Ankle Joint Arthroscopy

A Step-by-Step Guide

Francesco Allegra
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Editors

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Preface

Arthroscopic procedures on knee and shoulder joints developed during the 1980s and the 1990s, becoming a new tool at surgeon's disposition to address surgery in different compartments often hidden to open procedures. Arthroscopy has inaugurated a new epochal period in joint surgery, offering a direct capability to examine each side of the articular spaces, to reevaluate the anatomy, and to redefine the reasons and the nature of articular disorders even in complex and serried joints. This technique had been destined to be more than a simple curiosity for the surgeon, developing instruments and their appropriate clinical applications and defining indications and risk factors. Its dissemination of adequately proper knowledge in the scientific world allows surgeons to become technical experts, by using new methods and procedures. Examining what happened for major joints by becoming these arthroscopic procedures more popular, ankle arthroscopy proceeded on to a discover of its latent power. By years, new procedures, new techniques, new approaches to further joints like great toe and subtalar, new tool utilization in exploring tendon sheaths, and new algorithms for rehabilitation were developed. Pushing ahead this development, in the last 6–10 years, ankle arthroscopy changed dramatically the possibility to treat many further disorders around and inside the whole "ankle system," continuing to improve the outcome of our patients.

When we decided to write this book on ankle arthroscopy, we remain doubtful. Books on this topic edited by scientific experts, are available in the market. Comparing to those, we asked ourselves which kind of added value it should belong to. The personality of a written paper is strongly influenced by its author, who puts his/her own imprint in each word, sure not to disappoint readers' expectations. The author establishes a direct relationship with them, capturing their attention through high-level topics and by using a captivating personal style of expression, to convince them to espouse his point of view. To avoid each author shadowing other authors' work, we decided to work together by sharing our personal surgical experiences and connecting continuously with each other to have the final result of our book, which is less influenced by our own personality. Moreover, we decided to commit each chapter to a different author, the most from the Italian Orthopedic School, to remark our will of default by editors' conditionings. When evaluating indications and techniques for a procedure like ankle arthroscopy, nothing serves the surgeon better than a single source to learn step-by-step operative techniques, looking after the hidden traps and the surgical tricks to avoid them.

By writing these recommendations in a textbook, we had the aim to fill a gap at the initial trial of ankle arthroscopy training. Furthermore, surveying current ankle arthroscopic techniques in an easy and planned way, we hope to help both beginners and practiced surgeons.

This volume is dedicated to those surgeons already familiar with arthroscopic techniques, who would like to broaden their knowledge on the subject of ankle disorders and who desire to complement surgical options with minimally invasive technique.

Latina, Latina, Italy — Francesco Allegra
Rovereto, Trento, Italy — Fabrizio Cortese
Forli/Cesena, Italy — Francesco Lijoi

Presentation

Foot and ankle surgery represent the dawn of my professional career. Therefore, when I was asked to make a presentation to this book, beyond the honor that has been accorded me, it was like reliving ancient emotions, perhaps the same ones that drove the authors to undertake a long and difficult journey, putting together an educational manual of arthroscopic surgery of the ankle.

Over time, what was once inaccessible in open surgery, despite the complications and the application limits connected to it, has become possible, thanks to the introduction of arthroscopy and of different cartilaginous reconstructive surgical techniques, which were not available before. In fact, arthroscopy has made possible treatments that otherwise would not have been applicable in this joint. It requires particular attention for its execution, and only those who—like the authors—have dedicated to this all their own interest and passion have been able to deepen and develop. Their contribution to ankle arthroscopy diffusion is achieved in a decisive way, which leads them to appropriately consider accomplishing a volume that could lead "step by step" those who want to approach this surgical practice that addresses more and more clinical applications.

In order for a guide to really lead the beginner to the final goal, he/she must be absolutely clear and provide all the necessary information. From the fundamental anatomy and arthroscopic ankle techniques, proceeding to define the various pathological-related aspects, the therapeutic pathways are provided from the first steps of the procedure up to the most complex techniques. Of great help in understanding and learning the anatomical aspects, arthroscopic technique is enriched by many illustrations, even by the subtitled videos of the different surgical procedures, downloaded with the use of appropriate technology on PC, tablet, or mobile phone. Moreover, this great interactivity with the reader is implemented by the presence in each chapter of a "traps and tricks" table, where every surgical trap is associated with a trick to solve it.

As President of the Italian Society of Orthopedics and Traumatology (SIOT), I can only applaud training initiatives such as this manual represents. I am sure that all those who will read this manual will be affected by the same enthusiasm that the authors have poured into preparing it, which proves how much they have cared about, sharing the knowledge acquired in their professional career with those who like to approach this kind of surgery.

Francesco Falez
Italian Society of Orthopaedic and Traumatology, President
Cesena, Italy

Introduction

The text is organized in ten parts as a "step-by-step" guide on the management of ankle joint disorders. Each chapter on surgical procedures begins with an introduction, to clearly explain them. The volume is supplemented by under-titled videos for single-detailed technique, proceeding from outside the cutaneous layer and inside the joint. After a short tuning on the opinions of selected experts quoted in the reference section, the volume explains each technique, starting from the patient's assessment on surgical bed to last surgical steps. Illustrations and drawings help the reader to understand each surgical sequence to permit to replicate in an easy way. After discussing the main issues concerning each procedure, a table shares with the reader the common traps in performing the procedure and the corresponding useful tricks to avoid them.

The gross anatomy of ankle and subtalar joint is the introductory. The aim is to make easy the comprehension of interconnection of singular anatomic structures related to its own conformation. Its relevant function is emphasized by illustrating pertinent extra-articular anatomy: it makes easy the knowledge of their mutual integration and the sharp correlation understanding between each anatomic structure with the others. Intra-articular anatomy is alike explained, helping to find the correspondence between internal and external joint structures and to reinforce each point of operative procedures.

The second part deals with basic ankle arthroscopy. The required instruments, surgical patient assessment, and intraoperative setup for ankle arthroscopy are concisely pointed out. They have been derived from knee arthroscopy and went to be completed with dedicated scopes, making possible to address surgery into narrow spaces. Intra-articular anatomy is alike explained, showing the correlation between internal and external joint structures and to reinforce each step of operative procedures.

Impingement of anterior and posterior ankle is the topic of the third part. Approaching for the first time ankle arthroscopy, the surgeon begins generally his experience with this kind of pathology, underestimating it should represent a surgical trap. To avoid it, the novice can learn to resect the right quantity of pathological tissue. Moreover, because the joint exploration itself can represent often difficulties, advices are suggested to have a clear visualization because of the abundant soft tissue presence. The chondral and osteochondral ankle joint defects part represents the fourth part of the volume. Beginning from the etiology, classifications, and mechanism of action, this offers to the reader many and various options in the treatment of these

disorders, ranging over the most traditional, the most actual, and the up-to-date ones.

The ankle and subtalar joint instability are treated in Part V. After explaining the classification, treatment, and arthroscopic procedures of acute ankle sprains, progress in instability knowledge greatly enhanced the understanding of these difficult problems. The most used arthroscopic procedures are presented by different authors, pointing out each advanced technique in ligament repair and offering a wide outlook of the actual way of ankle joint stabilization.

Several additional and seemingly insurmountable problems still exist in ankle disorders. Treatment for upper and lower joint arthritis and severe chondromalacia remains beyond arthroscopic surgical abilities. Behind the scene of Part VI, there is a preliminary discussion about etiology and classification, but there are pour techniques that may benefit the unfortunate patients who suffer from these conditions, except debridement and joint fusion, the only ones that can be performed by scope with safety for the patient.

A significant number of procedures inside small joints have been developed in times, thanks to the use of 2.7 and 1.5 arthroscope supplied by companies: these tools have opened the new frontiers of treatment of tendinopathies by scope. In Part VII are presented the arthroscopic treatments of disorders which affect the biggest ankle tendons, both placed in the anterior and posterior compartments.

Part VIII deals with the endoscopic treatment of plantar fasciitis. Despite this topic has been arthroscopically treated by a while, its diffusion as procedure remained delimited only to few surgical centers. This pathology is a common cause of heel pain in adults, but only less than 10% undergoes surgical treatment. The endoscopic resection of the plantar fascia allows good visualization of the entire structure and provides a minimally invasive technique with patient satisfaction and valid outcomes.

Part IX is dedicated to great toe disorders. Considering the limited application and difficulties to be carried out, the arthroscopic treatment of first metatarsophalangeal joint is relatively rare. Because of the many indications such as hallux rigidus, spotted osteochondral lesions, synovitis, soft tissue impingement, and the presence of symptomatic loose bodies, the consensus is increasing among surgeons, thanks to the availability of dedicated instruments that yield this surgery possible.

Part X contains a closing chapter devoted to rehabilitation, which greatly differs in patients treated with arthroscopic procedures and those submitted to open surgery. The after-surgery rehabilitation aims to restore the main three joint aspects of strength, proprioception, and range of motion, furthermore conceiving the rehabilitation plan based on the joint biomechanical request by patient's activity.

Contents

Part X Rehabilitation

Part I

Anatomy of Ankle Joint

1 Anatomy of the Ankle Joint and Hindfoot

Miki Dalmau-Pastor, Matteo Guelfi, Francesc Malagelada, Rosa M. Mirapeix, and Jordi Vega

1.1 Introduction

The ankle is a highly congruent synovial, hinge-type joint, in which the talus fits perfectly into the mortise formed by the tibial plateau, and the tibial and fibular malleoli. This anatomical conformation allows movement through only one axis, the bimalleolar axis, through which dorsiflexion and plantarflexion movements are produced. Normal values of the range of motion are 13–33° for dorsiflexion and 23–56° for plantarflexion [1].

The talus is an irregularly shaped tarsal bone. Articular cartilage covers more than 60% of its surface and it does not have any muscle insertions. Articular facets for the tibia and two malleoli are present in the upper, lateral, and medial parts of the talus [2]. On the upper side, the talar dome is convex on its anteroposterior axis and slightly concave on the mediolateral axis.

During ankle movements, some triplanar motion occurs at the level of syndesmosis to adapt to the varying width of the talar dome, wider at its anterior part. When the anterior part of the talus engages with both the malleoli (dorsiflexion), the fibula moves proximally and in lateral rotation, and distally and in medial rotation during plantarflexion. This provides stability to the ankle joint [3–5].

Like in every synovial joint, the joint capsule covers the articular surfaces of the ankle joint bones. But unlike in other joints, its anterior insertion is placed at a distance from the cartilaginous layer, about 4 mm proximally on the tibia and 2.5 mm distally on the talus [6]. This has some surgical implications, as it will

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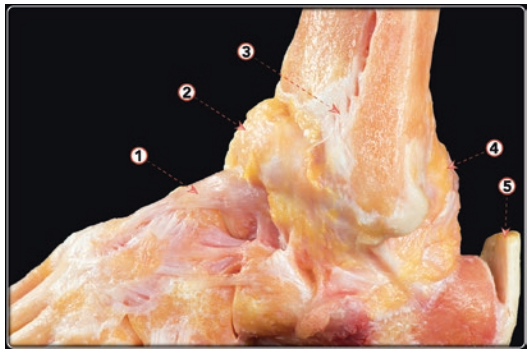

Fig. 1.1 Lateral view of an osteo-articular dissection of the ankle, where the ankle joint capsule has been preserved and injected with air. This demonstrates the anterior working area for ankle arthroscopy (1) Dorsal talonavicular ligament. (2) Anterior capsule of the ankle joint. (3) Anterior tibiofibular ligament. (4) Posterior capsule of the ankle joint. (5) Calcaneal tendon

allow resection of osteophytes on the anterior surface of the tibia or talus through arthroscopy (Fig. 1.1). It also has an anterior recess, more evident during dorsiflexion of the ankle, as the ankle joint capsule is taut in plantarflexion and relaxes in dorsiflexion.

Precisely this detail allows the creation of a working area during ankle arthroscopy. The high degree of congruency of the ankle makes difficult the introduction of instruments into the joint. But if the surgeon works with the ankle in a dorsiflexed position, the recess of the anterior ankle capsule permits to create the anterior working area and to work safely.

The posterior articular joint capsule also has a recess, but smaller than in the anterior part. The intermalleolar ligament, an intracapsular but extrasynovial ligament, reinforces the capsule and converts it into multiple small recesses that can be observed arthroscopically in the posterior joint capsule [7].

1.2 Ankle Ligaments

According to other authors, we divided the ligaments of the ankle into those that join the bones of the leg (tibiofibular or syndesmotic ligaments) and those that join the leg bones to the foot skeleton [1, 8].

1.2.1 Tibiofibular or Syndesmotic Ligaments

The distal parts of tibia and fibula are articulated through a syndesmotic joint known as the tibiofibular syndesmosis. This joint is stabilized by the tibiofibular ligaments, which ensure stability between the distal tibia and fibula and resists the axial, rotational, and translational forces that attempt to separate tibia and fibula [9]. There are three syndesmotic ligaments: the anterior tibiofibular ligament, the interosseous ligament, and the posterior tibiofibular ligament. In the central portion of the joint, tibia and fibula form a rectangular/oval recess that is located distally to the interosseous ligament. This recess is called the *synovial fringe*, and it contains adipose tissue. This tissue moves during ankle motion, retracting proximally in dorsiflexion and descending toward the ankle joint in plantarflexion [9]. Following a sprain, the synovial fringe may cause chronic pain in the ankle due to a syndesmotic impingement.

1.2.1.1 Anterior Tibiofibular Ligament (ATiFL)

This ligament originates on the anterior tubercle of the tibia. From that point and directed distally and laterally, it inserts on the anterior edge of the fibular malleolus [10] (Fig. 1.2). Its fibular insertion continues with the proximal insertion of the anterior talofibular ligament (ATFL). It is a ligament with a multifascicular appearance. The perforating branches of the peroneal artery are located among its fasciculi.

The distal portion of the ATiFL, which has been wrongly named Basset's ligament, appears to be independent of the rest of the ligament, since it is separated by a septum made of adipose tissue. This component covers the angle formed by the tibia and fibula, and is physiologically in contact with the lateral corner of the talus when the ankle is in the neutral position. A thickening of the distal fascicle of the ATiFL following an inversion trauma is often the cause of an anterolateral soft tissue impingement and may be associated with cartilage abrasion in the contact area. However, an impingement of this ligament may occur following increased anteroposterior translation of the talus

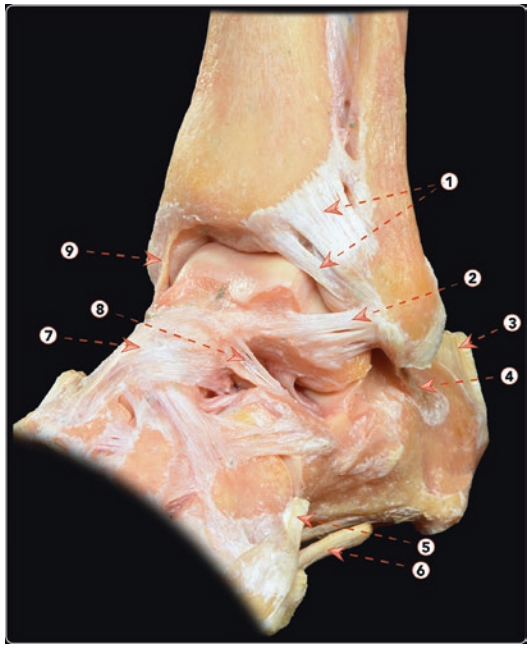

Fig. 1.2 Anterolateral view of an osteo-articular dissection of the ankle. (1) Anterior tibiofibular ligament (and its distal fascicle). (2) Anterior talofibular ligament. (3) Calcaneal tendon. (4) Calcaneofibular ligament. (5) Insertion of peroneus tertius tendon. (6) Peroneus longus tendon. (7) Dorsal talonavicular ligament. (8) Cervical ligament. (9) Anterior part of the medial collateral ligament

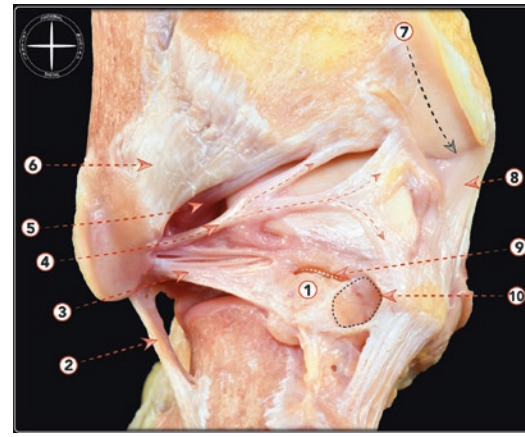

Fig. 1.3 Posterior view of an osteo-articular dissection of the ankle and subtalar joint. (1) Os trigonum. (2) Calcaneofibular ligament. (3) Posterior talofibular ligament. (4) Intermalleolar ligament. (5) Deep component of the posterior tibiofibular ligament (transvers ligament). (6) Superficial component of the posterior tibiofibular ligament. (7) Pathway of tibialis posterior tendon in the medial retromalleolar sulcus. (8) Posterior part of the medial collateral ligament. (9) Broken synchondrosis between talus and os trigonum. (10) Osteofibrous tunnel for flexor hallucis longus tendon

due to lateral instability on account of an ATFL lesion. Therefore, the status of the lateral ligaments should be assessed arthroscopically and instability should be treated, if appropriate [11, 12].

