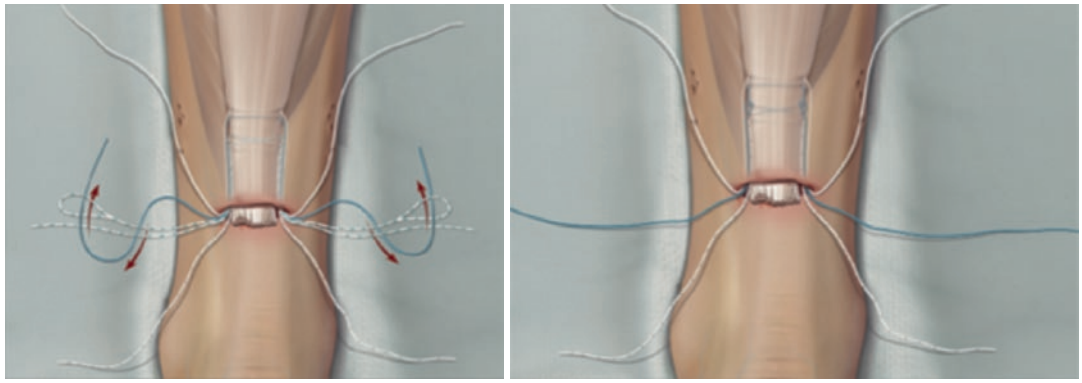


Fig. 34.6 Final suture construct prior to knot tying



Figs. 34.4 & 34.5 Creation of the locked suture know on each side of the proximal tendon

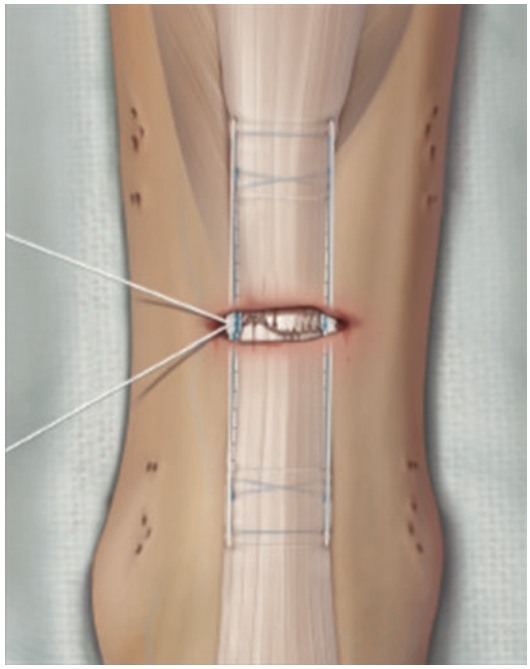


Fig. 34.7 Suture tying with the foot (not shown) in appropriate plantar flexion

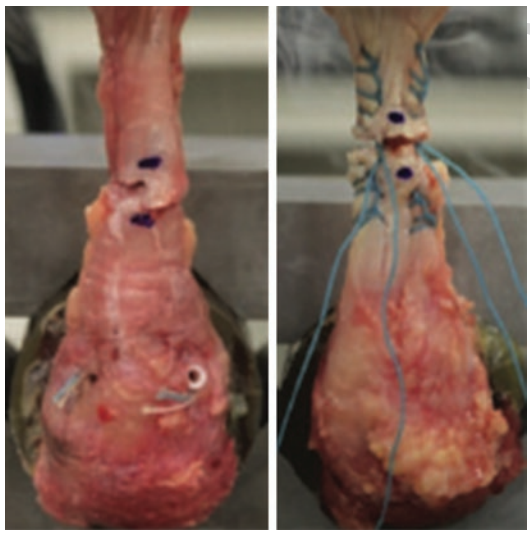


Fig. 34.8 Biomechanical comparison of PARS/Speedbridge combination construct compared to open Krakow repair

knots at the repair site as well as the potentially compromised tissue at the rupture site [9].

Biomechanical studies have demonstrated statistically significantly less cyclic displacement at 500–1000 cycles of this construct in comparison with a standard open Krakow suture repair [10] (Fig. 34.8).

The surgical technique is identical in positioning, incision and preparation of the proximal stump with the PARS device. Two stab incisions 2 cm apart are then made at the level of the Achilles insertion over the calcaneus and drilled using a 3.5 mm drill guide. These holes are then tapped in preparation for two 4.75 mm SwivelLock anchors (Figs. 34.9 and 34.10).

A Banana SutureLasso device is passed from each of the two distal incisions to capture the three sutures on each side of the tendon proximally, and these are pulled through into the distal incisions (Fig. 34.11).

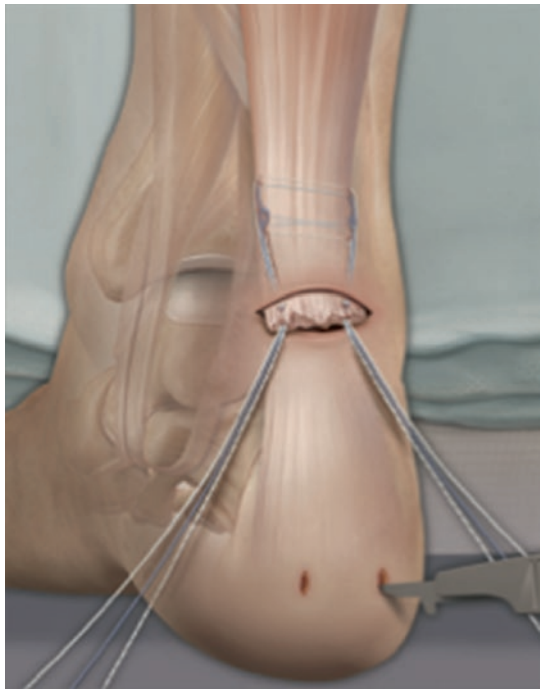
The anchors are then inserted at correct tension to achieve the final construct (Fig. 34.12). It is possible to place an epitendinous suture at the level of the proximal incision if desired.

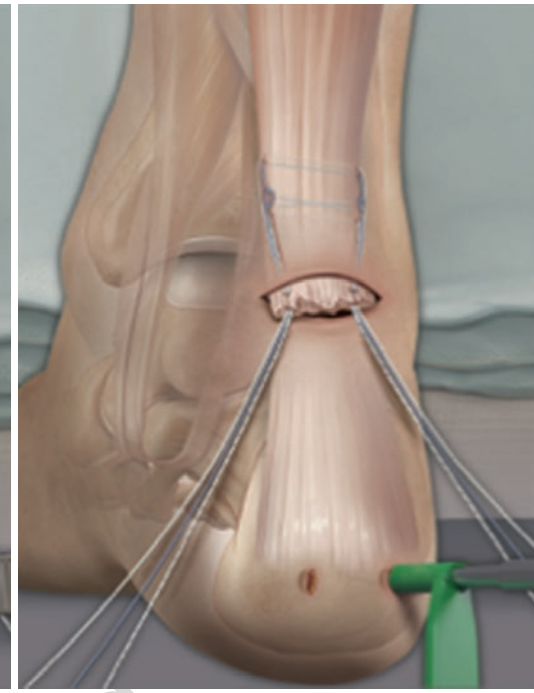
34.5 Pearls and Pitfalls

Use gentle pressure in a downwards direction on the tendon ends to hold the tendon in place when passing the sutures. We have found in very rare circumstances the proximal end of the tendon can be difficult to transfix with percutaneous sutures. In this case the PARS can be used for the distal stump, and a 'half-open' approach can be used, with a longer incision proximally. Given that it is the distal portion of the wounds that in our experience is most affected by wound issues in the open repair setting, this can be a useful adjunct in the difficult case.

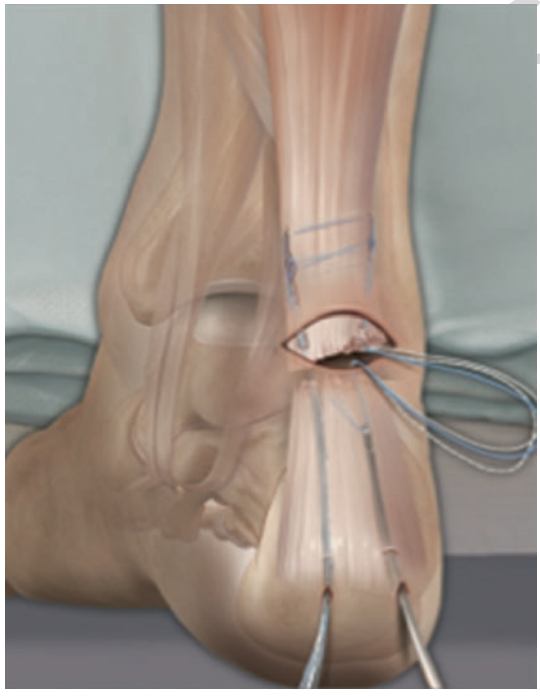
We have found it useful to vary the angle of the jig slightly in the axial plane while passing the sutures; this can facilitate sutures at a slightly different angle in the substance of the tendon, and is a useful technique if sutures are pulling out on testing.