1.2.1.2 Interosseous Ligament

This short ligament may be considered a continuation of the interosseous membrane at the distal part of the syndesmosis.

1.2.1.3 Posterior Tibiofibular Ligament (PTiFL)

This syndesmotic ligament is formed by two independent components: one superficial and one deep. The superficial one, which is usually referred to with the term PTiFL, arises from the posterior part of the lateral malleolus and inserts into the posterior tibial tubercle (Fig. 1.3). This component is the homologue of the anterior tibiofibular ligament and it is well evident in posterior ankle arthroscopy. The deep component is also known as the transverse ligament. It originates above the digital fossa of the lateral malleolus and inserts into the posterior border of the tibial articular surface [13]. The transverse ligament behaves as a true labrum and expands and deepens the articular tibial surface, providing joint stability and preventing posterior talar translation [8, 14].

1.2.1.4 Intermalleolar Ligament

The intermalleolar ligament runs obliquely from the lateral to the medial malleolus and slightly proximal. Medially it arises widely from the lateral edge of the medial malleolar sulcus, from the posterior distal edge of the tibia, from the flexor hallucis longus (FHL) sheath and from the posterior medial process of the talus until the joint capsule [15] (Fig. 1.3). In the anteroposterior direction, it is situated between the deep component of the posterior tibiofibular ligament (transverse ligament) and the posterior talofibular ligament. During ankle movements it tenses in dorsiflexion and relaxes in plantarflexion. This ligament can become the cause of disorders in both types of movement: a forced dorsiflexion trauma can cause injury or rupture of this liga-

ment; on the contrary, during plantarflexion it can be involved in soft tissue impingement caused by trapping between tibia and talus.

1.2.2 Ligaments That Join the Leg Bones to the Foot Skeleton

This group of ligaments that stabilize both tibiotalar and subtalar joints can be divided into lateral collateral complex and medial collateral complex (Fig. 1.4).

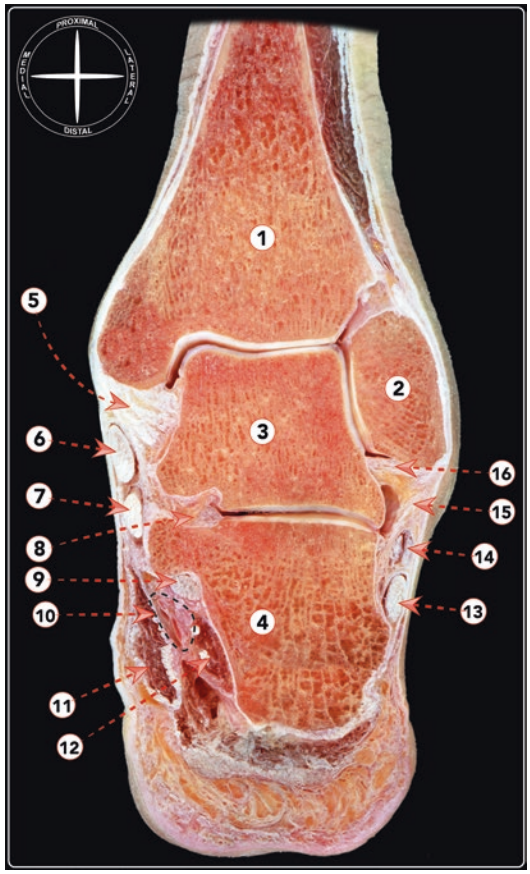

Fig. 1.4 Frontal section of the ankle and subtalar joint. (1) Tibia. (2) Fibular malleolus. (3) Talus. (4) Calcaneus. (5) Medial collateral ligament. (6) Tibialis posterior tendon. (7) Flexor digitorum longus tendon. (8) Interosseous talocalcaneal ligament. (9) Flexor hallucis longus tendon. (10) Tibial neurovascular bundle. (11) Abductor hallucis muscle. (12) Quadratus plantae muscle. (13) Peroneus longus tendon. (14) Peroneus brevis tendon. (15) Calcaneofibular ligament. (16) Posterior talofibular ligament

1.2.3 Lateral Collateral Ligament

The lateral collateral ligament (LCL) is the most commonly injured ligamentous structure of the lower limb, due to the inversion sprain [16]. It consists of the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL). When the foot is in neutral position, ATFL and CFL form a 105° angle on the sagittal plane and a 90–100° angle on the frontal plane [5, 8, 17]. These ligaments are not completely independent, since some fibers form an arch between CFL and the inferior part of ATFL [1]. Their tension changes during flexion and extension: in plantar flexion, the inferior bands of ATFL and CFL remain relaxed, while the upper ATFL band becomes taut [1]. In dorsiflexion, the upper band remains relaxed and the inferior bands of ATFL and CFL band become taut.

1.2.3.1 Anterior Talofibular Ligament (ATFL)

ATFL is the anterior component of the LCL, and it is the first ligament to be injured in an ankle inversion sprain. It controls the anterior tilt of the talus. It is a flat and quadrilateral ligament, in close contact with the joint capsule. Various anatomical variations of this ligament have been described, ranging from a single band to three bands (Fig. 1.2) [18]. However, recent studies have found that ATFL is a ligament always formed by two bands, and suggest that single-band ligaments are pathological ligaments where the superior band has been reabsorbed after injury [19]. This would be caused by the fact that the ATFL's superior fascicle is an intra-articular ligament [19, 20] (Fig. 1.5), which would impair its ability for healing, therefore explaining why percentages of chronic pain after an ankle sprain are so high after conservative treatment.

Between the two bands of the ATFL, there are vascular branches from the perforating peroneal artery and its anastomosis with the lateral malleolar artery [21]. The footprint on the lateral malleolus is about 10 mm proximal to the tip of the malleolus, just distal to the insertion of the inferior tibiofibular ligament, as the tip of the

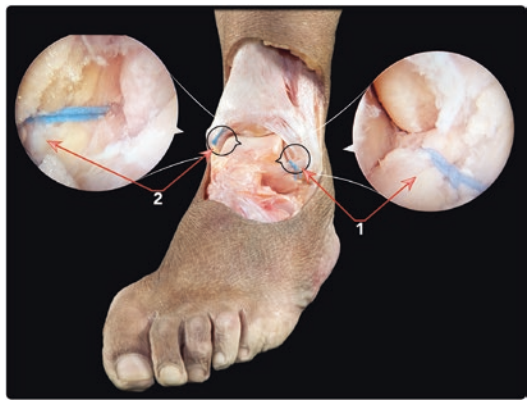

Fig. 1.5 Anterior view of a dissection performed after the arthroscopic procedure. Correlation of the arthroscopically sutured structures was obtained during dissection, showing intra-articular disposition of: (1) ATFL's superior fascicle. (2) Deltoid ligament (Anterior tibiotalar and tibionavicular ligaments). Figure reproduced with permission from copyright holder [20]

fibular malleolus is free from ligamentous insertions. Its direction varies according to the position of the foot. When the foot is in the neutral position, it is almost horizontal in relation to the ankle, whereas in plantar flexion its axis is parallel with the leg axis [22]. In this position the ligament is vulnerable to injury, particularly when the foot is inverted [23–25]. Injury will begin with the ATFL's superior fascicle and the inferior fascicle injury will follow. However, both fascicles behave differently: the superior fascicle is relaxed in dorsiflexion and tightens in plantar flexion, while the inferior fascicle is an isometric structure, always in tension, and is also connected with the Calcaneofibular ligament, forming the lateral fibulotalocalcaneal ligament complex [19].

ATFL lesions usually occur at its fibular insertion, which can be explained by its histologic characteristics: at the point where the ligament wraps around the lateral talar articular cartilage, a fibrocartilage exists, which seems to dissipate tension away from the talar enthesis, and consequently puts more tension on the fibular insertion of the ATFL [22].

The fact that the ATFL's superior fascicle is an intra-articular ligament that relaxes in dorsiflexion allows for its arthroscopic assessment [20], and indeed its exploration has to be included in the basic arthroscopic examination of the anterior ankle compartment [23].

1.2.3.2 Calcaneofibular Ligament (CFL)

The CFL is a cord-like ligament that runs from the anterior part of the tip of the malleolus toward the calcaneus (Fig. 1.2). It is the second most commonly injured ligament of the ankle. It is about 2 cm long and its diameter is about 6–8 mm [12]. It is covered by and in close contact with the fibular tendons and their sheaths. It crosses the ankle and subtalar joints, thereby stabilizing both of them. It is connected with the ATFL's inferior fascicle, forming the lateral fibulotalocalcaneal ligament complex of the ankle [19] (Fig. 1.6).

The lateral fibulotalocalcaneal complex becomes horizontal during plantarflexion and vertical in dorsiflexion, remaining taut throughout its entire arc of motion [1]. As per the CFL, it is the varus-valgus position of the ankle that considerably changes its tension: the ligament is relaxed in the valgus position and taut in the varus position.

An isolated lesion of CFL is rare. Its lesion together with the ATFL usually occurs in inversion sprains, approximately in 20% of traumas [24].

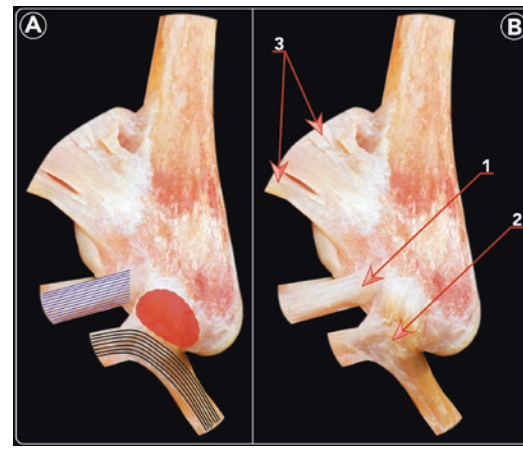

Fig. 1.6 Schematic view of the LFTCL Complex with the lateral malleolus disarticulated from the ankle. (**a**) View with the lateral ankle ligaments highlighted: ATFL superior fascicle (blue lines), LFTCL Complex (black lines) and area showing the common origin of the LFTCL Complex (red area). (**b**) Classic view of the LFTCL Complex. (1) ATFL superior fascicle. (2) LFTCL Complex. (3) Anterior tibiofibular ligament and distal fascicle. Figure reproduced with permission from copyright holder [19]

1.2.3.3 Posterior Talofibular Ligament (PTFL)

It is the posterior component of the LCL. The posterior talofibular ligament originates from the malleolar fossa in the medial posterior part of the lateral malleolus and, running horizontally, inserts along all the lateral posterior surface of the talus (Fig. 1.5). Because of its multifascicular morphology, it has a triangular shape with fibers inserting also into the talar tail or os trigonum, if present.

1.2.4 Medial Collateral Ligament (MCL)

The medial collateral or deltoid ligament is a multifascicular ligament that runs from the medial malleolus to the talus, calcaneus, and navicular bone. It crosses the tibiotalar and the subtalar joint, stabilizing both, restricting the valgus tilt, external rotation, and anterior translation of the talus. The most anterior part of the deltoid ligament is intra-articular, and therefore can be arthroscopically assessed (Fig. 1.5) [20, 23].

The division between fascicles of the MCL is confusing and somehow artificial, as the different fascicles are poorly defined [12]. Despite some confusion, all authors agree in that two layers are present, one superficial and one deep [8, 26, 27]. In the superficial layer a tibionavicular fascicle, a tibiocalcaneal fascicle (directed toward the sustentaculum tali) and a posterior superficial tibiotalar fascicle are present. In the deep layer, two fasciculi, a deep anterior and a posterior tibiotalar fasciculus, are found [8, 27–29] (Fig. 1.7).

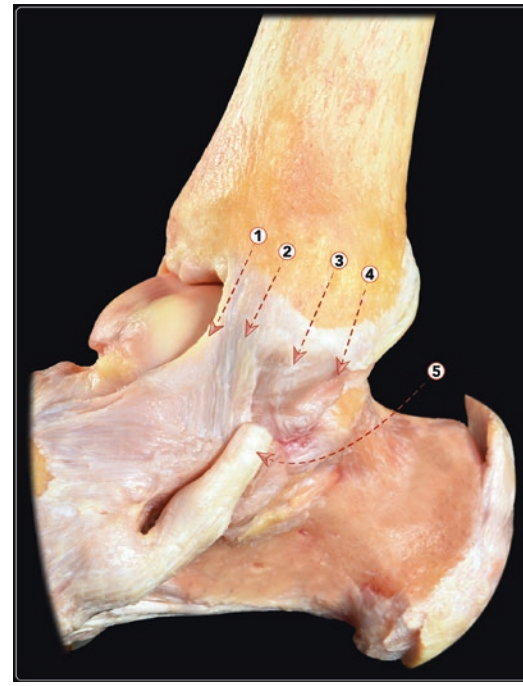

Fig. 1.7 Medial view of an osteo-articular dissection of the ankle. (1) Anterior tibionavicular fascicle. (2) Tibiocalcalneal fascicle. (3) Superficial posterior tibiotalar fascicle (4) Deep posterior tibiotalar ligament. (5) Posterior tibialis tendon

1.3 Hindfoot

Arthroscopically the hindfoot is a virtual triangular space in a sagittal section. It is limited plantarly by the upper facet of the calcaneus, posteriorly by the calcaneal tendon and retrocalcaneal fat pad (Kager's fat pad), and anteriorly by the posterior edge of the tibia, by the tail of the talus or os trigonum, if present. The latter, together with the ligaments we have described above, is surrounded by periarticular adipose tissue. To access the joint, this tissue should be carefully removed using FHL as the medial limit, not to be crossed on account of the presence of the tibial neurovascular bundle medial to it. The FHL tendon, the lateral talar process, the ankle joint capsule with the posterior ligaments of the joint, and the subtalar joint capsule can be identified arthroscopically. The joint and periarticular fat are separated by the retrocalcaneal fat pad from the fibulotalocalcaneal ligament (originally called Rouviere-Canela ligament). This is a thickening of the deep crural fascia, the fascia that separates the superficial posterior compartment from the deep posterior compartment. During posterior ankle arthroscopy, fascial transverse fibers of the Rouviere-Canela ligament can be easily recognized while removing periarticular fatty tissue. To access the joint, a working window has to be opened through the Rouviere-Canela ligament.

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2 Gross Anatomy of the Subtalar Joint

Matteo Guelfi, Miquel Dalmau-Pastor, Rosa M. Mirapeix, and Jordi Vega

2.1 Introduction

The subtalar joint is the main joint of the peritalar complex and is synonymous with the talocalcaneal joint, where the plantar part of the talus articulates with the dorsal part of the calcaneus. The subtalar joint can be divided into a posterior joint and an anterior complex, the latter composed of the anterior talocalcaneal joint (anterior subtalar joint), middle talocalcaneal joint, and talonavicular joint (Fig. 2.1).