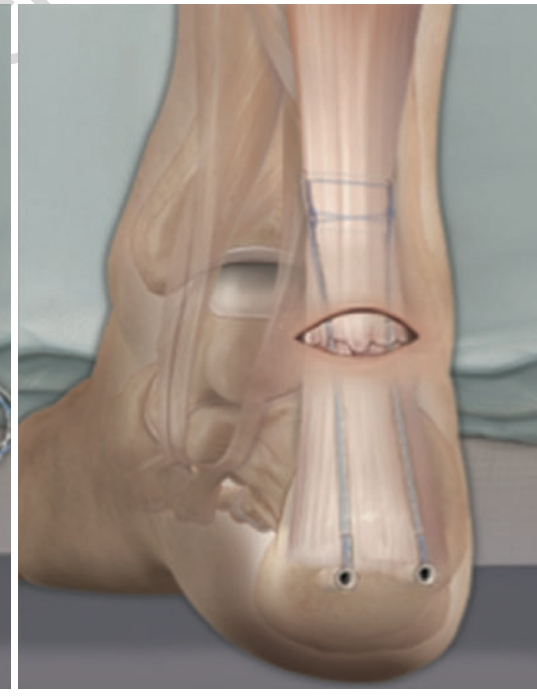
The authors use SutureTape® in distinction to a Fibrewire® or vicryl suture for repair. We have found this to be a lower profile suture reducing the size of knots and potential wound problems. SutureTape has also been shown to have a higher ultimate load to failure and greater tissue pull-through strength than a #2 FibreWire [11].





Figs. 34.9 & 34.10 Preparation of distal anchors after identical use of PARS jig in proximal tendon





Figs. 34.11 & 34.12 Sutures passage to distal anchor site and final construct after anchor insertion

In slight variation of the PARS only technique for suture knot tensioning, we have found that tying the locking sutures off first is best for approximation. Following this, we tie both sutures on one side of the Achilles tendon and slide the knot proximally by pulling on the suture on the opposite side. This leaves two knots proximally and away from the wound which we believe reduced the risk of wound problems due to suture bulk.

34.6 Post-operative Care

The wound(s) are dressed with an absorbent dressing and the patient is placed in a plaster dorsal slab with the foot in equinus of 20° to aid skin perfusion for 2 weeks.

We advocate DVT prophylaxis for 2 weeks whilst immobilised in a plaster slab; Achilles tendon injuries have the highest incidence of both radiologic and clinically relevant venothromboembolic events in foot and ankle surgery in a recent meta-analysis. The rate of DVT in general events was 1% and 13% for clinical and radiological VTEs, respectively, and 7% and 35%, respectively, for Achilles tendon ruptures [12].

Rest, non-weight bearing and elevation are advised to promote wound healing. From 2 weeks the patient may be placed into a removable boot with heel wedges and allowed to weight bear with crutches. Active, gentle ankle plantar flexion and dorsiflexion to neutral is commenced at 3 weeks to minimise paratenon adhesion. Wedges are removed weekly and the ankle should be plantigrade in the boot by 6 weeks and the boot removed by 7–8 weeks. Passive dorsiflexion should be avoided. A graduated therapy programme should aim for full recovery by approximately 6 months.

Acknowledgments All images courtesy of Arthrex Inc.

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Part V

Special Considerations

35 Outcome

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35.1 Introduction

Outcome measures are an important component of medicine in its broadest sense. Outcome measures are of great clinical importance to monitor treatment quality, to analyze the clinical effectiveness, as well as to compare the effectiveness of different surgical or nonsurgical treatment options. Ideally, outcome measures are frequently and practically applied in the clinic, so that one can have a complete, accurate, and objective overview of the actual situation of the patient at different times in the treatment protocol. Moreover, an optimal outcome measure will be easily applicable in the clinics and will have low costs.

Currently, a high number of outcome measures are available to assess outcomes in the foot and ankle [1–6]. The choice of a particular outcome measure strongly depends on personal preferences of the affiliated people, the specific outcome of interest, as well as the measurement properties of the measurement tool [7, 8].

To be as complete, accurate, and objective as possible it is important to know which instruments are available when selecting specific outcome measures. In this chapter we will outline an overview of the most important outcome measures that can be utilized in the assessment of foot and ankle problems. Furthermore, it is important to assess the psychometric properties of the instrument you want to use. Assessing the psychometric properties of an outcome measure can help you determine if the instrument actually fits its intended purpose. Therefore the aim of this chapter is to help you understand how to assess the psychometric properties of outcome measures and to give a clear overview of the best available outcome measures for foot- and ankle-related sports injuries.

35.2 Psychometric Properties

Psychometric properties are the properties of a tool or instrument which help determine how well this tool or instrument actually measures what it intends to measure. To be able to adequately assess if a certain outcome measure is fit for its intended purpose it is essential to have some basic understanding of psychometric properties.

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The most important psychometric properties include validity, reliability, and repeatability and will be discussed below.

35.2.1 Validity

As stated in the Standards for Educational and Psychological Testing (American Educational Research Association, American Psychological Association & National Council on Measurement in Education, 1999, p. 9) "Validity refers to the degree to which evidence and theory support the interpretations of test scores entailed by proposed uses of tests." More specifically this means three aspects are important regarding validity when selecting outcome measures:

1. To which extent does the tool measure what it intends to measure?
2. To which extent does the tool measure all facets of the intended outcome?
3. To which extent is the measure related to the outcome?

35.2.2 Reliability and Repeatability

Reliability is a degree relating to the amount of random error in measurements. There are different types of reliability which should be considered when selecting outcome measures.

- Test-retest reliability. This type of reliability assesses the extent of agreement when testing a tool in the same population at different time intervals in case there is no change in outcome. Test-retest reliability can also be called repeatability.
- Inter-observer reliability. When assessing foot and ankle problems it is important that different health care professionals measure the same outcomes when using the same tools.
- Intra-observer reliability. When performing the same measurement more than once, an observer should measure the same outcome both times in case nothing in the outcome has changed.

35.3 Outcome Measures in Sports Injury of the Foot and Ankle

35.3.1 Patient History

The first step in assessing any foot or ankle problem should always be history taking. Patient history is of vital importance in assessing the symptoms, concerns, medical-, social-, and psychological history. In this stage the problem of the patient needs to be identified. It can sometimes be a challenge to convert this subjective information into objective and quantifiable information. On the other hand, subjective information is also of great importance. For example, an os trigonum causing pain following repeated, forceful plantarflexion of the foot can have great impact on the quality of life of a ballet dancer. However, for the general population, not performing these specific tasks at such a level, this would not be a problem. Another problem that can only be identified with history taking is activity avoidance. A patient can score zero points on a numeric rating scale for pain, solely because this patient avoids activities that would lead to the particular pain. Therefore history taking is essential to identify problems and to individualize treatment. A tool that can be very helpful to individualize treatment is goal attainment scaling (GAS). With the help of GAS, specific goals that are important for an individual patient can be identified, measured, and evaluated.

35.3.2 Physical Examination

Physical examination can be used to find the right diagnosis as well as to assess recovery after treatment. Physical examination can be difficult to objectify. Parameters such as height and weight are easy to quantify. For other parameters, such as range of motion or strength, this is more difficult. Aids such as a dynamometer or a goniometer can be useful to quantify these parameters and get a more reliable outcome, which can be assessed longitudinally.

35.3.2.1 American Orthopedic Foot and Ankle Society Score (AOFAS)

The American Orthopedic Foot and Ankle Society score (AOFAS) is a clinician-reported tool which is designed to help physicians standardize the assessment of patients with foot or ankle disorders [9]. The survey contains both subjective and objective measures. The AOFAS score has been developed for four different regions of the foot: the ankle-hindfoot, the midfoot, the metatarsophalangeal (MTP)-interphalangeal (IP) for the hallux, and the MTP-IP for the lesser toes. Each questionnaires covers three categories: pain, function, and alignment. Because the AOFAS scale is such a widely used instrument it offers good comparison between different studies. However, the AOFAS scales have never been validated for the evaluation of the treatment of any foot and/or ankle pathology.

35.3.3 Imaging

A high number of standardized scoring systems exist for the radiological assessment of foot and ankle problems. For example, the Kellgren-Lawrence scale for the assessment of osteoarthritis [10]; the Weber classification for ankle fractures [11]; or the Berndt and Harty Classification for osteochondral lesions of the talus [12]. The aim of this chapter is not to give a complete overview of all the available scoring systems in imaging. However, there is an important key message in this paragraph: the most important aspect is to inspect and assess each individual patient. For this, teamwork is essential; the treating clinician, radiologist, and patient should collaborate closely in order to identify the right diagnosis and subsequently come to the optimal evidence-based treatment protocol.

35.3.4 Patient-Reported Outcome Measures (PROMs)

Patient-Reported Outcome Measures (PROMs) are standardized, validated questionnaires completed by patients to measure their perceptions of their own functional status and well-being [13]. A distinction can be made between surveys which are used to assess general health, pain or satisfaction levels and surveys which are used for specific symptoms or health problems. Generic health status measures are often less responsive to foot- and ankle-specific problems, but can be highly useful to assess the impact of different conditions on the quality of life or on the general health of the patients. The most relevant PROMs for injuries of the foot and ankle will be discussed below.

35.3.4.1 Short Form-36 and Short Form-12

The SF-36 is a generic health measure that contains a set of 36 questions in eight domains [14]. The SF-36 is used to assess general health and contains both physical and mental measures. It is a generic measure which means symptoms and problems specific for a certain condition, treatment or age group are not included. The SF-36 is a very useful tool for descriptive purposes and also for evaluating benefits of alternative treatments. A shorter form of this health survey is also available: the SF-12. This score adequately reproduces the physical and mental component summary score which can be derived from the SF-36 [15]. Because of the lower patient burden of the SF-12 compared to the SF-36, the SF-12 may be preferred over the SF-36.

35.3.4.2 EuroQualy of Life-5 Dimensions (EQ-5D)

Another instrument to measure general health status is the EQ-5D. The EQ-5D is a generic measure developed by the EuroQol Group [16]. The EQ-5D defines health in five domains: mobility, self-care, usual activities, pain/discomfort, and anxiety/depression. The EQ-5D is a useful tool in assessing the general health status of patients.

35.3.4.3 Foot and Ankle Ability Measure (FAAM)

The Foot and Ankle Ability Measure (FAAM) is a specific tool for foot and ankle problems and has been developed with the objective to develop an

instrument that can be used to evaluate changes in self-reported physical function for individuals with leg, ankle, and foot musculoskeletal disorders [17]. The FAAM consist of two subscales: the activities of daily living (ADL) subscale and the sports subscale. The FAAM is a reliable, valid, and responsive measure of self-reported physical function for individuals with a broad range of musculoskeletal leg, ankle, and foot disorders [17].

35.3.4.4 Foot and Ankle Disability Index (FADI)

The foot and ankle disability index (FADI) is also a specific tool for foot and ankle problems. Reliability and sensitivity of this score have been determined in patients with chronic ankle instability (CAI). A study by Hale and Hertel [6] in 2005 concluded that the FADI appears to be reliable in detecting functional limitations in subjects with CAI, sensitive to differences between healthy subjects and subjects with CAI and responsive to improvements in function after rehabilitation in subjects with CAI.

35.3.4.5 Foot and Ankle Outcome Score (FAOS)

The Foot and Ankle Outcome Score (FAOS) is a specific tool for foot and ankle problems and has been developed to assess the patients opinion about a variety of foot- and ankle-related problems. The FAOS consist of five subscales: pain, other symptoms, function in daily living (ADL), function in sport and recreation, and foot- and ankle-related Quality of Life (QoL). Studies have shown that the FAOS is a valid and reliable tool in patients with osteoarthritis, ankle instability, flatfoot deformity, and hallux valgus [18–21].

35.3.4.6 Foot Function Index

The foot function index (FFI) tool has been developed with the objective to measure impact of foot pathology on function [2]. The FFI consists of three subscales: pain, disability, and activity restriction. The FFI is a useful tool for low functioning individuals with foot disorders, but may not be useful to assess individuals who function at or above the level of independent activities of daily living [1].

35.3.4.7 Self-Reported Foot and Ankle Scores (SEFAS)

The self-reported foot and ankle score (SEFAS) has been designed with the purpose to evaluate disorders of the foot and ankle. This questionnaire is validated in patients with arthritis of the ankle and in patients with forefoot, midfoot, hindfoot, and ankle disorders [4, 22]. In patients with great toe disorders or hindfoot disorders the SEFAS showed similar or better psychometric properties compared to the AOFAS and was completed much faster after surgery [23].

35.3.4.8 Disease-Specific PROMs

Besides the PROMs specific for foot and ankle problems, there are also PROMs specifically developed for a specific disease or condition. An example is the Cumberland Ankle Instability Tool (CAIT), which has been validated to assess the severity of ankle instability [24]. Another example is the ankle osteoarthritis scale which is a valid and reliable instrument that specifically measures symptoms and disabilities related to ankle osteoarthritis [25]. To obtain an evaluation of the patient which is as complete as possible, it is recommended to combine a generic health measure, a foot- or ankle-specific health measure, and, if available, a disease-specific PROM.

35.3.5 Complications and Recurrence Rate

To be able to adequately assess safety and effectiveness of any surgical technique it is essential to monitor complications and recurrence after surgery. The Dindo-Clavien classification system is an example of a classification system which can be used to monitor the nature and number of complications [26]. Additionally every surgeon has to be able to inform his patients about the complications and recurrence rate prior to a surgical procedure. Despite the importance of adequately reporting complications and recurrence rates, these are often underreported. Reporting complications represent a conflict of interest for physicians despite the potential consequence of that underreporting complications and recurrence

rates may lead to unexpected treatment failure or complications in future patient. A reason for this can be the inherent fear of medicolegal consequences, even though over 95% of all surgical complications will never lead to a lawsuit [27]. Other reasons for underreporting complications and recurrence rates can be the potential loss of professional respect and a potential decrease in patient referrals and revenue [28].

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36 Outcomes Assessment for the Athlete

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36.1 Introduction

Foot and ankle problems are highly common in athletes. The ankle is the most commonly injured joint in sports and the ankle sprain is the most common injury of all joint injuries in sports practice [1, 2]. Injuries of the foot and ankle lead to considerable time lost to injury and disability. Considering athletes and all involved parties of this particular athlete, this may be a big problem. After an injury, athletes wish to return to sport as quickly as possible. However, deciding when exactly an athlete can return to sport can be a complex and multifactorial process. Firstly, athletes have a much higher demand on ankle function compared to people who do not undertake sports on a regular base as many sports include, for example, jumping, cutting edge movement, and running. As such, they will be able to perform higher level activities even when recovering from injury. This results in the potential of a ceiling effect in many existing scoring systems, as was demonstrated in the Foot and Ankle Ability Measure (FAAM) [3, 4]. Secondly, athletes have a different clinical and functional response to injury compared to the general population [3]. Another challenge is that many people, with different wishes, expectations, and goals, can be involved. For example, the athlete, who wants to return to sport as soon as possible; the coach, who wants the athlete to perform at least as good as before his injury; and the treating clinician, who wants safe return to sports with prevention of re-injury.

This chapter will focus on the complex outcomes assessment for the athlete which can make return-to-play decision-making challenging. Evaluation of specific sports-related risks will be discussed in this chapter, as well as factors which can modify the return to sport decision. Outcome measures to evaluate the general health status of the athlete will however not be discussed in this chapter, as they have already been described in the previous chapter of the present book. The underlying aim of this chapter is to give the reader insight into the current evidence regarding sport-specific outcomes.

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36.2 Sports-Related Risk Assessment

After assessing the general health of the patient, the sports-related risks should be assessed [5]. The sports-related risks can give information about the amount of stress that is applied to an injured tissue. If the amount of stress on tissue is bigger than the capacity that the tissue can withstand, the tissue in question shall not heal, and consequently injury or re-injury is likely to occur [5]. An important factor to assess the sports-related risks at injury is the type of sport that the athlete practices. Generally, noncontact sports such as swimming pose a lower risk on acute injury compared to contact sports and high-impact sports, such as basketball or soccer [6]. Contact sports, at their turn, pose a lower risk at acute injury compared to collision sports such as rugby or boxing [6]. However, more factors play a role in the sports-related risk of injuries. For example, the competitive level of the athlete. To quantify the activity level specifically for the ankle, the Ankle Activity Score (AAS) can be used [7]. This score is validated for the use in ankle instability patients and can be used in the evaluation of treatment of patients with ankle instability.

Another manner to systematically assess and categorize the amount of tissue stress is according to the frequency, intensity, timing, and type (FITT) principle [5]. FITT is based on the principle that there should be a training balance on four domains:

- *Frequency*: The optimal training frequency is when the training is frequent enough for the tissue to adapt and infrequent enough for the tissue to heal and adaptation to occur.
- *Intensity*: The optimal training intensity is when there is a balance between overloading and overtraining. In cardiorespiratory sports such as running, heart rate can be used to objectify training intensity. In resistance training, workload (amount of weight lifted, amount of repetitions and amount of rest between sets) can be used to objectify training intensity.
- *Type*: The type of training and the stress that increases with that type of injury. For example, when a ballet dancer presents with tendonitis of the Achilles tendon, the stress can be reduced by quitting the regular training and performing exercises to stretch and strengthen the calf muscles in order to reduce stress on the Achilles tendon instead.
- *Time*: In the optimal training duration there has to be a balance between the duration of exercise that causes enough stress for the tissue to adapt. However, there should not be stress to such an extent that the tissue damage is too severe to recover prior to the subsequent training session.

36.3 Risk Tolerance Modifiers

When the medical team has a clear image of the medical factors and the sports-related risks there is one more step that has to be assessed prior to making a treatment plan, that is, risk tolerance modifiers [5]. Especially in high-level athletes there can be a high number of additional factors that can be of influence considering the treatment plan. The StARRT framework describes common risk tolerance modifiers [5].

- Timing: deciding when to treat can be very important for an athlete. For example, an athlete and his treating physician may choose to accept a higher risk of (re-)injury right before an important match.
- Pressure coming from the athlete: when an athlete desires to compete the risk tolerance is higher.
- External pressure: pressure on the athlete not primarily of personal nature. This pressure is rather of external nature, derived from the affiliated parties (for instance the football club, the coach, the manager, family members, etc.).
- Masking the injury: in some cases analgesia can be effective to mask the injury so the athlete can continue to compete without doing further damage.

- Conflict of interest: Financial motives can motivate athletes to postpone treatment. For example, right before a transfer period when an athlete does not want potential buyers to know about an injury.

36.4 Return to Sport

After the assessment of medical factors, sports-related risks, and risk tolerance modifiers, the athlete and treating medical team can make a decision about when an athlete can return to sport. Return to sport is not just a decision at the end of a treatment process but a continuum influenced by a number of factors [8]. When an athlete is injured it may be necessary to completely remove an athlete from sport so that the athlete can recover. When recovering from an injury there are a number of stages between removal from sport and return to preinjury level or return to performance. These stages will be discussed in the following paragraph:

- Removal from sport: Complete removal from sport can sometimes be the optimal treatment option. In some acute cases it can be vital for the athletes' health that the athlete will be immediately removed from sport. In cases where symptoms gradually increase over time, it may also be necessary to completely remove the athlete from sport to in order to initiate or speed up the recovery process.
- Return to participation: In this stage the athlete is physically active but has not yet returned to his or her desired sport [8]. The athlete is, for example, rehabilitating by following an adjusted training program.
- Return to sport: The athlete has returned to his desired sport but has not yet reached his or her preinjury level [8].
- Return to performance: The athlete has returned to his desired sport at or above his preinjury level [8].