Anterior and posterior articular surfaces of the talus and calcaneus are divided by a sulcus. When the two bones are articulated, this creates a structure known as the sinus and canal tarsi. From a lateral view of the subtalar joint, the sinus tarsi is observed, a cone-shaped structure that contains fatty tissue, blood vessels, and the talocalcaneal interosseous ligament. The sinus tarsi, wide opened at its lateral part, narrows medially, forming the tarsal canal, which has a small medial exit [1] (Fig. 2.1).

A single or a series of traumatic events can lead to a sinus tarsi syndrome (STS). These injuries cause instability of the subtalar joint resulting in chronic anterolateral pain or discomfort. Several theories have been proposed to explain the etiology of STS like a interosseous ligament tear, synovitis, and nociception and proprioception disorders [2, 3].

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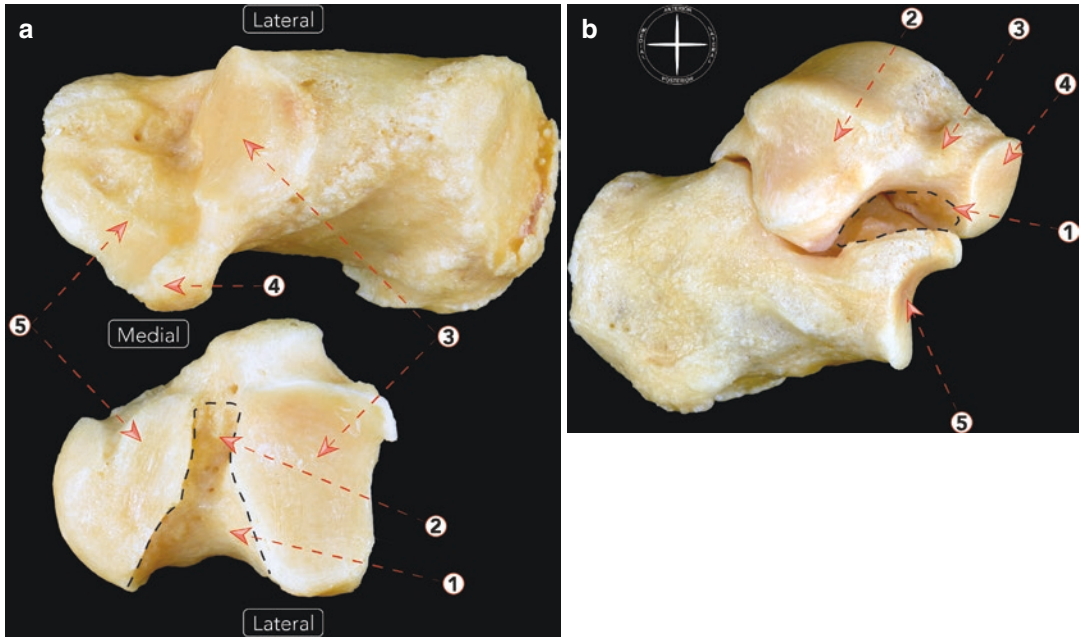

Fig. 2.1 (**a**) Dorsal view of the calcaneus and plantar view of the talus, demonstrating the articular surfaces of the subtalar joint. (1) Sinus tarsi. (2) Tarsal canal. (3) Posterior subtalar articular surfaces. (4) Sustentaculum tali. (5) Anterior and middle subtalar articular surfaces. (**b**) Lateral view of the talus and calcaneus forming the subtalar joint. (1) Sinus tarsi. (2) Lateral talar articular surface. (3) Talar neck. (4) Talar head. (5) Anterior articular surface of the calcaneus (for the cuboid bone)

The posterior subtalar joint is a synovium-lined articulation formed by the posterior concave calcaneal facet of the talus and the posterior convex talar facet of the calcaneus [4].

The anterior subtalar joint can't be analyzed alone but should be considered as part of the talocalcaneonavicular joint, also known as the *coxa pedis* [5, 6]. The anatomical shape of this structure has several similarities with the coxofemoral joint, imitating a ball and socket joint. The posterior articular surface of the navicular bone, the anterior and middle calcaneal articular surfaces, and the spring ligament complex form a concave surface where the head of the talus fits.

The movements of the subtalar joint are almost exclusively of pronation and supination. The latter corresponds to two-thirds and pronation to one-third. Normal range of motion of the subtalar joint is around 25–30° of supination and 5–10° of pronation [7].

Arthroscopically, only the posterior subtalar joint can be well visualized and assessed trough lateral subtalar portals or endoscopic posterior ankle portals (Fig. 2.2). Although anterior subtalar arthroscopy has been described [8], the talocalcaneonavicular joint is mostly considered inaccessible for arthroscopic examination because of its anatomic configuration [4].

2.2 Subtalar Ankle Ligaments

Numerous ligaments stabilize the subtalar joint. Some of them are intrinsic ligaments, and others cross also the ankle joint, stabilizing the joint (e.g., calcaneofibular, deltoid, and spring ligament). In addition, the inferior extensor retinaculum (IER) acts as an ulterior subtalar joint stabilizer. In this section, the CFL and deltoid and posterior ligaments will not be discussed, as they have been already treated in the previous chapter about ankle joint anatomy.

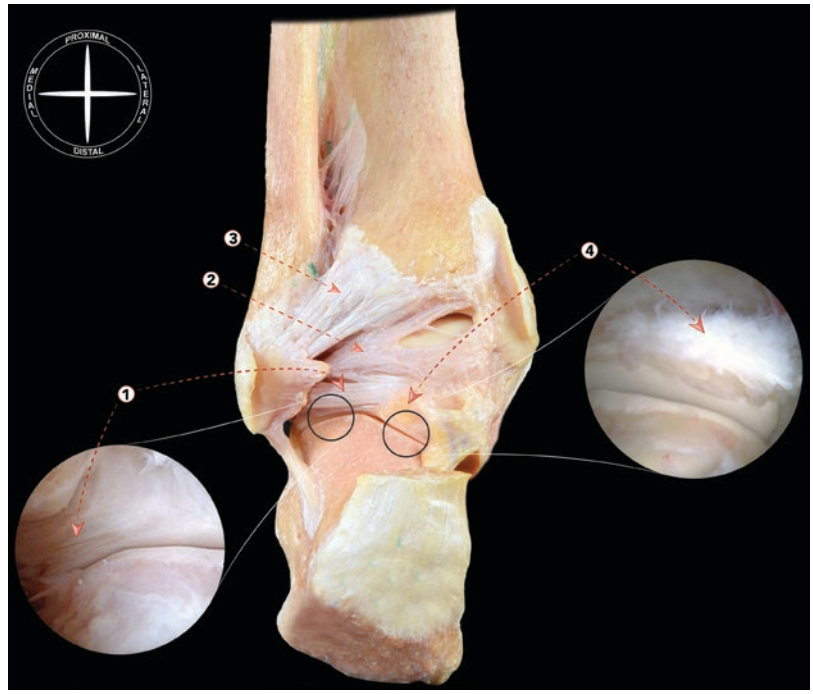

Fig. 2.2 Posterior view of an osteoarticular dissection of the ankle and subtalar joint and correlation with arthroscopic view from subtalar arthroscopy. (1) Posterior talofibular ligament. (2) Intermalleolar ligament. (3) Posterior tibiofibular ligament. (4) Talar posterolateral tubercle

2.2.1 Interosseous Talocalcaneal Ligament (ITCL)

The interosseous talocalcaneal ligament (ITCL) is probably the most important subtalar joint stabilizer. It is a flat, thick, band-like ligament occupying about half of the medial part of the tarsal canal. The ITCL extends from upper medial to lower lateral running obliquely from the talus toward the calcaneus [4, 9, 10] (Fig. 2.3).

The ITCL stabilizes the subtalar joint by maintaining apposition of the talus and calcaneus [11]. Being a strong and thick ligament, it is rarely injured alone; however, a sudden deceleration of the talus compared to the calcaneus with the foot in maximal plantarflexion may cause an injury of the ITCL [12, 13].

Clinical and experimental studies have demonstrated that a rupture of the ITCL results in an increased range of subtalar rotation especially in inversion and in severe instability of the ankle-subtalar joint complex especially during the stance phase of walking [11, 13–15]. In addition, an ITCL tear has been reported as a possible cause of sinus tarsi syndrome [2].

2.2.2 Cervical Ligament

The cervical ligament (CL) originates from the superolateral calcaneal surface just in front of the sinus tarsi and running medially inserts on an inferolateral tubercle on the talus neck. It has an orientation of approximately 45° on the horizontal plane and is formed by multiple bands (Fig. 2.4). These extend in different directions to indicate an adaptation to different functional needs of the talus during movements [10].

As the ITCL, the cervical ligament acts as a subtalar intrinsic ligament and provides a strong connection between calcaneal and talar joints. Following single or multiple traumatic events, an injury of the ITCL and cervical ligament can be responsible for a sinus tarsi syndrome and a subtalar joint instability, resulting in excessive supination and pronation movements [11, 15, 16].

2.2.3 Spring Ligament Complex

The spring ligament complex is a thick triangular structure stabilizing and expanding medially the coxa pedis. It is composed of the superomedial and inferior calcaneonavicular ligaments and the calcaneonavicular component of the bifurcate ligament (explained below).

The inferior calcaneonavicular originates from the coronoid fossa of the calcaneus and inserts on both the navicular beak and tuberosity [17]. It is important for maintenance of the longitudinal arch, and its medial margin is continuous with the superomedial calcaneonavicular band.

The superomedial calcaneonavicular ligament shares its origin with the tibiocalcaneal part of the superficial deltoid ligament and joins laterally with fibers of the tibionavicular component of the deltoid ligament [18]. This ligament was initially described as a component of the tibiocalcaneonavicular ligament [19].

The spring ligament (in particular superomedial bundle) is subjected to stress at the level of the talonavicular joint and as a result is covered by fibrocartilage. This provides a smooth transitional surface for the talar head articulation [20] (Fig. 2.3).

Chronic posterior tibial tendon dysfunction (PTTD) may cause a spring and deltoid ligament stretch and lose competence over time. This condition leads to valgus heel and acquired flatfoot deformity.

In addition, the spring ligament can be involved in medial ankle instability. According to Hinterman, in type III medial ankle instability, a tear of the deltoid ligament is associated with an injury of the spring ligament [21].

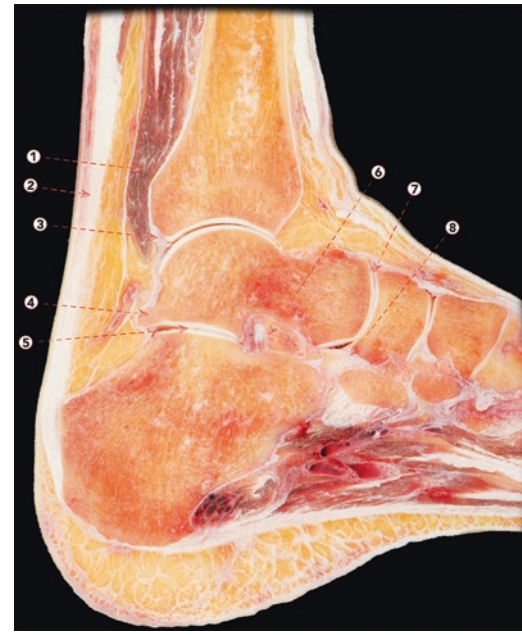

Fig. 2.3 Sagittal section of a foot. (1) Flexor hallucis longus muscle belly. (2) Calcaneal tendon. (3) Deep component of the posterior tibiofibular ligament (posterior ankle labrum). (4) Talar posterolateral tubercle. (5) Subtalar joint. (6) Talocalcaneal interosseous ligament. (7) Talonavicular joint. (8) Spring ligament complex

2.2.4 Bifurcate Ligament

The bifurcate ligament has a "Y" or "V" shape spanning from the calcaneus to navicular bone and cuboid bone. It is formed by two different components: a calcaneonavicular and a calcaneocuboid one [18, 22]. The medial component of the bifurcate ligament is the calcaneonavicular band that arises from the anteromedial angle of the sinus tarsi, just anterior to the anterior talar articular surface. Running anteriorly, dorsally, and medially, it is inserted at the superior or dorsal segment of the navicular bone and provides the lateral limit of the coxa pedis [18].

The calcaneocuboid ligament originates from the anterior aspect of the intermediary tubercle of the calcaneus (anterior part of the sinus tarsi), lateral to the origin of the calcaneonavicular band. It runs anteriorly and inferiorly attaching into the dorsal aspect of the cuboid bone [18].

The angle formed by the two bands of bifurcate ligaments is approximately 20° in the sagittal plane and 30° in the transverse plane [23].

2.2.5 Inferior Extensor Retinaculum

The inferior extensor retinaculum (IER) is an aponeurotic structure that is continuous to the crural fascia. This structure prevents bowstringing or subluxation of the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius tendons [24]. It has a Y or X shape

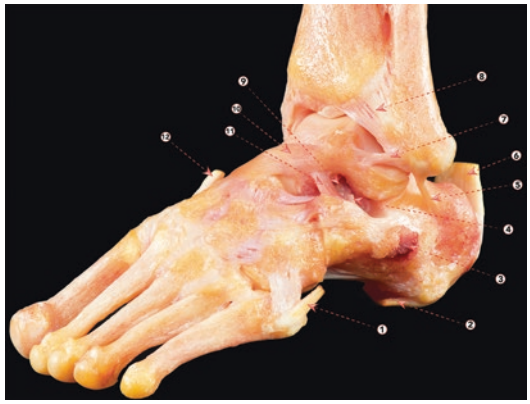

Fig. 2.4 Anterolateral view of an osteoarticular dissection of the ankle and subtalar joint. (1) Peroneus brevis tendon. (2) Plantar aponeurosis. (3) Peroneal trochlea. (4) Lateral root of the inferior extensor retinaculum. (5) Calcaneofibular ligament. (6) Calcaneal tendon. (7) Anterior talofibular ligament. (8) Anterior tibiofibular ligament. (9) Talocalcaneal interosseous ligament. (10) Dorsal talonavicular ligament. (11) Cervical ligament. (12) Tibialis anterior tendon

and plays an important role in subtalar stabilization [25].

The stem part of the Y shape has three roots that arise from the canal and sinus tarsi: a lateral, an intermediate, and a medial one [10]. It runs medially and divides into the oblique superomedial band, which inserts on the anterior aspect of the tibial malleolus, and into the oblique inferomedial band, which splits to insert on the abductor hallucis muscle and on the navicular and medial cuneiform. In approximately 25% of the cases, an additional oblique superolateral band is found [26]. This band, which varies considerably in size and gives an X-shaped morphology to the IER, crosses the ATFL and inserts on the lateral surface of the lateral malleolus. It seems that in the Brostrom-Gould procedure, this is the band used to augment the ATFL repair [25].

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Part II

Basic Ankle Arthroscopy

3 Instrumentation and Operative Setup for Anterior and Posterior Ankle Arthroscopy

Cecilia Pasquali, Clara Terzaghi, and Elia Paiusco

The instrumentation for ankle arthroscopy originated from knee arthroscopy; however, since 1990, a new system of instrumentation dedicated only to the smallest joints such as the ankle and the most recent procedures have recently been introduced into surgical practice.

3.1 Irrigation

Irrigation to maintain sufficient intra-articular pressure can be obtained using sacks of saline or Ringer's solution using a gravity drip at a height of approximately 2 mt, positioned at the patient's head; the irrigation occurs due to gravity or, in rare cases, by using an arthroscopic infusion pump in this case the pressure must be set between 40 and 50 mm.

The inflow and the outflow are facilitated by the use of an arthroscopic cannula system.

Other cannula accessories guarantee the maintenance of the portals in cases of inversion of the instrumentation, above all in posterior arthroscopy.

3.2 Arthroscope

The 4 mm arthroscope can also be used for ankle arthroscopy; the preferable optic for the ankle is 30°, but a 70° optic can also be used. The 6 mm-diameter cannula offers the advantage of better irrigation (Fig. 3.1).

There are also varied arthroscopes dedicated to the small articulations of 2.2–2.9 mm which are shorter and have an image quality comparable to that of traditional arthroscopes (Fig. 3.2).

3.3 Light Source

The transmission of light to the arthroscope, thanks to a fibre-optic cable, is obtained from the same light source used in knee arthroscopy.