Prior to starting treatment it is important to discuss the return to sport continuum with the athlete. For some athletes it is enough to return to participation or return to sport, but they do not need to return to performance. In other cases it may not be realistic to aim for the athlete to return to his or her preinjury level. To come to the best treatment plan and outcomes patients, coaches and clinicians should work together in an intensive manner. One should also pay close attention to the psychological parameters that can be involved concerning the return to sport process [9]. A systematic review by Ardern et al. [10] focused on different studies including athletes returning to sports after an ankle injury as well as reporting at least one psychosocial property. This study concluded that there is preliminary evidence showing that positive psychological responses are associated with higher rates of return-to-sports. This indicates that it is of clinical importance as a medical team to also focus on the mental health of athletes.

36.5 PROMS in Sports

A number of patient-reported outcome measures (PROMs) have a sport-subscale or are specifically designed to assess outcomes in relation to sport. These outcome measures can be utilized in the outcome assessment of athletes after a specific treatment protocol. PROMs for the foot and/or ankle with a specific sports subscale or PROMs for the foot and/or ankle specifically designed to assess outcomes in relation to sport are described in this section of the book chapter.

36.5.1 Sports Athletes Foot and Ankle Score (SAFAS)

The Sports Athletes Foot and Ankle Score (SAFAS) is a PROM which is developed with the purpose to create a valid, self-administered score for high performing athletes [11]. The scoring system was based on conducting interviews with professional athletes, prior to actually creating the scoring system itself. During these interviews, the athletes were asked to comment on existing scoring systems, such as the FAAM, the FAOS, and the FFI [4, 12, 13]. The athletes

who were requested to participate in these interview sessions participated in different types of sports, such as rugby, football, cricket, and so on. All athletes had had some type of foot and ankle injury, being associated with sports, in the past. The SAFAS is a valid instrument in the assessment of sports-related foot and ankle problems and it detects change between the healthy and injured high-level athlete [11]. The subscales assessing the levels of symptoms, pain, daily living, and sports are all included in the SAFAS.

36.5.2 Sports Ankle Rating System (SARS)

The sports ankle rating system (SARS) has been developed to assess functional outcomes in patients with ankle injuries and consists of the following three instruments: The Quality of Life Measure (QOL), the Clinical rating score, and the Single Assessment Numeric Evaluation [14]. The results of a study of Williams et al. [14] showed that the SARS is effective at assessing the impact of an ankle sprain on an athlete's functional and psychological status; is responsive to changes in an athlete's ankle-related health status; and is valid and reliable in the assessment of the functional and psychosocial status of athletes with lateral ankle sprains. The authors chose to validate the SARS in patients having sustained an ankle sprain, as this is not solely the most commonly occurring injury of the lower extremity in athletes, but also the most frequently occurring injury in athletes overall. Further research concerning this specific system of scoring functional outcomes in athletes should focus on validating the score in athletes suffering from other more specific pathologies of the foot and ankle.

36.5.3 FAAM-Sports

The Foot and Ankle Ability Measure (FAAM) is a specific tool for foot and ankle problems and has been developed with the objective to create an instrument that can be used to evaluate changes in self-reported physical function for individuals with leg, ankle, and foot musculoskeletal disorders for athletes and nonathletes [4]. The FAAM consist of two subscales: the activities of daily living (ADL) subscale and the sports subscale. The sports subscale, on its turn, consists of eight questions aiming at assessing the level of difficulty of specific sporting activities related to the movement of the lower extremity. The subscale, for example, consists of questions concerning jumping, running, landing, performing low-impact activities, and the level of ability of being able to participate in the desired sport of the patient. A study by Carcia et al. [3] indicated that scores on the sports subscale of the FAAM were greater in healthy athletes compared to athletes with chronic ankle instability (CAI) and were greater in athletes who indicated that their ankles were normal compared to athletes who indicated that their ankles were nearly normal or abnormal [3].

36.5.4 FAOS Sports

Although the previous chapter in the present book focused on the Foot and Ankle Outcome Score (FAOS) in general, this chapter will devote some of its information to the specific sports outcome subscale of the FAOS questionnaire [13]. The questions focusing on the sports outcomes of a patient with foot and/or ankle pathology consist of a degree of difficulty when performing specific tasks over the past week. These tasks consist of running, squatting, jumping, twisting, and/or pivoting the injured foot/ankle, as well as kneeling. The FAOS is however more suitable to assess clinical outcomes at group level than for monitoring specific patients or athletes [15].

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37 Advances in Rehabilitation Techniques

Konstantinos Epameinontidis, Mohsen Abassi, and Pieter D'Hooghe

37.1 Introduction

Injuries and disorders of the lower leg are very common in athletes, particularly in high-impact and contact sports. Clinicians constantly face the challenge to assist athletes in restoring function to pre-injury levels within the shortest possible timeframe. Current trends in sports rehabilitation embrace this challenge and promote concepts and techniques which provide evidence of a speedier return of homeostasis and function of the injured area, while carefully monitoring the load imposed on healing tissues. In this chapter, we will present two different tools used at our institution, which assist in training of the lower limb and assessment of the functional status of the ankle joint. The interventional technique is called blood flow restriction (BFR) training and the assessment tool is a new device called QF-AROM for the ankle joint.

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37.2 Blood Flow Restriction Training in the Rehabilitation Setting

Athletes need to continuously provide training stimuli to their bodies in order to develop beneficial adaptations and improve performance. The same principle applies during all phases of sports rehabilitation, but each phase has specific goals to achieve, while certain restrictions may apply (e.g., immobilization, or non-weight-bearing conditions). It is widely acknowledged that the necessary initial protection of the injured limb, with motion and/or weight-bearing restrictions, will impose some strength loss and reduction of muscle volume. Clinicians utilize therapeutic exercise regimes in an effort to counteract the negative effects of restrictions and activity limitations. Low-intensity or low-load exercises, when used appropriately, may reverse some of the negative effects on joint range of motion and local muscle activation, but do not offer sufficient stimulus for strength development and muscle growth. According to the American College of Sports Medicine's Position Stand on resistance training, strength and hypertrophy gains can be achieved with training loads that exceed 65–70% of one repetition maximum (1 RM) [1]. However, the use of these high loads may exceed the current loading capacity of the injured tissues and should be avoided, at least in the early phases of rehabilitation.

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Recently, it has been proposed to perform low-load exercises (up to 30% of 1 RM) with the addition of blood flow restriction (BFR) to the exercising limb [2]. BFR is applied with the use of inflatable cuffs or elastic bands to the most proximal part of the limb. The aim of this technique is to stop the venous return from the muscles distal to the cuff and partially restrict the arterial inflow to those muscles. The restriction in the normal blood flow creates a hypoxic environment and blood pooling, which in turn trigger various biochemical cascades that lead to upregulation of muscle protein synthesis [3]. BFR originated in Japan and since the late 90s there's extensive scientific evidence suggesting that low-load training with BFR (LL-BFR) induces significant gains in muscle strength and hypertrophy, comparable to high-intensity resistance training (HIRT) [4]. The obvious advantage of utilizing LL-BFR training on patient populations cannot be overemphasized, as it provides the benefit of the essential muscle strengthening stimulus, without the threat of damaging the healing tissues with high mechanical loads.

37.2.1 What Is BFR and How Does It Work?

BFR is applied with the use of inflatable cuffs or elastic bands to the most proximal part of the limb. The aim of this technique is to stop the venous return from the muscles distal to the cuff and partially restrict the arterial inflow to those muscles. The restriction in the normal blood flow creates a hypoxic environment which in turn triggers various biochemical pathways that lead to muscle protein synthesis. Various mechanisms of BFR action have been proposed in the scientific literature; however, scientists still do not agree on the predominant mechanism. Proposed mechanisms include hormonal responses (i.e., increases in growth hormone), translation initiation via intracellular pathways (mTORC1), metabolite accumulation (i.e., lactate), increased fast glycolytic fiber type recruitment, increased satellite cell activity, and muscle cell swelling [5]. It appears that during the use of BFR without exercise, the predominant mechanism of action is cell swelling [6]. When low-load exercise is used, multiple mechanisms may play a role in the hypertrophic and strengthening effects observed with BFR training [5].

37.2.2 How Is BFR Applied in the Clinical Setting?

In the rehabilitation setting, inflatable cuffs are preferred for BFR use since they provide graded exposure to restriction pressure and allow for precise measurement of pressure applied via the use of a manometer. Complete arterial occlusion is not desirable due to increased risk of side effects, while the extra pressure needed for complete occlusion does not seem to offer greater benefits than partial occlusion. Various percentages of arterial occlusion have been reported in the literature, with benefits observed even with pressures as low as 50 mmHg. The most commonly used pressure is at 80% of arterial occlusion, which can be easily measured with the use of a portable Doppler ultrasound unit (commonly used for evaluation of fetal sounds in the uterus) (see Fig. 37.1). Maximal occlusion pressure is reached when the sound emanating from posterior tibial artery is not audible. Then, the pressure of the cuff is adjusted at 80% of that maximal arterial occlusion pressure.