3.4 Instruments

Basic instruments: Spinal needle, 18 G, 3.5 IN; scalpel handle n. 3 and blade n. 11; mosquito forceps; Kocher forceps.

Special instruments: Straight and curved probe, 3 mm; baskets and straight grasping for-

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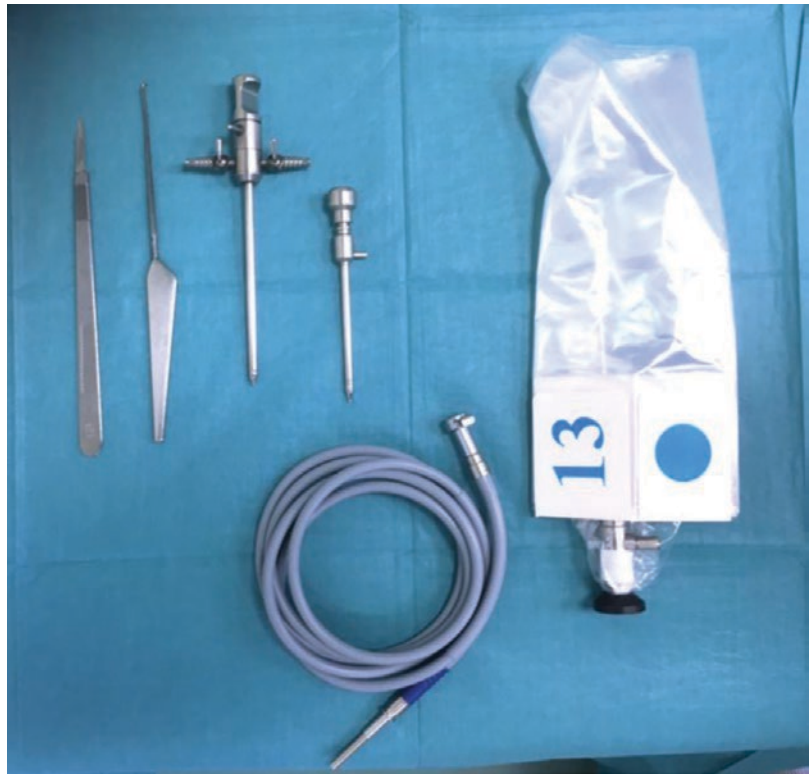

Fig. 3.1 Arthroscope and optic cable

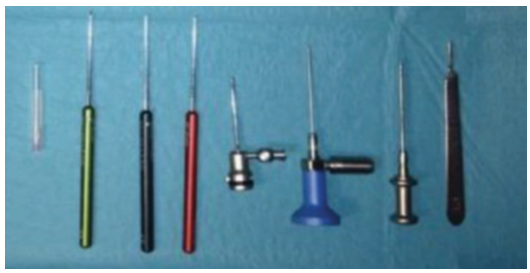

Fig. 3.2 Small joint arthroscopes

ceps; 4 mm straight osteotome, curved elevator; closed curette 15° and 90°; open 4 mm and 6 mm curette; 65° chondro pick, straight osteotome and curved elevator, hammer, and pick assist; 2.7 mm and 4.5 mm arthroscopic forceps.

The Ferkel compass or the Micro Vector drill guide system permits transmalleolar or transtalar access**.**

The cauterization is carried out using a laser scalpel for procedures on soft parts.

The shaver for large joints can also serve the ankle, and the blades which are most used are the following:

- Full radius, 4.5.
- Full radius, 3.5.
- Incisor, 3.5.
- Razor-cut, 4.5.
- Cutter, 3.5.
- Cutter, 4.5.
- Full-radius curve, 4.5.
- Abrader, 4.0.
- Notch-Blaster Abrader, 4.0.
- Acromionizer, 4.0.

When it is used as a small joint shaver, the blades which are mostly used are the following (Fig. 3.3, 3.4, 3.5, 3.6, 3.7, 3.8, and 3.9):

- Cutter, 2.9.
- Barrel Abrader, 2.9.
- Abrader, 2.9.
- Full radius, 2.9.
- Razor-cut, 2.9.

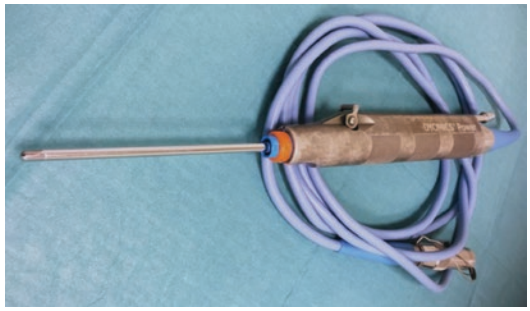

Fig. 3.3 Shaver

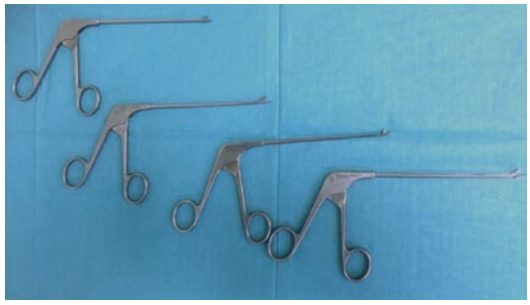

Fig. 3.4 Baskets

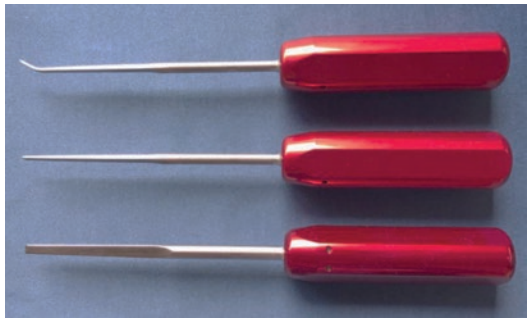

Fig. 3.5 Osteotomes

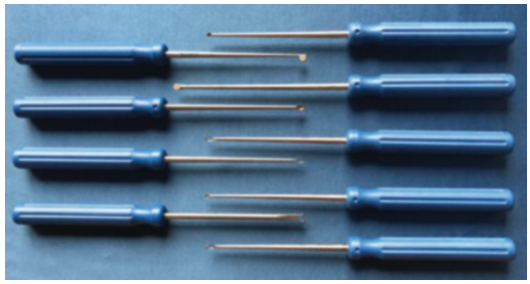

Fig. 3.6 Curettes

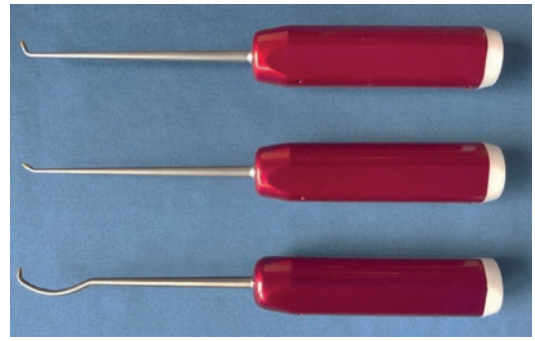

Fig. 3.7 Chondro pick, straight and curved

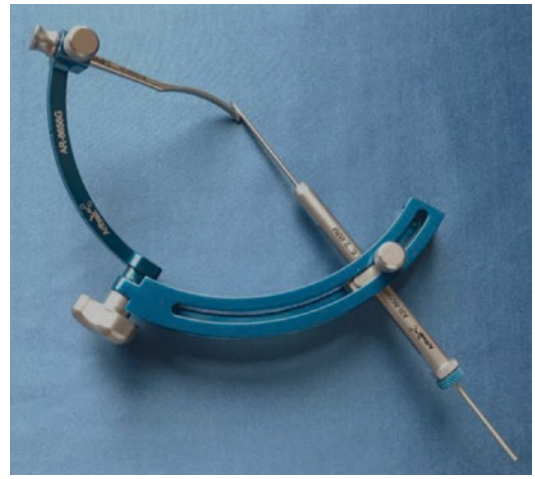

Fig. 3.8 Ferkel guide

Fig. 3.9 Small joint shaver blades

3.5 Distraction Instruments

Distraction instruments are not usually necessary for the ankle; however, in certain cases, these instruments can enlarge the articular space between the tibia and the ankle bone creating better arthroscopic vision and creating an ease of access and use of the necessary instruments.

3.5.1 Non-invasive Distraction

Manual distraction with a distractor foot strap or by gravity (Fig. 3.10).

In this case, the leg is positioned on a support, and the knee is bent.

3.5.2 Invasive Distraction

Traction by a trans-heel spur cord.

External fixtures with monolateral fishes (tibial and heel), type Guhl.

These mentioned systems are recommended in cases of serrated ankles or osteoarthritis. They can, however, cause excessive articular stress and do not permit easy changes to the position of the foot in respect to the leg during the intervention.

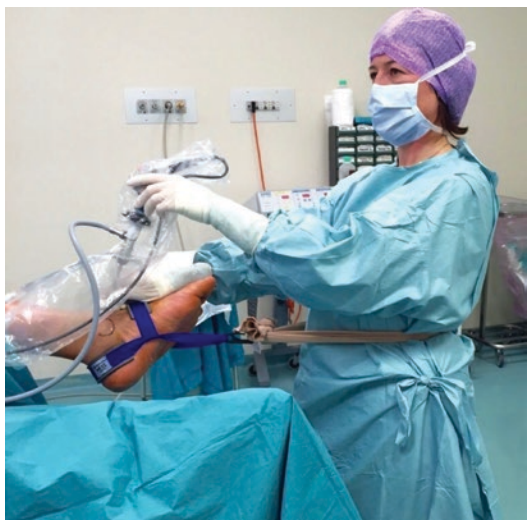

Fig. 3.10 Non-invasive distraction

3.6 Anterior Arthroscopy Using the Dorsiflexion Method

The patient is placed under a general anaesthesia/epidural. The tourniquet is placed on the thigh; or, in some cases, for loco-regional anaesthetic reasons, the tourniquet is placed on the leg.

The position is supine with the foot to the inner extremity of the operating bed; the surgeon can completely dorsiflex the ankle by leaning against the sole of the patient's foot. Using this method, traction is not necessary because the anterior working area becomes enlarged [1, 2].

3.6.1 Portals

A very important instrument is the skin marker (Fig. 3.11a, b).

The principal portals are the anteromedial and the anterolateral; a posterior lateral accessory portal can be used for the inflow, but it is not necessary if one uses a 4.6 mm arthroscopic cannula.

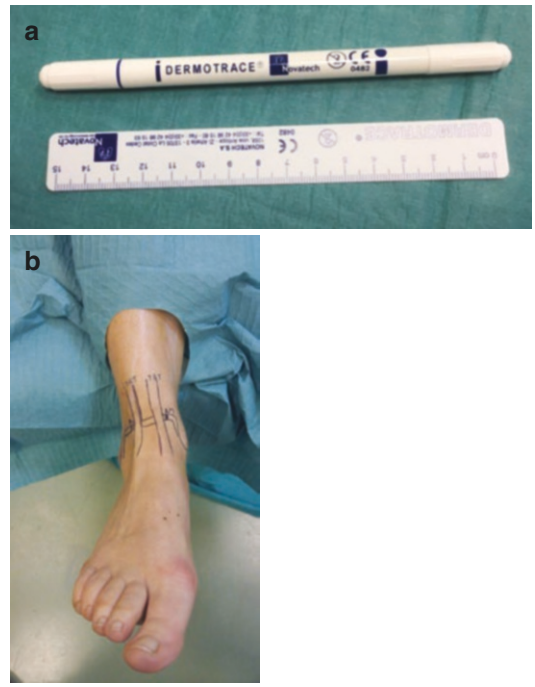

Fig. 3.11 (**a**) Skin marker. (**b**) Anteromedial and anterolateral portals

The ankle has to be plantarflexed and inverted to highlight and outline with the skin marker the superficial peroneal nerve.

The anteromedial portal is the first to be carried out because it is easy to perform without risk to the neurovascular structures.

A spinal 18-G needle and 15 cc of physiological solution are inserted through this portal to dilate the joint.

The anteromedial portal is performed vertically incising the skin with a scalpel blade n. 11 at a "soft spot" just medial to the anterior tibial tendon, on the joint line, maintaining the ankle in a dorsiflexion position; in this way, the portal is proven to be more central.

Dissection under the skin is done with a curved mosquito forceps, and the arthroscope is always introduced with a dorsiflexed ankle.

Once irrigated and inspected the joint, the arthroscopy continues with the execution of the anterolateral portal under direct control, and translucency can help to avoid lesions to the small lateral branch of the superficial peroneal nerve which passes under the skin superficially to the extensor digitorum longus tendon.

The anterolateral portal is lateral in respect to the peroneus tertius tendon and the extensor digitorum longus tendon, slightly proximal in relation to the joint line. Incising the skin vertically with a scalpel blade n. 11 and dissecting under the skin with a curved mosquito forceps we can introduce the instruments or the trocar.

The accessory portals are the anterocentral, anteromedial, anterolateral, posterolateral portals and the transmalleolar and transtalar portals.

The anterocentral accessory portal is above the extensor digitorum longus tendon. This accesory portal may cause lesions to the dorsal pedis artery, the deep branches of the peroneal nerve and the medial branches of the superficial peroneal nerve. Thus, it should be avoided.

The anteromedial accessory portal is positioned from 0.5 to 1 cm below the tip of the medial malleolus, 1 cm anterior in respect to the margin of it; the incision in the skin must follow the fibres of the deltoid ligament.

The anterolateral accessory portal is carried out 1 cm anterior to the margin of the lateral malleolus; the incision in the skin must follow the fibres of the anterior talofibular ligament.

The posterolateral portal will rest laterally to the Achilles tendon with the ankle in plantarflexion, and the incision in the skin is made 1.2 cm from the tip of the lateral malleolus.

The potential risks of this access relate to the small saphenous vein which is found at an average distance of 9.5 mm (2–18 mm) and to the sural nerve which is found at an average distance of 6 mm (0–12 mm). The posterolateral access, according to Golanò et al. [3], lies inside a triangular area delineated by the tibia, the Rouvière ligament and the posterior intermalleolar ligament.

The transmalleolar and transtalar portals are commonly used to treat osteochondral talar lesions; execution can be facilitated by drilling with a Kirschner wire under arthroscopic control; articular distraction is useful in this case [4].

3.7 Posterior Arthroscopy

This technique, for the anatomical characteristics of the area, has been referred to in the past as "dangerous" and "insidious."

An important development in this procedure is the description of the two arthroscopic posterior portals by Van Dijk et al. in 2000 [5–7].

The patient is in a prone position with the foot over the edge of the operating table, and we suggest non-invasive distraction.

3.7.1 Portals

The two portals to access the posterior area of the ankle and the subtalar joint are the posterolateral and the posteromedial portals.

The first is really lateral in respect to the Achilles tendon; on the horizontal plane, it is nearly 1 cm proximal to the tip of the lateral malleolus with the ankle in a plantigrade position (Fig. 3.12).

After the incision into the skin, dissect the underlayers with a curved Klemmer forceps, in the direction of the first interdigital space of the forefoot.