37.2.3 How Can It Be Used in Rehabilitation of Foot and Ankle Injuries or Surgery?

BFR can be used in all stages of rehabilitation of an athlete. Loenneke et al. [7] proposed a staged model of BFR application. The first stage involves BFR during immobilization, the second stage involves low-load aerobic activities like walking and cycling with BFR, and in the third stage BFR is utilized with low-load resistance exercise to promote maximum strength and hypertrophy benefits. During periods of prolonged immobilization, the cuff is inflated at 80% of maximal occlusion pressure for 5 min and then deflated for 5 min for reperfusion to occur. This process

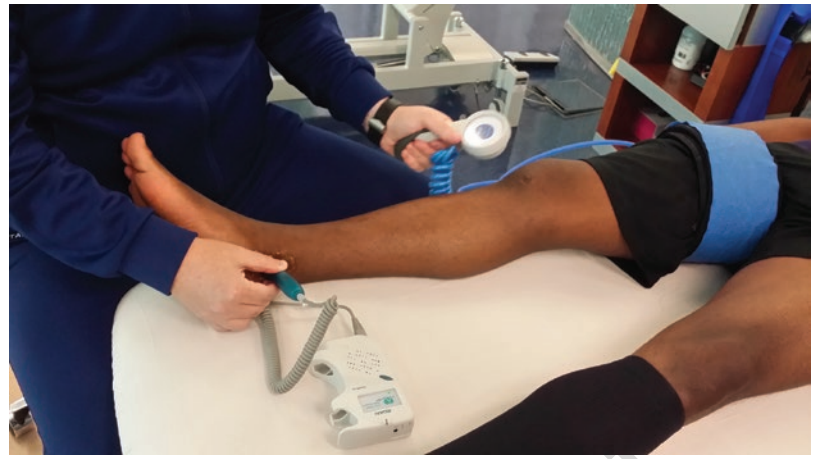

Fig. 37.1 Determining maximal occlusion pressure via Doppler US device

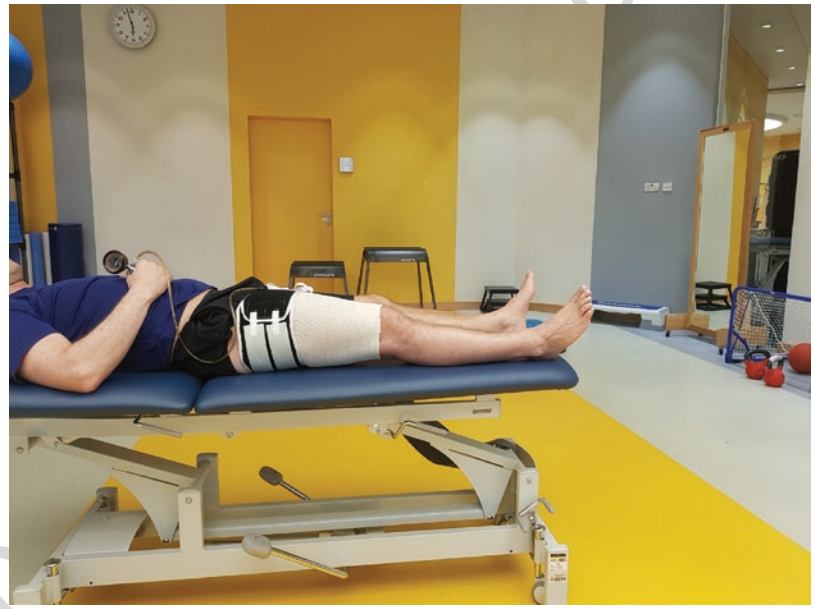

Fig. 37.2 BFR use during immobilization period

is repeated five times during a session and it can be repeated five times during the day (Fig. 37.2).

It has been demonstrated that BFR use during periods of complete immobilization can attenuate the loss of strength and muscle volume in postoperative patients [8]. When the patient is allowed to use active ROM (either full, or partial), BFR can be used with active movements of the ankle in all directions until muscle fatigue ensues. Light elastic resistance can be added when appropriate (Fig. 37.3).

In cases where partial weight-bearing (PWB) activities are indicated and allowed, BFR can offer substantial muscular load without jeopardizing the injured ankle. An example of PWB exercise with BFR is seen in Fig. 37.4.

When weight-bearing exercises are allowed, then BFR can be utilized in conjunction with walking (Fig. 37.5) or cycling and offer the advantage of muscle strength and cardiovascular endurance gains for the recovering athlete. Twenty minutes of walking at 45% of maximum heart rate has been found to increase both cardiovascular fitness and offer small but substantial improvements in thigh muscle strength. These strength gains are not evident when such low-load activities are performed without BFR [9].

Fig. 37.3 BFR with light elastic resistance

Fig. 37.4 Partial weight-bearing exercise with BFR

When resistance exercise at low-loads is allowed by the athlete's condition, then BFR can offer its greatest advantages. Resistance can either be applied by own bodyweight, elastic resistance, or gym equipment (Fig. 37.6). Scott et al. [3] provide a concise overview of the acute variables of BFR resistance training, based on best available evidence. Four sets are proposed with the following order of repetitions: 30, 15, 15, 15. Rest interval is set at 30 s and the cuff stays inflated in order to enhance the effect of metabolite accumulation. In healthy populations, 30% of 1 RM is the most common resistance load applied during BFR exercise; however, in patient populations 1 RM testing may not be feasible or safe. In our insti-

Fig. 37.5 Walking on treadmill with BFR use

tution, we propose that clinicians incrementally increase low loads until the athlete completes the assigned set and repetitions and reports a rate of perceived exertion of eight out of ten at the end of each exercise. Multiple exercises can be used at this stage; however, it is advised not to exceed a total time of 30 min per session. Five minutes of rest between BFR exercises has been proposed in order to allow reperfusion to the leg muscles and enhance the hypertrophic effect.

When the athlete is allowed to train with high loads, LL-BFR can be an excellent tool to assist in the appropriate management of training load during the final stages of rehabilitation. By alternating sessions of high resistance training with LL-BFR, the athlete is exposed to beneficial training stimuli while the injured ankle is spared from overload.

37.2.4 Is Training with BFR Safe for the Patients?

As with any form of exercise, BFR training has its limitations and contraindications. Patients with severe cardiovascular disease and peripheral vascular disease are not candidates for BFR training. BFR training with low-loads elicits similar hemodynamic responses to high-intensity resistance training without BFR and should not be a concern for athletes who are regularly exposed to this stress. Interestingly, BFR training with low loads does not seem to induce muscle dam-

Fig. 37.6 Low-load resistance exercise with BFR on the leg press

age compared to high resistance training and is therefore a safe option for patients with concerns for muscle tissue function. However, caution is needed with patients that have not been exposed to any type of training for long periods of time in order to avoid acute reactions of overtraining. For an excellent review on safety consideration of LL-BFR training, please refer to Loenneke et al. [10].

In summary, LL-BFR training is a recently introduced modality in the rehabilitation setting that can promote muscle strength and hypertrophy of the lower limb while avoiding detrimental loads to the healing tissues. When used appropriately, it is a safe procedure and can greatly enhance the efficacy of our rehabilitation protocols.

37.3 Assessment of Range of Motion in the Rehabilitation Setting: What's New?

Range of motion (ROM) of the ankle is of a high importance in restoring its normal function as well as the whole body functionality such as walking, running, and jumping and thus it is very

important to restore full ROM after an injury or surgery to allow the athlete to return to full sports activity. Measuring ROM is not an easy task as we are used to utilize the traditional goniometer, whose reliability is clinician-dependent, as the landmarks used and positioning can change from one practitioner to another. On the other hand, using the universal goniometer in a supine position doesn't reflect the true actual ROM of the ankle due to unnecessary tensioning of some antagonists, but also due to the non-functionality of the ankle in that position.

If the ankle ROM is measured in a functional manner, these measurements may then best reflect the different ankle positioning situations and so the real ankle ROM during playing. Unfortunately, this is missing in the literature and nothing was found to respond to these needs except the lunging position for ankle DF measurement due to its mounting evidence of being a risk factor for ankle sprains. Actually, while the DF is being measured functionally with knee to wall position using a ribbon meter to take the big toe to wall distance or using an inclinometer at the anterior side of the tibia shaft, the three other ankle ROM directions are forgotten and we don't know much about them yet.

Recently, we developed a device that can measure the main four ankle ROM positions (DF, PF, INV, EV) of the ankle in a realistic functional manner mimicking playing situations such as tackling, jumping, and changing directions.

37.4 Device

Our apparatus (QF-AROM) includes a base, a pair of sidewalls, a 30° inclined upper surface and a foot retainer 180° rotatable, secured to the inclined upper surface and serves to receive the patient's foot. A sliding mounting plate is attached to one of the pair of sidewalls, such that it is selectively moveable along a direction parallel to the inclined upper surface for proper positioning with respect to the patient's foot being measured.

An elongated rod, having opposed upper and lower ends, is further provided, with the lower end attached to the mounting plate vertically to the inclined upper surface. This lower end serves to calibrate the rotating rod (vertical to the inclined upper surface). An inclinometer, such as a digital inclinometer, angle sensor, or the like, is secured to the upper end of the elongated rod to measure the angular displacement of the elongated rod with respect to the inclined upper surface of the device.

A retaining bar is secured to the elongated rod, adjacent to its upper end extending substantially orthogonal to it and adapted to be positioned adjacent to the leg and slidably mounted on the elongated rod to adjust its height in order fit with the mid-shaft of the leg, allowing the device to measure the ankle ROM in patients having varying heights and body types.

37.5 Testing

The patient stands upright behind the device using one hand as support against a wall and then places his uninjured foot in the foot retainer which is locked in the sagittal plane; once the foot is secured to the retainer with straps, the mounted sliding plate (holding the rod) is moved to align with talo-crural joint axis, the rotating rod is calibrated vertically to the inclined upper surface, and the digital inclinometer is zeroed in this starting position. The patient is then asked to perform his maximum PF in the talo-crural axis by bringing his body backward so that the leg is pulled to its maximum tolerated position without losing any plantar foot contact with the foot retainer. When the patient reaches his maximum PF the rod is rotated until the retaining bar touch the leg and the angular displacement shown in the inclinometer is recorded as Full Functional PF ROM (FF PF ROM). Next, the patient is asked to make anterior lunge until maximum DF is obtained in a knee over toe technique (avoiding excessive pronation), while keeping the heel flat in the foot container, where angular displacement on the inclinometer is recorded for FF DF ROM. Once completed, the foot retainer is unlocked and the patient is instructed to move to the side of the device (next to one of the side-walls) rotat-

ing the foot retainer 90° with his foot until it is aligned with the axial plane of the device where it is locked. The patient then stands next to one of the sidewalls in a position allowing measurement of inversion. The patient is instructed not to lift the lateral border of the foot from the retainer while stretching into inversion. The sliding plate is then repositioned to fit with the rotational axis of the foot. Once the inversion angle is recorded, the patient moves to the opposite side for eversion measurement with instructions to keep his medial foot border completely adherent to the foot retainer. The technique is then completed for all four measures on the injured side.

37.6 Conclusion

In our study on 87 male athletes with injured ankles who reached sports-specific stage of RTP and 25 healthy athletes we found Statistically significant reductions in range of motion with moderate to large effect sizes for plantar flexion, dorsiflexion, and inversion and not for eversion. Plantar flexion had the largest reduction and eversion had the smallest when comparing injured to healthy group. Standard errors were 2.1° for PF and 4.1 for Ev. Good reliability was for DF (0.76–0.87) and Inv (0.75–0.86) and excellent for PF (0.9–0.95), however it was fair with Ev (0.49–07).

This innovative device showed the ability to measure the functional ankle ROM as well as highlights the reduction in PF ROM at time of RTP which may help clinicians to improve their rehabilitation protocol and most probably reduce the re-injury rate.

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38 Foot Orthotic Advances for the Athlete

Craig Tanner and Pieter D'Hooghe

38.1 Background

Foot orthoses are a well-established tool in the prevention and/or management of a wide range of sports injuries of the lower limb. The simple act of inserting a molded insole into a shoe can have a significant preventative effect on lower limb injuries [1]. Foot orthoses are relatively inexpensive and, if prefabricated, can provide almost immediate benefit. In addition, the risks of side effects are minimal as they can be easily removed if a potential problem arises.

Their use, however, lacks standardization. Foot orthotic therapy varies greatly around the world in both theory and application. Practitioners that produce or prescribe orthoses come from a range of professions and there can be quite diverse approaches to the management of the same pathology [2, 3].

Irrespective of the design rationale, the digitization of the design and manufacture of custom foot orthoses (CFOs) at the practitioner level is a significant recent advancement in lower limb sports medicine. In order to appreciate why, it is useful to understand the differences in comparison to the traditional process of design and manufacture.

38.2 Traditional Manufacture of Custom Foot Orthoses

The manufacture of CFOs is a multistep procedure. The whole process may be performed by the individual practitioner, but a substantial component is often undertaken by commercial foot orthotic laboratories following a written prescription. Almost every stage is open to variability and individualization.

In order to capture the geometry of the foot, foam impression boxes, plaster slipper casts, or vacuum bladders are some of the more common traditional methods utilized. All of these methods create a negative model, which may be manipulated by the practitioner by having the foot weighted, unweighted, or semi-weighted. All of these options will affect the shape differently.

From this, a positive model of the foot is then constructed—most commonly with plaster. In many instances, the model may not be modified further and this is the extent of the "customization."

Over the years, however, different philosophies have emerged as practitioners have strived for improved effectiveness. The positive model may

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be modified significantly to implement specific changes. A "prescription" may be applied involving intrinsic posting, skiving, and shaping of the design based on the requirements defined by the practitioner [4–6]. Depending on the theoretical aim of the device, the shape may be altered significantly from the original model. Although the resultant orthoses may appear similar, these changes can have a significant impact on how forces act across the foot.

The orthosis is created by heating, molding, and shaping a material around this positive model. A wide range of different materials may be used ranging from Ethyl Vinyl Acetate (EVA) or Polyurethane (PU) foams of various densities, to harder shell materials such as polypropylene or carbon fiber composites. These materials may have varying stiffness properties through the availability of different thickness blanks.

Following the molding, further modification is required to remove excess material and allow the orthosis to be fitted into a shoe. Finishing may also involve fixing additions such as stabilizers, pads, and wedges as well as a final top cover.

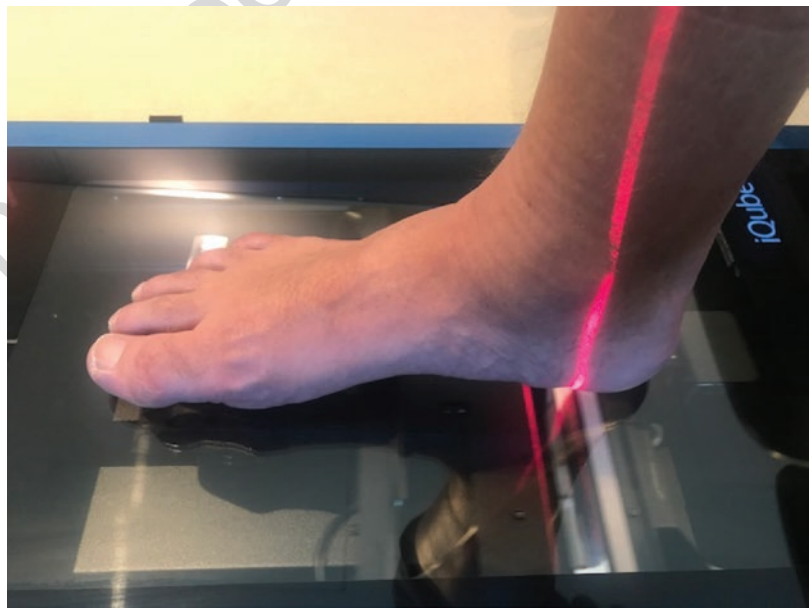
38.3 Digital Manufacture

Over the past 20 years the digitization of this process has evolved. CADCAM (Computer Aided Design Computer Aided Manufacture) has been introduced and has completely changed how orthoses may be produced.

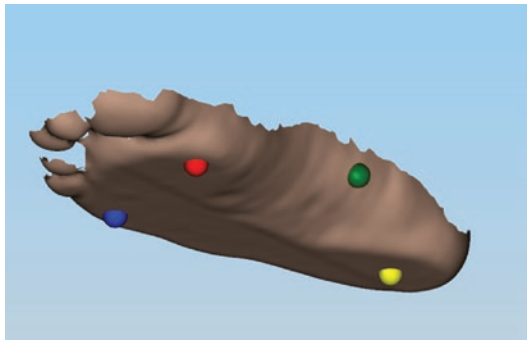
Due to the large initial investment being required, it was commercial laboratories that first implemented this technology. A variety of scanning systems were used to digitize the traditional plaster casts or foam box impressions. There was no need for practitioners to change or invest in new technology.

More recently, the technology has become economical enough that many practitioners will have a scanner which allows direct capture of the foot shape. In addition to the obvious benefits of being cleaner for both the practitioner and patient, it is a significantly faster process. The speed of direct digital capture means that it is feasible for multiple scans to be taken at once for later reference if so desired.

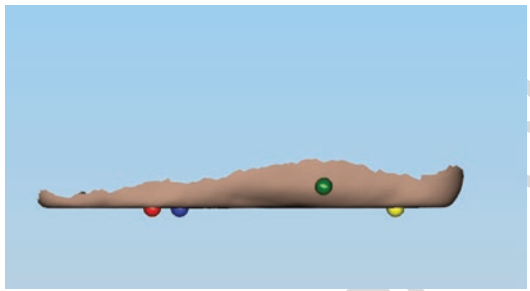
Partial weight bearing scan with 3D laser scanner



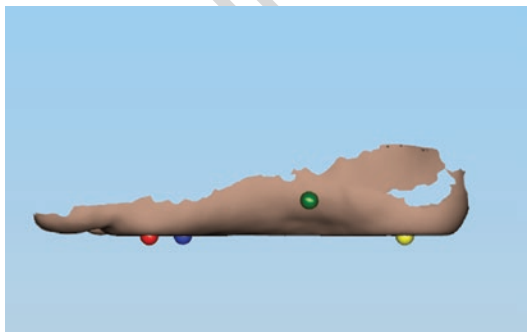
As with traditional capture methods, there are many options as to how this can be taken. The choice of technique used can obviously have a significant impact on the shape of the final orthosis.