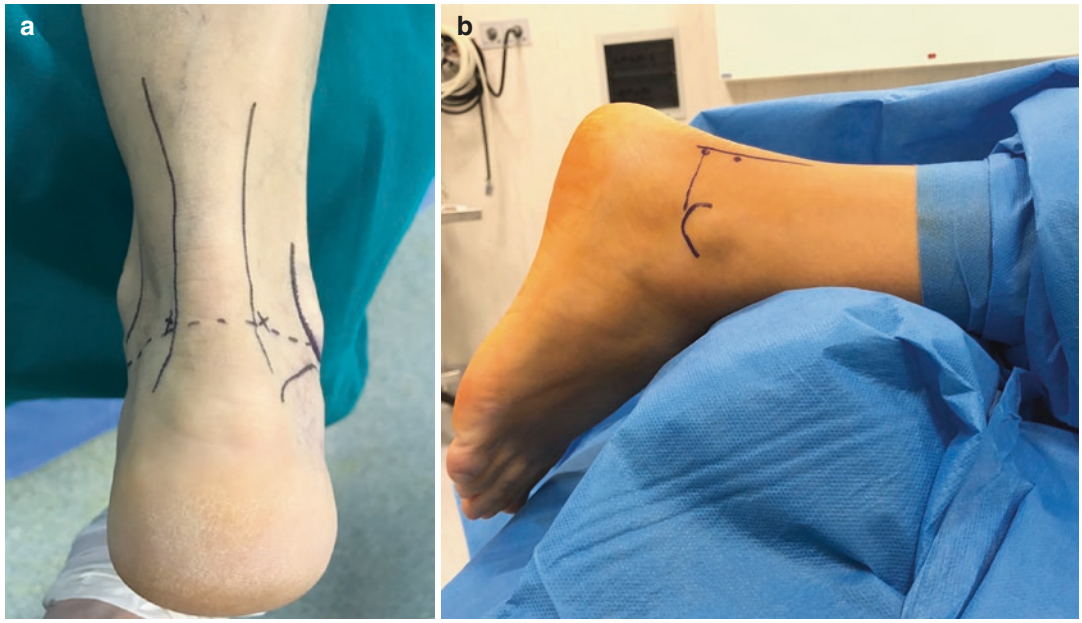

Fig. 3.12 Posterolateral and posteromedial portals

Then we replace the Klemmer with the trocar: it is possible to palpate the posterior process and enter laterally and in proximity to this.

Posteromedial access is performed medially to the Achilles tendon to the same level of the posterolateral portal (Fig. 3.13).

A Klemmer forceps is introduced from the posteromedial portal at 90° to the trocar, the direction of the vision of the optic mast be lateral and the Klemmer forceps is introduced using the trocar as a guide until this doesn't reach the tip of the optic.

Backing the optic lightly away brings the point of the Klemmer forceps into sight.

The introduction of the instruments across the posteromedial portal must follow the same technique using the optic as a guide and leaving it in the direction of the first interdigital space (Fig. 3.14).

The flexor hallux tendon must be used as a point of reference which one must not pass.

For the posterior access to the to the subtalar joint, the 4.0 mm arthroscope is suitable; the 2.7 mm arthroscope is not necessary. A workspace is created adjacent to the joint removing part of the joint capsule and the tissue.

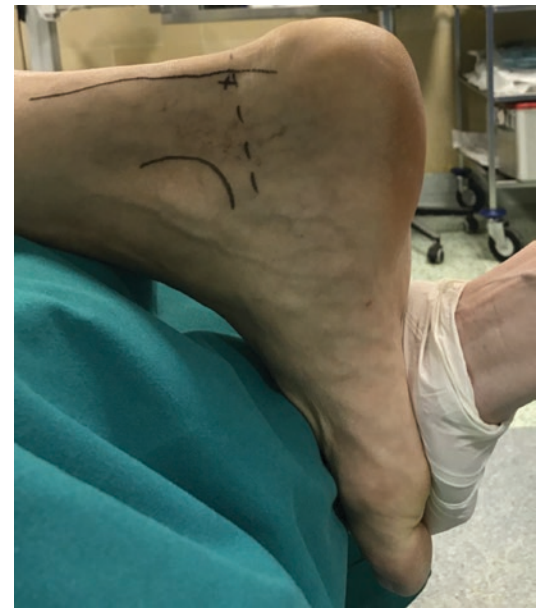

Fig. 3.13 Posteromedial portal

Further portal accessories can be used for extra-articular procedures.

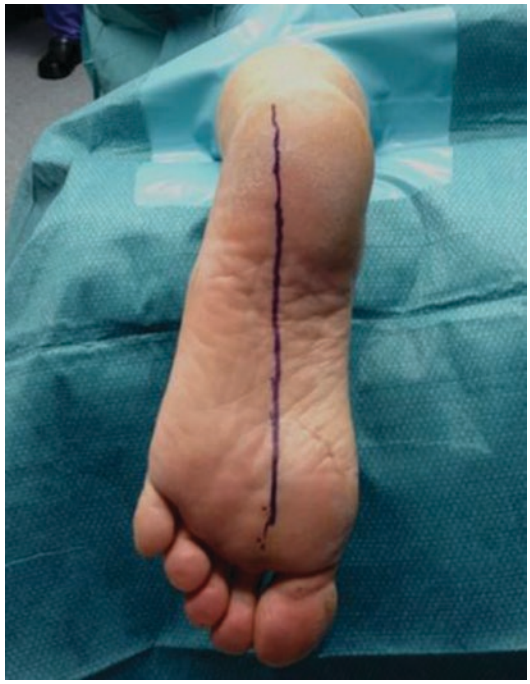

Fig. 3.14 Instrument's direction

Traditional portals (posteromedial and posterolateral) should be used for all surgeries which require subtalar joint investigation.

For calcaneoplasty, the two portals are performed laterally and medially to the Achilles tendon, immediately next to the upper part of the calcaneus.

Peroneal tendon tendoscopy necessitates a 2.7 mm arthroscope for small articulations; the patient is in lateral decubitus, and the arthroscopic access can be along the whole length of the tendons.

Posterior tibial tendon tendoscopy is conducted with the patient supine, and the portals are located over the tendon, 2 cm distal and 2 cm proximal to the tip of the medial malleolus.

The Achilles tendon tendoscopy is performed with the patient in prone position, and the two portals are distal on the lateral border of the Achilles tendon and proximal on the medial border of the Achilles tendon.

3.8 Lateral Approach to the Subtalar Joint

This approach is used to treat some pathologies such as subtalar arthrodesis and for the removal of adipose tissue in sinus tarsi syndrome.

In this procedure, three portals are expected [8]: the middle portal, the anterolateral portal, and the posterolateral portal. The patient is in lateral decubitus with the knee positioned 90° leaving the extremity of the limb free to be operated on (Fig. 3.15).

The middle portal is approximately 1 cm anterior to the tip of the fibula over the sinus tarsi.

The anterolateral portal is located 1 cm distally and 2 cm anteriorly to the tip of the peroneal malleolus; the cutaneous branches of the sural nerve run alongside at 8 mm inferior and 17 mm anterior to the portal, respectively.

The posterolateral portal is approximately 2 cm posterior and 1 cm proximal to the tip of the fibula, close to the Achilles tendon.

The arthroscope is usually introduced through the anterolateral portal and the instruments through the posterolateral, allowing the posterior articular facet of the talar bone to come into sight; to see the most lateral portion of this, it is necessary to invert the portals.

The middle portal in association with the anterolateral portal allows vision of the anterior and lateral portions of the articular posterior facet and of the sinus tarsi.

Pitfalls	Tricks
Make a treatment plan preoperatively	Mark all anatomical landmarks before starting the arthroscopy
Find the working space in the anterior arthroscopy with the undistracted dorsiflexed position of the ankle; the relaxed structures are less susceptible to injury	Make visible the superficial peroneal nerve by bringing the foot in forced plantarflexion and inversion
In the posterior arthroscopy make the posterolateral portal looking at the foot sole, directing the mosquito forceps between the first and the second toe. Then make the posteromedial portal and introduce the mosquito forceps in a 90° angle compared to the scope	Look for the posterior talar process removing the ligament of Rouvière; then identify the flexor hallucis longus tendon and stay lateral of this

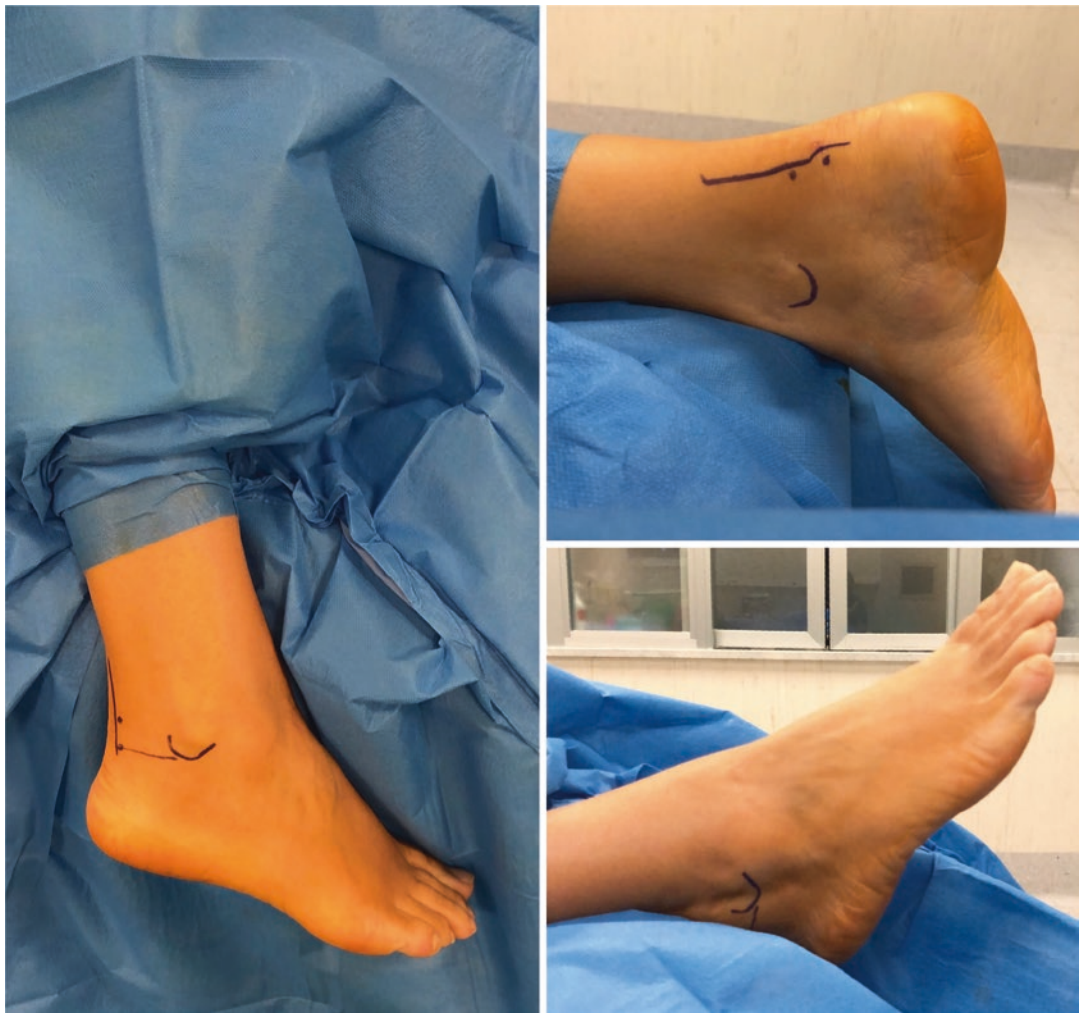

Fig. 3.15 Supine, lateral, and prone positioning

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4 Diagnostic Arthroscopy for the Anterior/Posterior Ankle Joint

Clara Terzaghi, Cecilia Pasquali, and Elia Paiusco

Ankle arthroscopic surgery is a minimally invasive procedure which became more and more common in the last few years, in order to reduce big scars on the skin and to minimize soft tissue insults. It allows a good direct visualization on the joint without making arthrotomy by malleolar osteotomy.

To achieve a good examination and a successful result in ankle arthroscopy, the first step to follow is to standardize the method: the approach has to be reliable, repeatable, and reproducible.

For surgeons who approach this kind of surgery firstly, we recommend to adopt a method to visualize the ankle joint standardizing each.

Considering the coronal plane of the ankle surface, we can recognize three different longitudinal areas of the joint, the medial side, the central part, and the lateral side, both anteriorly and posteriorly.

We call the anterior cavity A and the posterior cavity P and the lateral side 1, the central area 2, and the medial side 3.

This methodological inspection picks out six different compartments, which allows a reproducible approach to identify and classify the intra-articular pathologies (Fig. 4.1).

The lateral area (1) starts from the lateral malleolus and ends up at the sagittal groove of the talus laterally, the central area (2) starts from the shoulder of the talus until the medial talus border, and the medial area (3) starts from the sagittal groove of the talus medially to the medial malleolus.

The surgeon is positioned at the bottom of the table, looking at the front of the ankle. The assistant will move the ankle in dorsiflexion.

Visualization is usually supported by two standard portals: AM and AL [1].

The anteromedial portal has to be done first, while laterally the translucency in the anterolateral compartment permits to approximately identify the intermediate branch of the superficial peroneal nerve (Fig. 4.2).

Using the AM portal, the scope has to be put into the tibiotarsal joint.

The central part of the tibia has to be examined first (2A), sweeping the scope in and out and rotating to achieve better visualization. Advancing the scope through the medial portal, the talus surface is identified at the bottom. The talus shape is identified: the central surface of the talus shows a small depression articulated with the horizontal configuration of the tibia above (Fig. 4.3).

Electronic Supplementary Material The online version of this chapter (https://doi.org/10.1007/978-3-030-29231-7_4) contains supplementary material, which is available to authorized users.

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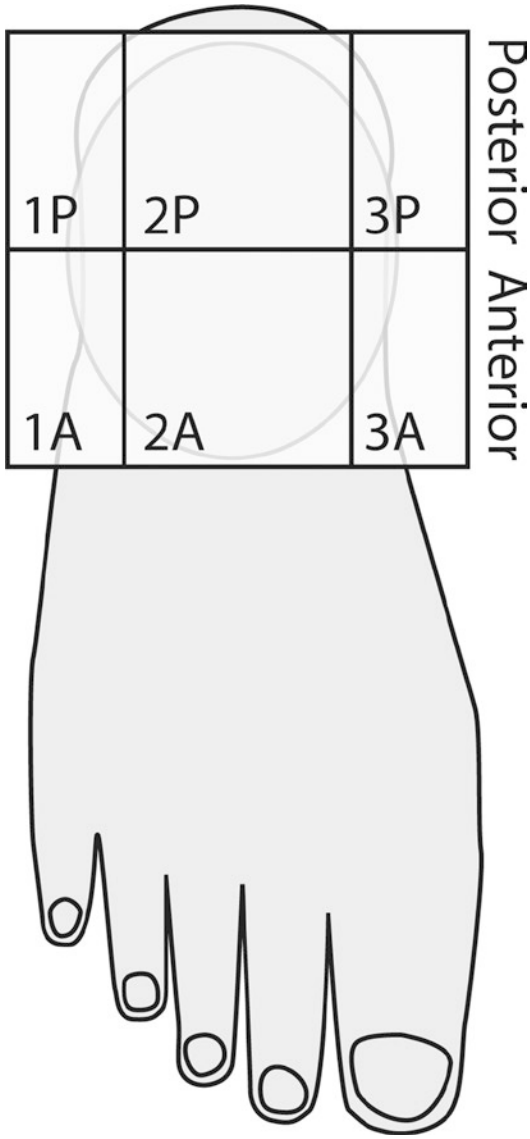


Fig. 4.1 The simple and schematic ankle joint visualization, identifying six ankle compartments to achieve a standard diagnostic arthroscopy. Illustration by Tomaso Baj

Medially and laterally, the talus becomes more convex on the coronal plane where the talus shoulders match with the medial and lateral notch of the tibia surface. The talar medial side is frequently involved in osteochondral lesions that can be treated easily from the AM and AL accesses.

The space in the central area allows maneuvering in this region the scope, whose recoiling permits a better view of the whole surface of the talar dome. Rotating the light cable at 90°, right and left, allows a direct and complete visualization of the ankle joint forefront. The anterior-inferior gutter represents the reflection of the capsular joint, which inserts directly on the talar neck, where sometimes osteophytes can be present. During the dorsiflexion movement of the ankle, the osteophytes at this level might conflict against the anterior tibial osteophytes, bearing the anterior impingement.