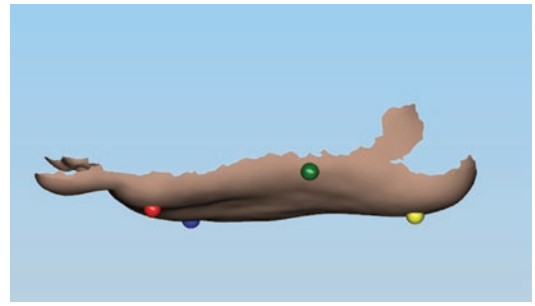
Resultant digital scan from laser scanner (Partial Weight Bearing Scan). Markers on central plantar heel, first and fifth plantar MTPJ and point of medial arch apex



Medial view of full weight bearing scan—Lowest medial longitudinal arch profile. Soft tissues distorted by the surface



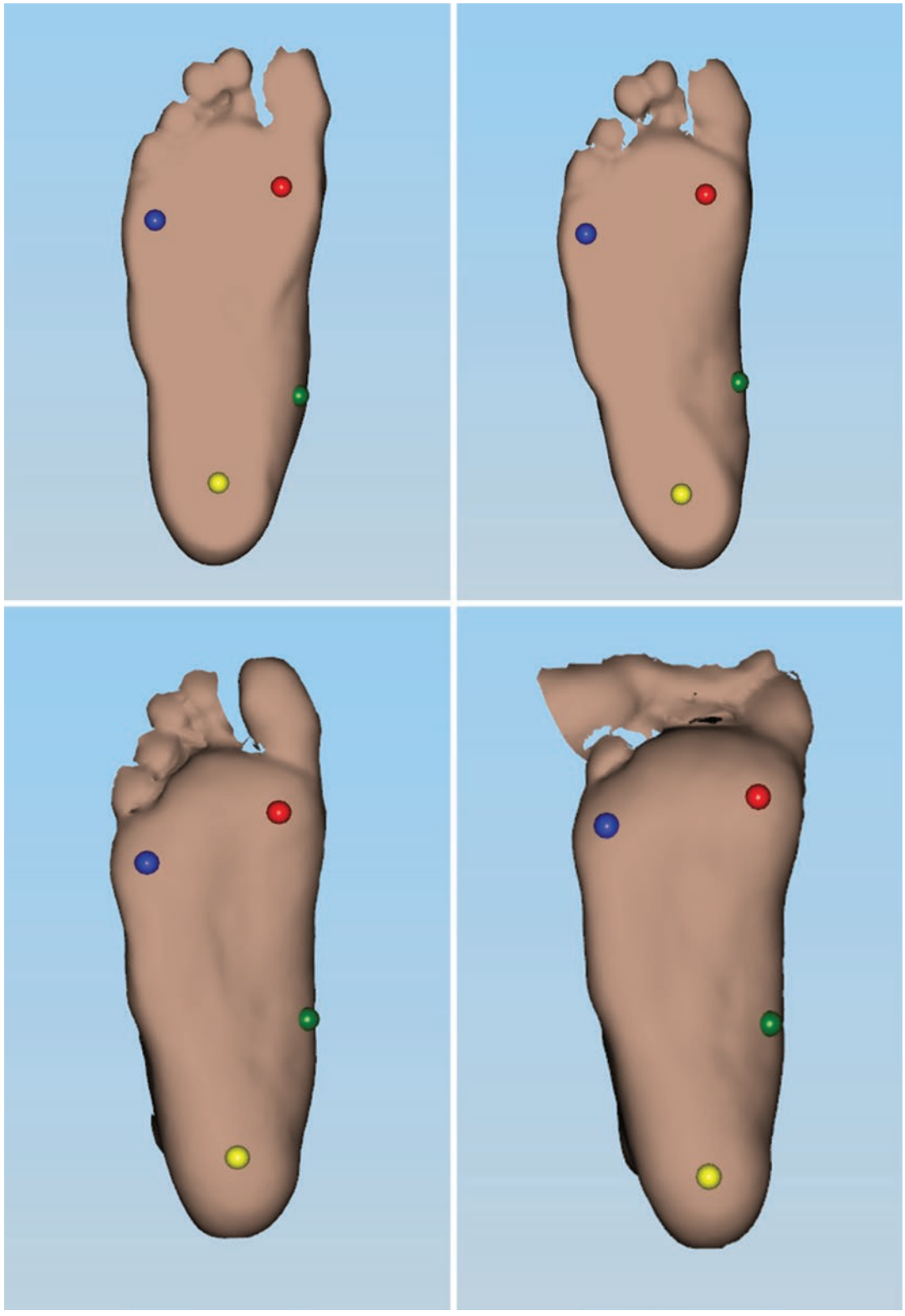
Medial view of partial weight bearing scan—Arch profile unloaded and therefore higher, but soft tissue structures distorted by the surface



Medial view of non-weight bearing scan—Foot is above the surface of the scanner glass

Medial view of non-weight bearing scan with practitioner dorsiflexion of the first MPJ. The windlass mechanism creates the highest medial longitudinal arch

Whether directly scanned or a scanned cast, the digital master copy of the foot may be saved indefinitely. This may be useful as a reference point in future designs. In many traditional methods, if a new design is indicated, a new physical model must be produced by re-casting.



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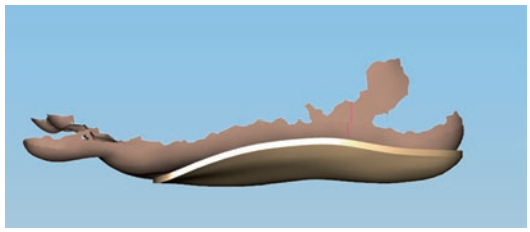
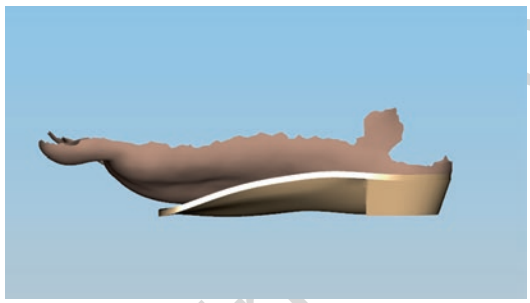
Plantar View of four scanning conditions—From top, left to right, Full WB, Part WB, Non WB, Non WB with MTPJ dorsiflexion. Clear differences in foot geometry can be seen

Digital design essentially bypasses the steps involved in model manufacture and proceeds to directly designing the finished orthosis. Many additions that would usually require changes to a model can be applied directly to the digital design. Importantly, this is can be performed in steps to create multiple designs with small variations. Until the last few years, this was the realm of laboratories only. Individual practitioners now have access to CAD software that allows the full design to be manipulated on a computer monitor in real time.



A foot orthosis designed to follow foot contours



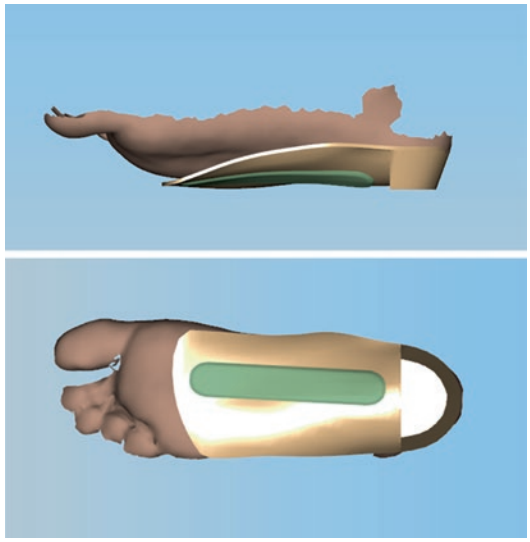
The same foot, but now with an inversion modification. This shape of the device is the same apart from the point of contact under the heel and lateral column. This technique would traditionally involve replication of the arch of the foot in plaster when the model is produced [4]. Research suggests that can be an effective way of applying an inversion force to the plantar foot [7]

The creation of a physical orthoses from a digital design uses CAM software to create a toolpath for a computer numeric controlled (CNC) carver or router. This is known as "subtractive rapid prototyping" as the design is created by carving, or subtracting, material out of a solid block.

The hardware required for this is scalable. Large commercial laboratories are able to manufacture dozens of pairs of orthoses at once. It is, however, now also feasible for practitioners to do the same on a much smaller scale.

As with traditional manufacturing, a variety of materials with differing characteristics may be used. Rather than different thickness blanks, a shell type of orthosis may be machined by varying tool offsets to alter the thickness and therefore manipulate stiffness. Sections of the device may also be modified discretely to provide more flexible—or stiffer—areas with minimal weight changes.

CADCAM EVA and Polypropylene foot orthoses. Same digital design, but machined out of different material



Previous design with examples of modifications. The material is thinner overall which allows for flexibility, but it is reinforced with an element that will maintain longitudinal stiffness. The extrinsic heel stabilizer is smaller in length which will decrease longitudinal stiffness. There is an accommodating flare added under the talo-navicular area

CADCAM technology has improved efficiency in every step of the process of manufacture. Traditional methods require plaster to cure, and for material to heat and cool. Additionally, there is significant labor involved. In realistic terms, a laboratory would allow at least 48 h for this as a "rapid turnaround" service to produce a CFO. CADCAM design and manufacture can produce a fully bespoke pair of foot orthoses in less than 60 min.

The vastly improved efficiency of digital manufacture is fundamental as to why this is such an important advance in orthotic therapy application. In order to understand why, it is useful to briefly review the research into foot orthoses and their mechanism of action.

38.4 Foot Orthoses: Implications of Research

Research into foot orthoses and their relationship to pathology is complicated for a number of reasons.

The first consideration is the wide variety of approaches used around the world with conflicting terminology and definitions. As described previously, a custom foot orthosis can vary between a device that is simply a soft insole that is molded to shape of a foot, to a stiff material that has been molded around an extensively modified model. Yet researchers often do not make any distinction between these approaches, defining all orthoses that are based on a foot model as being custom.

In addition, there is significant conjecture as to the theoretical basis and mechanism of action of foot orthoses. The traditional view that they act to support or align the lower limb has very little evidence to support it [8, 9]. Extensive research into potential mechanisms have found no single answer. There is evidence that foot orthoses influence kinematic variables such as rearfoot eversion and tibial rotation as well as kinetic variables such as loading rate and vertical impact forces [10]. There is also good evidence of neuromotor effects through EMG studies, suggesting a role in altering muscle loading and function [10, 11].

However, the most important recurrent observation of all of this research is there is significant variability in responses to the different orthoses tested. Applying the same intervention to different subjects often gives very different effects. A range of simple wedges applied to a pre-made orthosis demonstrated inconsistent, subject-specific responses which were also often contrary to what was expected [12–14]. A similar conclusion was noted in respect to center of pressure (COP) and knee joint moments [15]. Identical interventions can have substantially different results for different subjects.

This observation is perhaps not so surprising when you consider the high level of variability observed when foot motion is closely examined [16, 17]. For this reason, traditional models that suggest there is a measurable "normal" foot have faced criticism [8, 9, 18, 19]. Newer paradigms may still approach orthotic design based on an individual cast or scan, but with tailored modifications designed to alter load on structures by shifting the application of forces on the plantar surface of the foot [5, 20]. Orthoses that changes kinetic variables have shown to be effective when

previous designs have failed. It should be noted that even within these subjects the responses are variable [7].

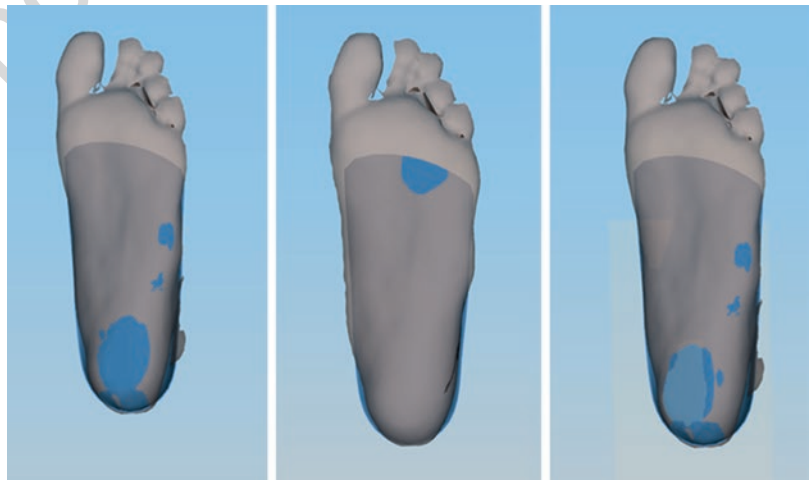
This variability impacts the relevance of some research into the effectiveness of foot orthoses in injury management. The challenges of high-level research design will lead to a standardized intervention protocol either through the use of prefabricated orthoses or through a predefined design of CFO. However, it should be expected that the generic application of an orthotic intervention to a cohort of individuals will have variable effects within the group. The most robust design studies do not account for individualized interventions. The response therefore is likely to significantly milder that it could potentially be. Meta-analyses on foot orthoses tend to be comprised of studies like this may and therefore report small effects if any at all [21, 22].

It is argued that this limits the relevance of such studies as they do not reflect usual clinical practice. There is a growing contention that foot orthoses should be viewed in a similar way to drugs in the pharmaceutical industry whereby a practitioner should be aiming for a "dose" which is best for the individual [23]. Based on the evidence mentioned above, this is not likely to be a simple linear relationship, but rather a solution which is specific tailored to the individual.

The ability to make precise incremental adjustments to a CADCAM orthotic design has allowed some exploration of the concept of orthotic dosing. The efficiency and accuracy of production allows many versions of a foot orthosis to be manufactured from the same scan and then modified in steps. This has yielded some linear relationships, but also much variability [24, 25].

The limitation even with this type of research is that it is only relevant to a certain prescriptive approach. However, one significant advantage of CAD software is that it does not tie the practitioner to any particular approach, or to variables that are defined by an external laboratory. It allows a practitioner to have the same level of design control as if they are making a solid model themselves. The only difference is that the model is on the computer screen.

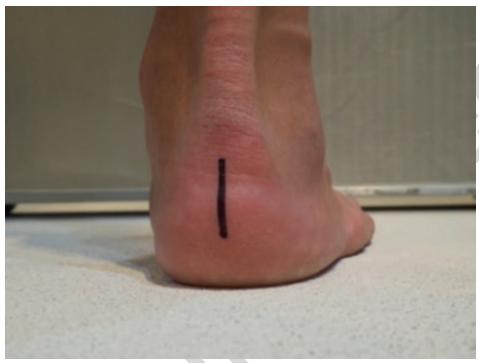
This highlights why the use of CADCAM orthoses at the clinic level is such a significant advancement. A practitioner is able to make an individual design decision and then view the resultant effect. The improved efficiency, repeatability, and accuracy of the technology means that it is now viable to make specific design variations directly based on clinical response. Adjustments can be made almost immediately to a wide range of parameters.



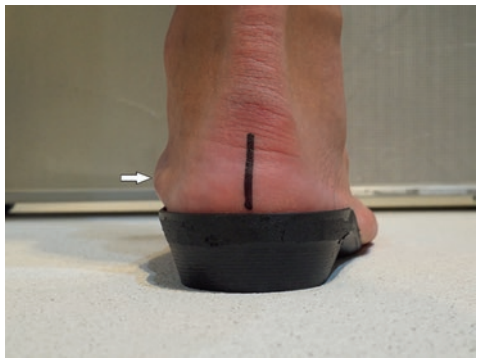
Left: 15I Inverted orthosis; Center: Same device with 15° 4 mm Medial heel Skive; Right: Same with 25° 4 mm Medial Heel Skive. All these modifications should apply a slightly different inversion force around the subtalar joint when applied to the same foot

In simple terms, an individual practitioner can design and modify an orthosis to their specific requirements and know that the end result is a reflection of this. Any subsequent modification of the design will also be more specific and accurate.

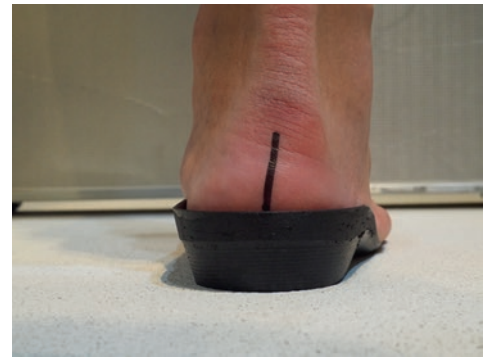
Left: 15° medial heel skive; R 25° medial heel skive. This is the manufactured previous digital design. The flat area of the heel applies a force in slightly different direction in relation to the heel. The two devices are otherwise identical



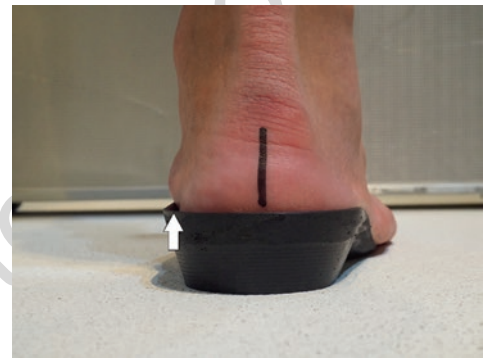
Resting foot position



Static assessment of foot orthosis shows good fit, but bulge in soft tissue under talo-navicular region may indicate increased load on the medial arch



Static assessment—Orthosis has an inverted modification. Force on the medial heel applies an inversion moment which decreases direct pressure on talo-navicular area, so previous bulging is less evident



Static assessment—Further increase in inversion through medial heel skive modification now suggests less control. Foot is being shifted laterally as evidenced by the small gap to the medial heel cup edge. In this case, more is less

It is important to understand that when foot orthoses are used as part of a management plan, they need to be assessed as to whether they are working as intended. The first step in the application of orthotic therapy is establishing why an orthosis may have a benefit and how it should be designed to achieve this. Reviewing orthoses should therefore assess whether the response reflects the design aim.

Some outcomes such as resolution of pain are obvious. The solution, however, must also be comfortable and functional. This is even more critical in high-level sports. The design versatility and manufacturing efficiency of CADCAM orthoses allows this in a more predictable and repeatable manner.

Manufacturing efficiency allows additional orthoses to be produced with design adjustments in response to slightly different footwear or activity requirements. It may be advantageous to have a slimmer orthosis in a football shoe in comparison to a running shoe. Another option may be to adjust material thickness in certain areas to allow flex points or decrease weight.

38.5 The Future

The main disadvantage of this technology is that the production is not efficient with respect to material use. Subtractive manufacture is wasteful with perhaps 95% of material carved away. 3D printing—also known as additive manufacture—has now entered the market place. The capital cost is high, however, and it is not as fast as subtractive manufacture when manufacturing small volumes. The ability to make large quantities at once, at this stage, makes it only viable for commercial laboratories.

3D printing technology has clear environmental advantages as there is negligible material waste. It also creates additional possibilities for manufacturing with even greater freedom of design. As an example, foot orthoses can be produced with a lattice structure for decreased weight. It is reasonable to believe this will be the dominant type of manufacture in the near future [26].

38.6 Summary

Digital foot orthotic design and manufacture:

- Allows fast and accurate production of foot orthoses.
- Allows high level of design versatility that is under the control of the practitioner.
- Can allow the practitioner to account for the high level of variability of patients and how they respond to an orthosis.
- Is repeatable and modifiable to allow orthoses to be easily applied across different footwear and situations.

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