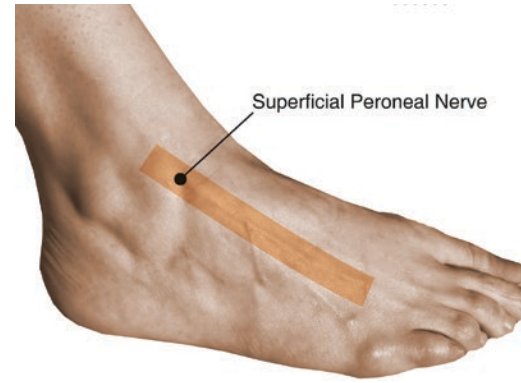


Fig. 4.2 The superficial peroneal nerve clearly appears on the skin surface putting slightly the foot in plantarflexion and supination. Using the light cable through the AM portal, visualization can be facilitated. Illustration by Tomaso Baj

Rotating the light cable at 90°, the lateral side of the joint is exposed. The anterolateral structures are identified, moving the scope laterally (1A). The syndesmotic area, "meeting point" of the tibial plafond, the lateral dome, and the fibula, called trifurcation, is identified. The distal epiphysis of the fibula is bounded by the tibiofibular ligament with the interposition of synovial tissue (Fig. 4.4).

In patients affected by hyperlaxity, the view of the fibula is easily manageable (Fig. 4.5).

The anterior tibiofibular ligament (ATFL) runs approximately at 45° angle anteriorly from the tip of the fibula to the inferior lateral portion of the talus and merges with the anterior capsule of the joint.

Identification of the ATFL can be not easy sometimes because intracapsular. Therefore, when ankle sprain occurs frequently and the ATFL is torn, easily is recognized the hole into the capsule. The anatomical studies of

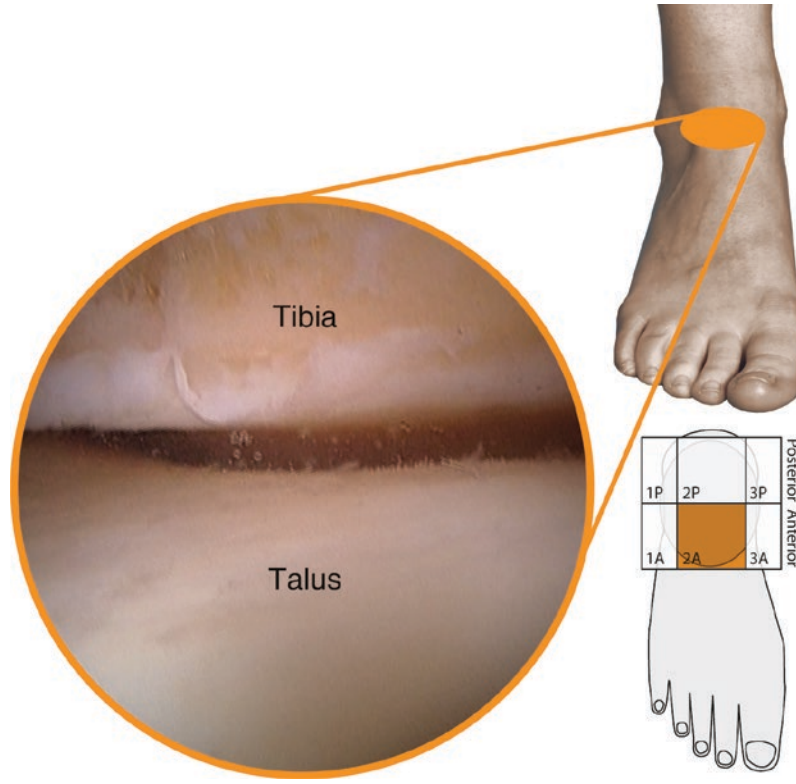


Fig. 4.3 The central part of the ankle joint, simply called "2A area." At this level, usually osteophytes develop on the anterior margin of the tibial lip, reducing the visualization. Illustration by Tomaso Baj

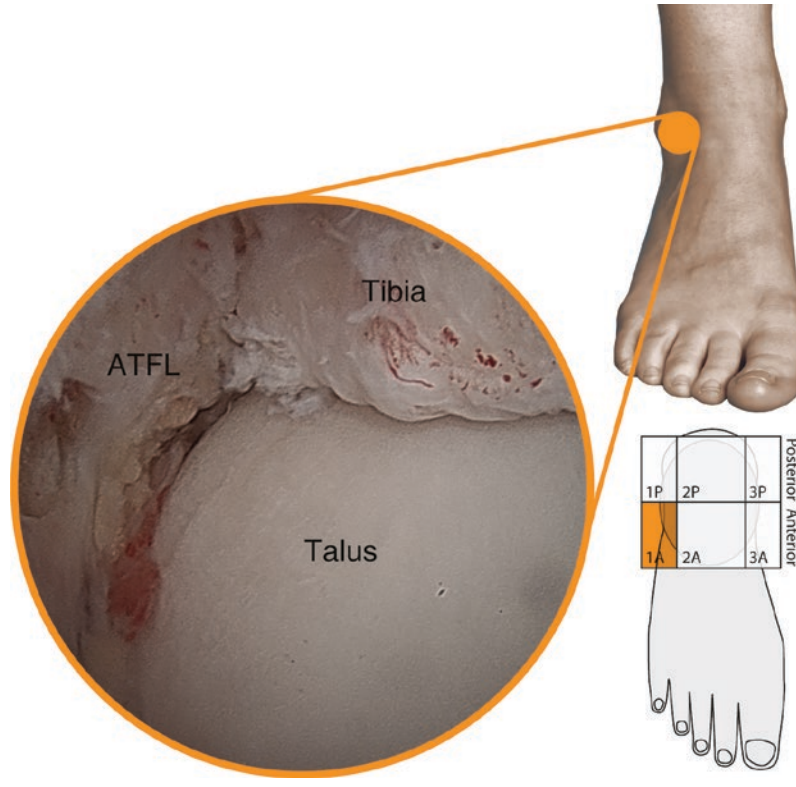


Fig. 4.4 The lateral forefront of the ankle joint, simply called "1A area." It may note the "trifurcation" point. Illustration by Tomaso Baj

Basset et al. [2] have shown up two separated fascicles of the ATFL, which cause sometimes an anterior impingement.

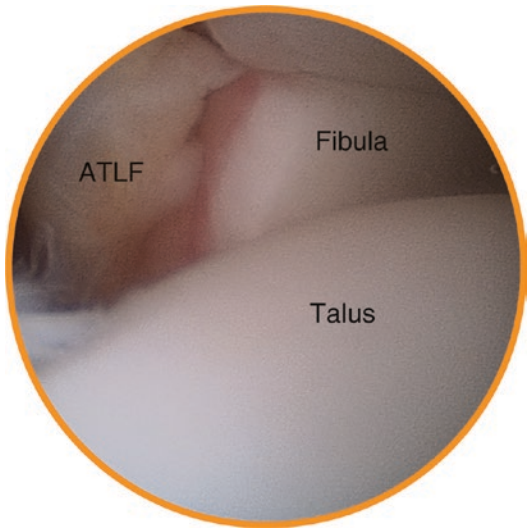


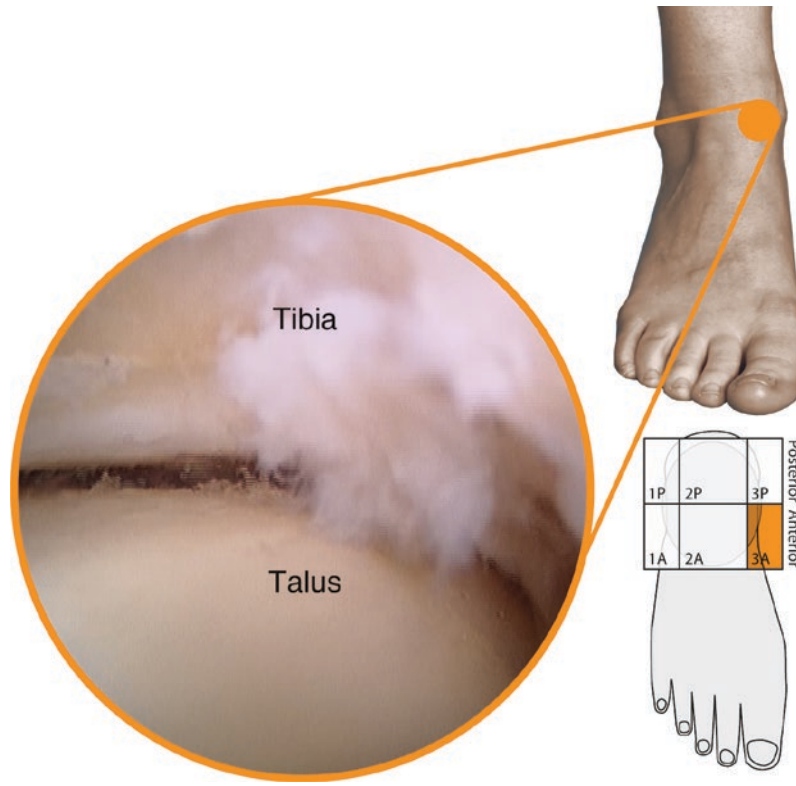
Fig. 4.5 The fibula can be shown applying a small traction on the ankle. Usually, patients affected by congenital hyperlaxity allow an easier visualization to this area

Pulling back the scope and moving it anteriorly, the space between the fibular border and the talar one is identified. The anterior inferior tibiofibular ligament comes down with the anterior tibiofibular ligament. Usually, it is a bare area or just fulfilled by the soft tissue envelope of the capsule.

After the lateral step, the anteromedial area is evaluated. It is important to be able to use the scope in order to switch it from medial to lateral portal possibly using two switching sticks to make it easy (Fig. 4.6).

Better visualization is achieved matching the scope through the AL portal. Looking directly at the medial side of the joint, the first structure examined is the deep portion of the deltoid ligament, proceeding from the tip of the medial malleolus vertically to the medial surface of the talus. In this area, ossicles might be found, inside the gutter, the deltoid ligament, and the medial dome of the talus. Be careful in maneuvering the scope in this area close by the notch to avoid iatrogenic cartilage damage.

Fig. 4.6 The anteromedial part of the anterior ankle joint, called "3A area," where osteochondral lesions are often found. Illustration by Tomaso Baj



An effective distraction allows to visualize the posterior central part of the ankle (2P), even the best visualization is performed from the posterior accesses. In fact, the treatment of the posterior compartment pathologies has to be made from behind.

Sliding medially from the lateral shoulder of the talus, the posterior inferior tibiofibular ligament (PITFL) is exposed, moving the scope about 45° angle from the posterolateral-central portion of the tibia to the lateral side of the talus.

The transverse tibiofibular ligament lies in the central part, appearing very different in dimensions and thickness, sometimes very thick, sometimes very large and strong. Shifting the scope medially, the medial portion of the tibiofibular ligament represents a capsular reinforcement of the flexor hallucis longus (FHL) tendon, appearing backward to the medial shoulder of the talus. The movement of the great toe helps the identitifaction of the FHL inside the joint. Often, it is quite difficult to check it from the anterior approach because of the synovial tissue in front.

The posterior-central dome of the talus is also a quite common area for osteochondral lesions of the talus, which can be set easily from the posterior approach with patient in prone assessment.

The 1P and 3P areas have to be shown through posterior portals, the same accesses we make for the subtalar arthroscopy [3]. The patient is in prone position, and the standard portals are opened: the posterolateral portal, above the tip of the lateral malleolus just lateral to the Achilles tendon, and the posteromedial portal, just medial to the Achilles tendon at the same level.

By these portals, the fatty tissue present in this area makes quite difficult any visualization. The soft tissue has to be removed with a shaver, pointing its tip anytime with the scope to avoid damages to anatomical structures. After removing this tissue, the posterior tibiofibular and talofibular ligaments appear: performing a sweeping motion and moving forward and back the scope, the thin subtalar capsule joint is visualized together with the subtalar joint (Fig. 4.7). Moving up the arthroscope, applying sometimes a manual distraction, the posterior compartment of the ankle joint can be seen.

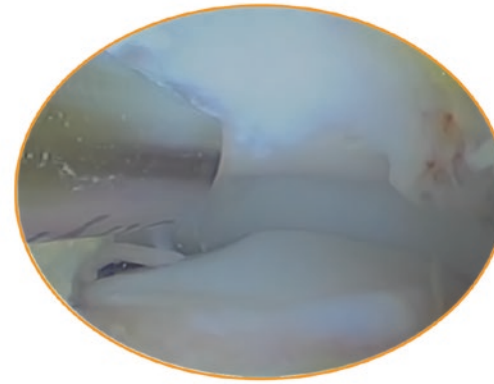


Fig. 4.7 The subtalar joint is visualized, after removing by a shaver the abundant soft tissue located in between

Rotating 90° the scope, it is possible to identify medially the flexor hallucis longus tendon, the most important landmark of the posterior procedure because it represents the barrier not to be passed because of the damage risks to the neurovascular posterior tibial bundle (Fig. 4.8).

The medial part of the posterior gutter (3P) reveals the deltoid ligament and posteromedial capsule which delimits the posterior from anterior gutter.

On the posteromedial talar dome are frequently placed osteochondral cystic lesions or defects which can be treated. The whole surface of the talus can be inspected as well (2P) (Fig. 4.9).

The tibial plafond is often obscured by the transverse tibiofibular ligament, which can be elevated or partially removed. The os trigonum and the posterior talar process are sometimes involved in fractures and/or partial detachment. Moving the scope laterally, the posterolateral side of the joint can be explored (1P) (Fig. 4.10.). The posterior syndesmotic ligament appears at 45° until the lateral gutter where the fibula lies on the talus.

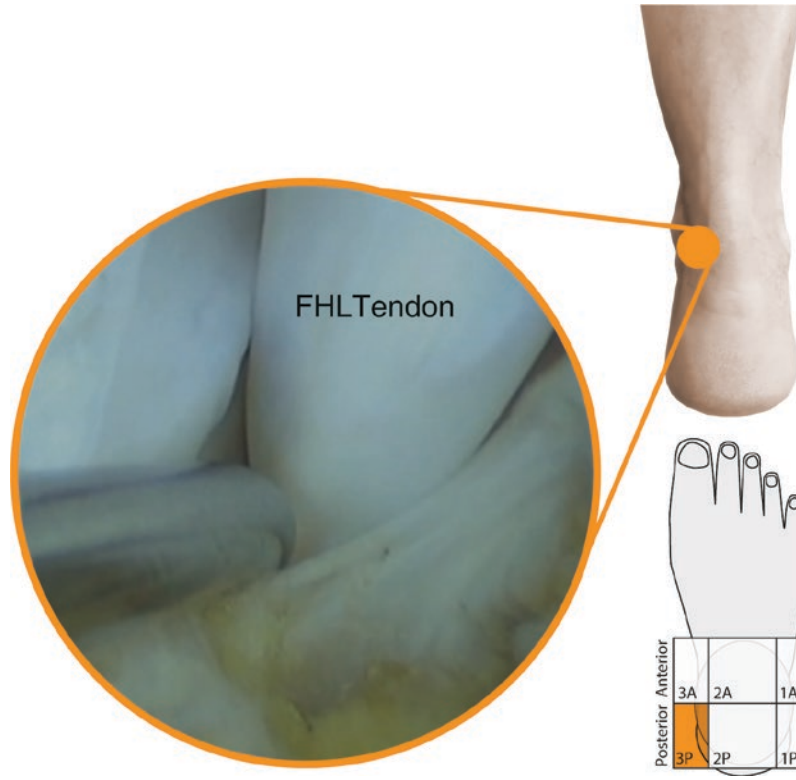


Fig. 4.8 The flexor hallucis longus tendon is visualized looking at the posteromedial area. This important landmark has not to be passed to perform a safe posterior procedure. Illustration by Tomaso Baj

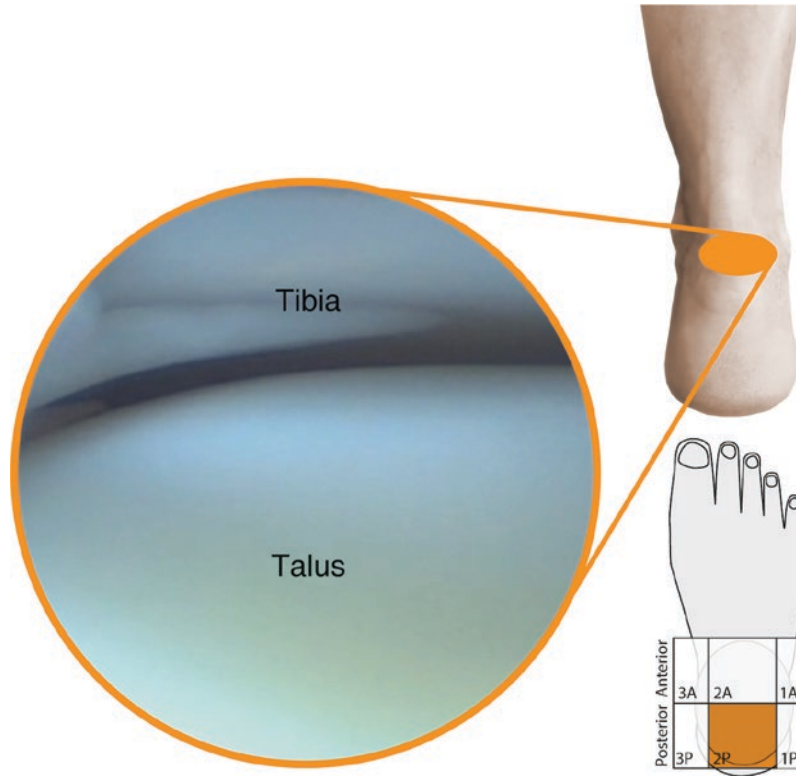


Fig. 4.9 The "2P area" shows a good view of the whole surface of the talus and tibial plafond. Illustration by Tomaso Baj

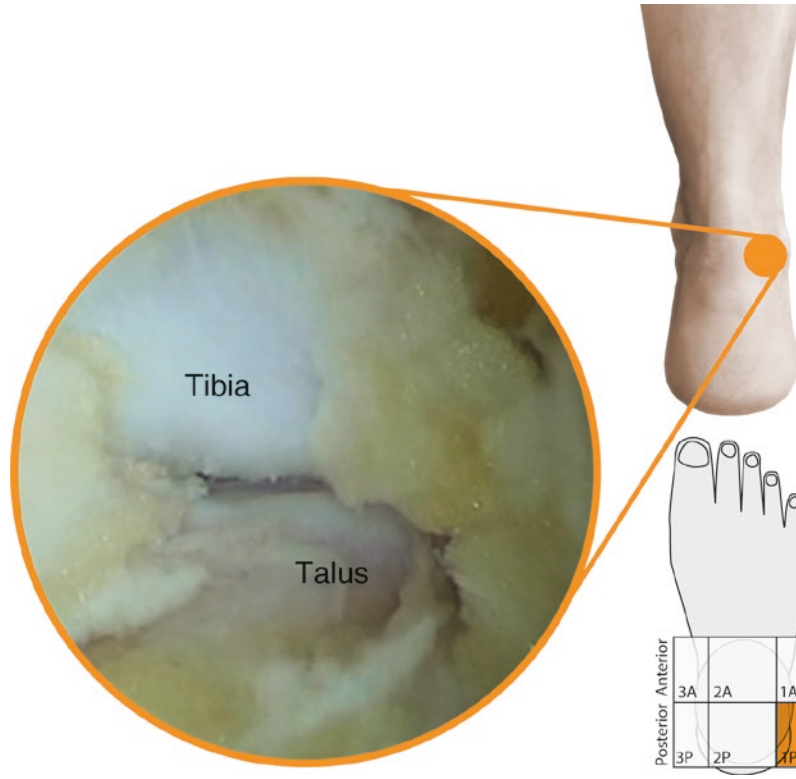


Fig. 4.10. The posterolateral side of the posterior ankle joint. Illustration by Tomaso Baj

4.1 Diagnostic Arthroscopy for the Subtalar Joint

For subtalar joint arthroscopy purposes, the joint can be divided in two different compartments, anterior (talocalcanealnavicular joint) and posterior (talocalcaneal joint). These two kinds of joint are anatomically divided by the sinus tarsi [4].

The indications included the posterior impingement, os trigonum, debridement and drilling of osteochondral lesions, talocalcaneal joint evaluation, loose bodies, Haglund's deformity, management of subtalar arthrodesis, and synovectomy.

The patient can be assessed in supine, prone, or lateral position, depending on pathologic condition that has to be treated and on concomitant procedures needed. If the patient is positioned prone, the subtalar arthroscopy is managed through the posterior portals, the same used for the posterior ankle arthroscopy and well described by van Dijk et al. [2].

If the patient is in a *prone position*, the foot and ankle hang over the end of the table with a small support placed under the distal tibia to make the ankle movement as free as possible and to displace the contralateral leg which might interfere during surgical maneuvering.

Small vertical incision is made slightly above the tip of the lateral malleolus. The mosquito clamp is used to direct, pointing toward the first interdigital space, until the bone is touched. At this point, the mosquito clamp is changed by a blunt trocar and then an arthroscope.

The second vertical incision is made medially at the same level of the posterolateral portal. Dissection of the subcutaneous tissue is performed with a mosquito clamp directed horizontally to the scope shaft, which is used like a guide when the trocar slips anteriorly until the ankle joint.

When the trocar is visualized, the 4.5 mm shaver is introduced. The fatty tissue makes difficult the visualization and has to be removed carefully. It is better to start from the lateral

side moving slowly to the FHL (flexor hallucis longus). This first part of the posterior arthroscopy takes few minutes to achieve a good visualization.

Na Smith et al. [5] divides the posterior hindfoot structures in four quadrants for a systematic description of the relevant anatomic structures: the superomedial, the superolateral, the inferomedial, and the inferolateral compartments.

This classification allows the identification of the extra-articular structures and shows a different point of view compared to the visualization from inside using the anterior accesses.

The subtalar joint appears, can be appreciate when the fatty tissue appearing in front of the arthroscope has been removed, starting from the lateral side, and the surgeon can find a working space and identify important extra-articular structures: a: the calcaneal-fibular ligament attached to the lateral malleolus delimits the lateral side, and keeps working in a counterclockwise direction; b: the posterior talofibular ligament (PTFL) which is identified secondarily, runs horizontally toward the posterolateral talar process (talus type I or normal tubercle). In this area, we might find the principal pathologies of the hindfoot, responsible for the posterior impingement: when the talar process is extremely enlarged, wide and symptomatic, is called Stieda process (talus type II), when is possible to identify an accessory bone this is called os trigonum (Talus type III); when there is a synchondrosis or a synfibrosis between the os trigonum and the talar process this called Talus type IV. Usually, the talar process or its variants are connected with the inferomedial structures, the posterior talocalcaneal ligament (PTCL), and the FHL retinaculum, which must be both released to allow its removal with a small osteotome working on either side.

Just behind the flexor hallucis longus retinaculum runs the FHL tendon, an important landmark that has not to be passed to maintain the safety procedure.

Sometimes, the hypertrophied intermalleolar ligament can be responsible for posterior impingement.

The visualization of the subtalar joint is challenging due to a small space between the two articular surfaces. Distraction is sometimes required. Different methods have been described to facilitate the view and the maneuvering in this area.

Recently, Beals et al. [6] described a minimally invasive technique utilizing a transosseous calcaneal K wire to achieve a distraction across subtalar and ankle joints having the patient in prone position. The 1.8 mm K wire, drilled through the calcaneus parallel to the floor, is tensioned with a traction device. Sartoretti et al. [7]suggest the angioplasty balloon catheters, placed into the ankle joint using the anteromedial portal or accessory portals. This technique has not been described for the subtalar joint.

Non-invasive ankle distraction, using the manual device and applying 135 N (30 lb) of force [8], increases the ankle space by 30% on average. However, the distraction is associated with nerve conduction changes, even though reversible, especially if used for 1 hour or longer [9].

We recommend a probe to lift up and distract the subtalar joint.

However, to achieve a full diagnostic subtalar examination or to treat specific pathologies of the anterior compartment or to performe a subtalar arthrodesis, it needs to use all portals on either side.

If the patient has to be positioned *laterally* the view is made through the anterolateral portal made just above the angle of Gissane and is about 2 cm anterior and 1 cm distal to the fibular tip and the middle portal placed about 1 cm anterior to the tip of the fibula [10]. The 2.7 mm 30° arthroscope and the 3.5 mm full-radius blade and burr are recommended in this technique.

Usually, placing the scope through the anterior portal and the instrumentations through the middle portal, the sinus tarsi is shown.

The lateral cervical ligament obstructs the view together with the medial, intermediate and lateral roots of inferior extensor retinaculum, and the dissection is blind at the beginning. After creating some space using the shaver, the

deep interosseous ligament filling the tarsal canal appears. Rotating the light cable at 90°, we look directly and anteriorly to the calcaneus neck. Rotating the light cable to the opposite direction, the posterior talocalcaneal joint can be evaluated.

Looking at the anterolateral side, shifting slowly the arthroscope backwards, we can identify the talocalcaneal ligament first and the calcaneofibular ligament just after. The talus and calcaneus articular surfaces are observed centrally, and rotating the device around its axis, they can be examined.

The posterolateral gutter may be seen from the anterior portal, sometimes just applying a small traction, and the Stieda process visualized as well.

Using the lateral positioning of the patient and placing the arthroscope through the anterior portal, we can reach enough view to the posterior facet. It allows the instruments maneuvering through the posterolateral portal.

However, placing the instrumentation through the anterior portal, it is possible to access the lateral aspect of the posterior facet.

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Part III

Impingement of Anterior/Posterior Ankle and Subtalar Joint

5 Bony Impingement: Aetiology, Classifications, Treatment, Arthroscopic Procedures, Pitfalls and Tricks

Fabrizio Cortese, Domenico Mercurio, Maria Pia Pasquali, and Piero Giardini

5.1 Anterior Bony Impingement

Anterior ankle impingement syndrome is very common in athletes who perform repeated ankle dorsiflexions (football players, American football players, dancers, gymnasts and runners).

Aetiopathogenesis is related to repeated sprains, chronic instability and repeated microtraumas associated with the formation of osteophytes.

Anterior bony impingement is due to the formation of osteophytes along the anterior margin of the tibia and/or on the talus neck [1].

Once the osteophyte has formed, ankle dorsiflexion causes trapping and inflammation of the soft tissue like the anterior ankle capsule, generating pain during the activity and limiting the articular range of motion.

In front of the joint, we find a triangular area of soft tissues composed of synovial membrane and adipose tissue that is normally compressed between the talus and the tibia at 15° dorsiflexion. If there are anterior osteophytes, the space for this tissue becomes smaller, causing its impingement and leading to chronic inflammation [2–4].

In 1943, Morris was the first to describe ankle impingement as a frequent cause of pain in professional football players or in those athletes who perform repeated ankle dorsiflexion and plantarflexion movements [5].

Also McMurray, later in 1949, described this chronic lesion in professional football players over the age of 25 years with anterior pain and ankle pain in dorsiflexion.

Morris and McMurray called this condition "athlete's ankle" or "footballer's ankle" [5, 6].

Over the years, several authors have associated anterior bony impingement with various sports activities, such as running, dancing and volleyball [7].

The term "footballer's ankle" was therefore replaced with "anterior ankle impingement syndrome" and was differentiated between impingement of the soft tissues and bony impingement [8–10].

We will later see that the term "footballer's ankle" indicates a specific type of impingement, which has its own characteristic pathogenesis and localisation.

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5.2 Classifications

5.2.1 Topographic Classification of Anterior Impingement

Impingement is classified according to the site of formation of the osteophytes. The anterior portion of the ankle is divided into three areas (anteromedial, anterior and anterolateral). It is very interesting that the site of osteophytes formation corresponds to a different pathogenetic mechanism [11].

5.2.1.1 Anteromedial Impingement

Anteromedial impingement is characterised by tibial and talar osteophytes due to repeated supination-sprains that create a chronic microtraumatism by impact between the bone surfaces. Chronic microtraumatism secondarily causes the formation of hyperostosis and calcifications.

5.2.1.2 Anterior Impingement (Fig. 5.1)

Anterior impingement is the characteristic "footballer's ankle". The osteophytes are along the anterior margin of the tibia and talus. The formation mechanism is mainly due to repeated microtraumatism such as the one created during the instep shot in football. Some authors had proposed a mechanism for the formation of traction osteophytes on the anterior articular capsule. Subsequent anatomo-pathological studies by Tol and van Dijk showed that the site of osteophyte formation is not at the point where the articular capsule fits into the bony surfaces, but is instead intra-articular [12].

Therefore, the activities that produce repeated traumas are more related to the development of an impingement than those that produce capsular traction only [4].

5.2.1.3 Anterolateral Impingement

Anterolateral impingement is rarely bony, but is generally caused by soft tissues and lies outside this subject matter.

5.2.2 Arthritis Classification According to van Dijk

This classification was proposed for ankle arthritis, but it is also very interesting for the bone impingement chapter since it provides important prognostic information. The clinical result that can be achieved with isolated removal of the osteophytes will be much better the less the joint is affected by arthritis process.

This classification is made up of four stages: grade 0, normal joint or subchondral sclerosis; grade 1, osteophytes without reduction of joint space; grade 2, reduction of joint space with or

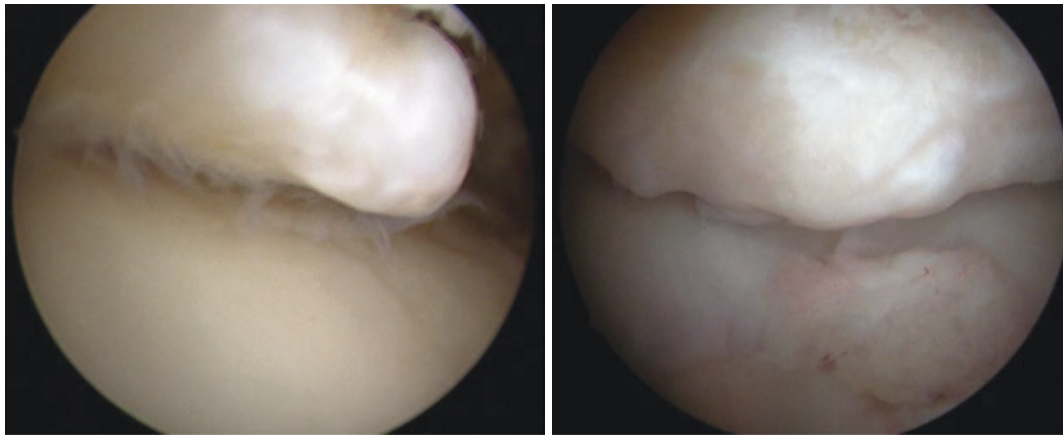

Fig. 5.1 Arthroscopic view of anterior bony impingement

without osteophytes; and grade 3, (sub)total disappearance or deformity of the joint space [13].

5.3 Clinical and Diagnostic Investigations

The patient generally reports anterior pain initially associated with physical activity and, at the ending stage, also at rest.

The physical examination will be marked by pain with digital pressure, especially in the specific impingement area (anteromedial, anterior or anterolateral), by positive results in stress tests (pain in forced dorsiflexion) and by limitation in the articular range of motion.

The first diagnostic investigation is conventionl radiography that provides us many information regarding the bone impingement. The lateral view will clearly show us the classic anterior impingement (footballer's ankle) characterised by osteophytosis along the anterior margin of the tibia and along the anterior border of the talar neck. In order to diagnose the anteromedial impingement with radiography, it will be essential to perform the oblique anteromedial view described by van Dijk (30° external leg rotation, oblique radial inclination 45° with respect to the longitudinal axis of the leg) [14] (Fig. 5.2).

Completing the diagnostic procedure with an MR examination is mandatory before all surgical approach, especially in order to highlight any associated osteochondral lesions.

5.4 Conservative

Conservative treatment is the initial approach to the management of both anterior and posterior impingement for a period of at least 3–6 months, altought there is no approval or validation of the conservative treatment method.

In acute cases is recommended rest, ice therapy and oral/local anti-inflammatory drugs; while in chronic cases are indicated orthesis and, rehabilitation protocols to improve stability, proprioception and development of the peroneal muscles.

Various authors have reported an improvement in symptoms following corticosteroid injections [4, 15, 16].

Surgical treatment is indicated where symptoms persist after 6 months of conservative treatment.

In bony impingement, surgical treatment consists of the removal of the synovial hypertrophy and osteophytes.

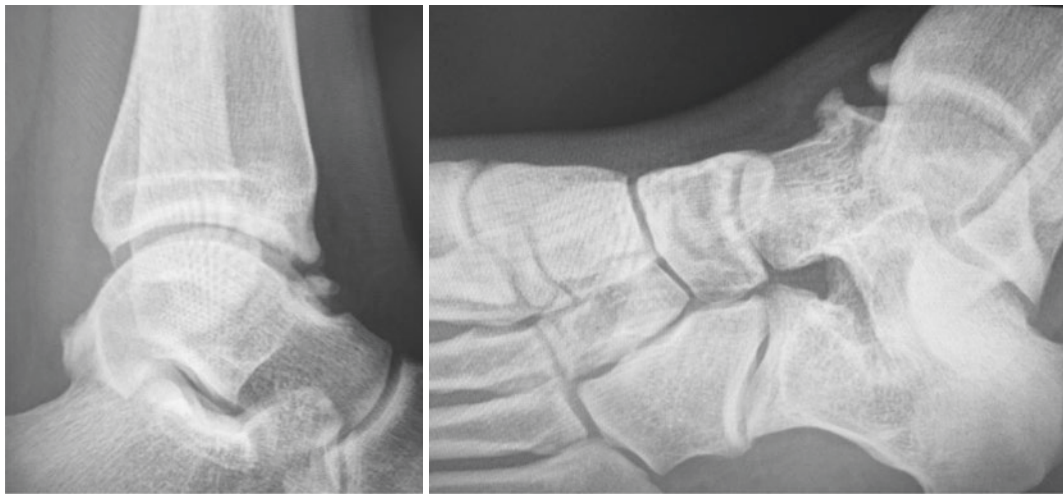

Fig. 5.2 Lateral view shows anterior ankle impingement; van Dijk view shows anteromedial impingement

5.5 Arthroscopic Debridement: Step-by-Step Tricks to Avoid Pitfalls

Arthroscopic debridement has replaced arthrotomy debridement. Although there are no differences in surgical time, the athlete returns to the sport quicker with arthroscopic treatment, and there is a lower risk of complications [17, 18].

5.5.1 Position

The patient should be placed in the supine position with the foot on the edge of the operating table, so that a passive dorsal and plantar flexion can be performed during surgery. The operated ankle must be higher than the contralateral ankle so the instruments can be manoeuvred freely and easily.

It is advisable to keep the ankle free for the arthroscopic procedure, without traction, following the technique described by van Dijk. In this way, it is possible to work on the osteophytes and on the structures of the anterior chamber in complete dorsal flexion, protecting the cartilage surface and the anterior neuro-vascular structures [11, 18].

5.5.2 Arthroscopic Accesses

Following the technique, first, the anteromedial access is performed near the medial margin of the tibialis anterior tendon in maximum dorsal flexion of the ankle. If a too medial access is made, the approach to the lateral malleolus and the peroneal osteophytes may be difficult because of the convexity of the talar bone.

The anterolateral access should be made along the lateral edge of the third peroneal tendon under arthroscopic transillumination while being very careful not to injure the superficial peroneal nerve. To protect the nerve, it is usually evidenced before the procedure with a dermographic pen; or, if this is not possible, it is highlighted in transluminency during arthroscopy. In the case of a medial lesion, it is recommended to not stay too lateral.

If the impingement is medial, the working arthroscopic access is medial, or vice versa if you have to work laterally. If we are faced with the classic "footballer's ankle" with anterior osteophytosis, both portals have to be used for the instruments, which makes frequent changes of portals necessary for the optics.

5.5.3 Removal of Osteophytes

First of all, the synovial hypertrophic tissue is thoroughly cleaned, and then the osteophytes are removed. Several instruments can be used for removing osteophytes, such as little osteotomes, a cutter blade for arthroscopy or small burs (acromionizer) inserted through the arthroscopic portals (Figs. 5.3 and 5.4).

For tibial osteophytes, resectioning should be performed from proximal to distal and for the talus osteophytes from distal to proximal in order to define and follow the anatomical border of the anterior tibia and talar bone [11].

5.6 Posterior Bony Impingement

A painful impingement syndrome may also affect the posterior ankle, caused by both overuse and the post-traumatic condition.

Dancers, runners (above all those who run downhill) and gymnasts (high, long and triple jump) are the athletes most affected by this painful syndrome caused by overuse; but also football, basketball and volleyball players are subject to it. The mechanism that causes the painful syndrome is an athletic movement that requires repetition of forced plantar flexions such as those performed in classical ballet (frequent "en pointe" and "demi pointe" positions) [11, 19, 20].

5.7 Anatomical Substrate and Aetiopathogenesis

The posterior bone conflict is created between the posterior edge of the talus bone and the posterior edge of the tibia. The talus bone is posteriorly

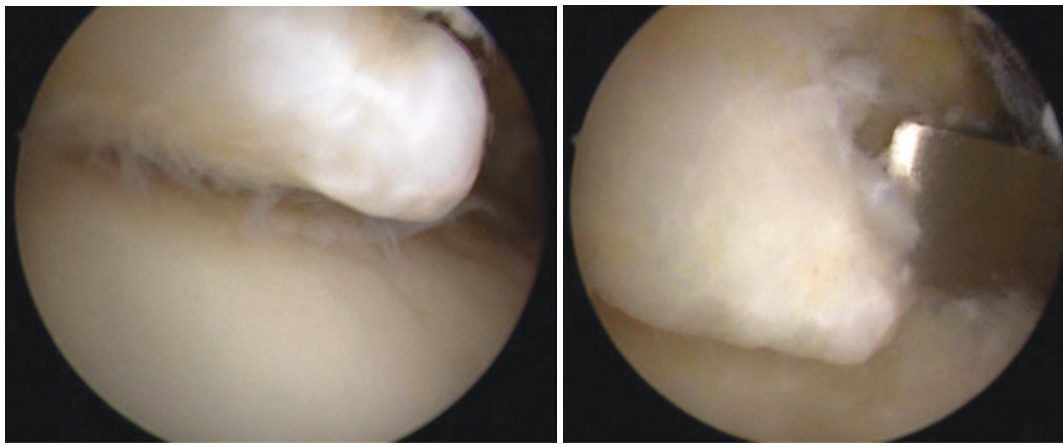

Fig. 5.3 Anterior ankle impingement treated with osteotome

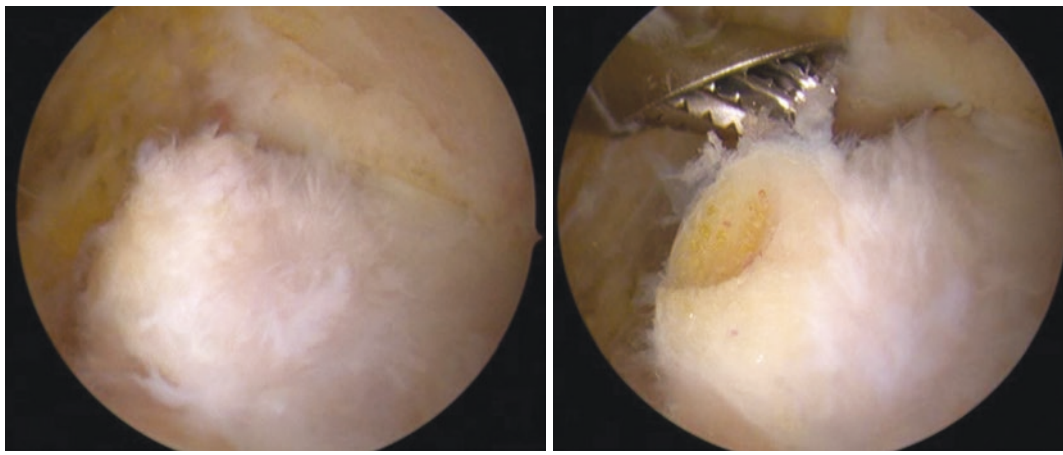

Fig. 5.4 Anteromedial ankle impingement treated with acromionizer

characterised by posterolateral and posteromedial processes. The two structures form the groove for the flexor hallucis longus (FHL) tendon. The anatomical relationship between the posterolateral process and the FHL often results in the association of bone conflict syndromes with FHL tendinopathy.

5.7.1 Overuse Impingement Syndromes

The posterolateral process develops from a distinct growth nucleus compared to that of the talus. About 7% of the adult population does not complete fusion with the talus bone, so it remains an accessory bone called os trigonum, connected with the talus by a fibrous synchondrosis [21].

Os trigonum represents the most frequent anatomical substrate on which a chronic stress or a single traumatic event can trigger the syndrome.

During plantar hyperflexion, altered/abnormal structures such as an os trigonum or a hypertrophic posterior articular capsule may be compressed (mechanism called "nutcracker").

Chronic stresses, such as those performed by footplayers (plantar flexion when the ball is hit) or dancers, or a traumatic event in forced plantar flexion may lead to mobilisation of the os trigo-

num from the talus and therefore to a secondary inflammation with formation of fibrous tissue. Pain is caused by both inflammation of the posterior tissues (capsule, synovia) and the secondary FHL tendinopathy, due to the impingement with the os trigonum [22–24].

5.7.2 Traumatic Causes

A trauma in forced plantar flexion can cause the fracture of the posterolateral process of the talus (Shepherd's fracture), creating symptoms very similar to those caused by the os trigonum with which it enters into differential diagnosis. It should be kept in mind that the prognosis is better in the case of overuse syndromes as compared to traumatic cases [20].

In the posterolateral process, the posterior talofibular ligament (PTL) is inserted. It is the most resistant lateral ligament of the ankle and is tensioned in maximal dorsiflexion. Therefore, a trauma in hyperdosiflexion may result in the avulsion of the posterolateral process or of the os trigonum [11].

5.7.3 Other Causes

A posterior impingement may also result from previous fractures or from arthritis that leads to the formation of sub astragalic and tibiotarsal joint osteophytes. A posterior conflict may also be due to calcifications caused by chronic inflammation of the soft tissues.

Further causes are pseudoarthrosis of fractures of the posterior malleolus, talar or calcanear loose bodies and chondromatosis.

5.8 Clinical and Diagnostic Investigations

5.8.1 Clinical Examination

5.8.1.1 Medical History

The diagnosis of posterior ankle conflict is based initially on the medical history in order to understand the cause, localisation and duration of the symptoms. Usually in acute trauma, the patient reports a trauma in forced hyperplantarflexion, while in chronic cases the pain is due to a repetitive movement in forced plantar flexion, often associated with the presence of the os trigonum. The pain is usually located posterolaterally; in case of FHL involvement, the pain is posteromedial [25].

5.8.1.2 Objective Examination

Posterolaterally palpation reveals pains between the peroneal tendons and the Achilles tendon, where is located the posterolateral process. Posteromedially, digital pressure can cause pain especially if an FHL tendinopathy is present. Attention should be paid to the exact localisation of the painful spot because the symptoms may be due to a tenosynovitis or a posterior tibial nerve dysfunction and not to a posterior impingement [26].

There may be articular limitation, especially in plantar flexion. In chronic cases, inflammation of the FHL tendon may be associated, which causes reduction of the motility of the hallux (especially in dorsiflexion) as a result of secondary fibrosis [25].

Pain can be reproduced with the passive plantar flexion test carried out with rapid hyperplantarflexion movements with the patient's knee flexed at 90°. Also a slight external or internal rotation movement can be applied. Applying a rotation movement at the point of maximum plantar flexion generates a compression (grinding) of the posterior process of the talus or of the os trigonum between the tibia and heel. On the other hand, a negative test makes diagnosis of posterior impingement less likely, but there are no studies on the specificity and sensitivity of the test in diagnosing posterior impingement [26, 27].

Finally, a complete neurovascular evaluation should be carried out to exclude tarsal tunnel syndrome [28].

Injection of local anaesthetic helps to confirm the diagnosis. Differential diagnosis includes different conditions: fractures of the posterior tibiotalocalcaneal complex, osteochondral lesions of the talus and/or tibia, tarsal *synostosis,* soft tissue lesion and tendon disorders [25].

5.8.2 Investigations (Fig. 5.5)

Antero-posterior and latero-lateral (AP-LL) X-rays may show an acute or chronic fracture of

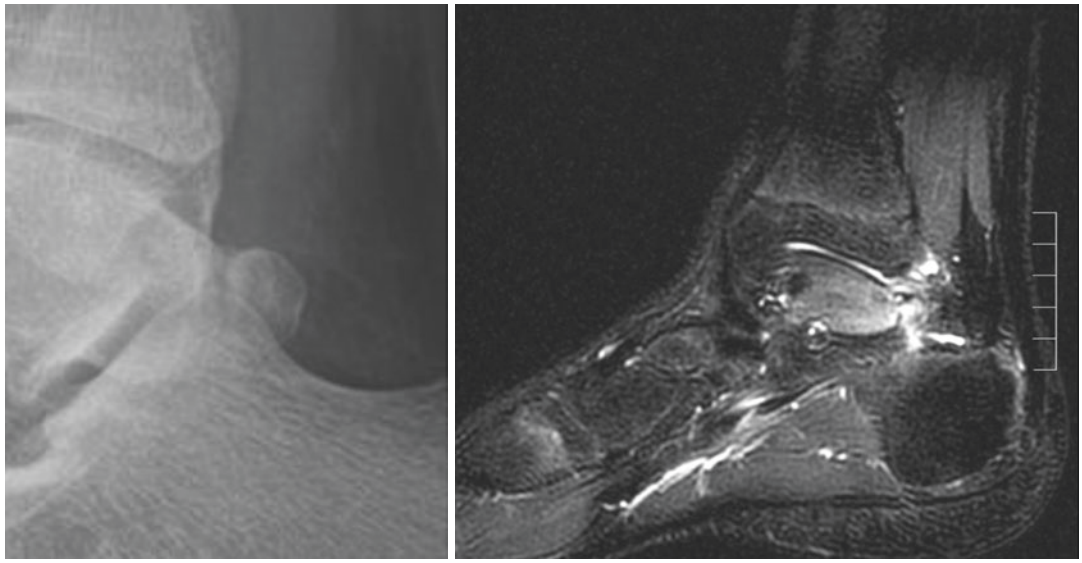

Fig. 5.5 Lateral X-ray shows os trigonum; MR shows bone and soft tissue posterior edema

the posterolateral process, as well as the presence of an intact or fractured os trigonum, arthrosis or chronic inflammation of soft tissues with calcifications [29, 30].

Any presence of a posterior talar process or of an os trigonum is usually found in lateral view. However, these posterolateral anatomic variants are not always visible in lateral view because they are covered by the medial tubercle of the talus. Therefore, a lateral view with the foot in 25° external rotation is recommended. Dynamic lateral view in hyperplantarflexion and dorsiflexion may show abnormal bone contact [31].

The MR shows any possible bone edemas of the os trigonum or of the posterolateral process, effusions, concomitant synovitis of the FHL tendon, chondral lesions and muscular anomalies (peroneus quartus, flexor digitorum accessorius longus) [32, 33].

5.9 Conservative

5.9.1 Treatment

Acute and chronic cases of posterior bone impingement syndrome show no differences in recovery and return to the sports activity.

Pain control and resumption of the plantar flexion can, in the first instance, be achieved with physical therapy and rehabilitation. The initial treatment of a posterior bone conflict syndrome should be focused on reducing inflammation: NSAIDs, cortisone and local anaesthetics infiltrations and movement restrictions (avoid plantar hyperflexion).

During rehabilitation, the symptoms can be relieved by reducing the activity of the gastrocnemius muscle and by performing exercises to strengthen the muscle and proprioception exercises.

Once the pain has lessened, patients can follow a physical therapy programme that includes isometric exercises, Achilles tendon stretching and customised isometric reinforcement.

In a study by Hedrick et al., non-surgical treatment consisting of physical therapy and rehabilitation has shown to be effective in approximately 60% of patients with posterior ankle impingement syndrome [29, 34–36].

5.9.2 Surgical Treatment

It is indicated if the symptoms persist 3–6 months after conservative treatment.

Just like anterior impingement, the aim of the surgical treatment is to remove the anatomical cause. Symptoms are commonly resolved with the removal of the painful os trigonum and with debridement of the inflamed and hypertrophic soft tissues.

Surgical treatment may be open, by medial or lateral access, or arthroscopic.

Dancers rappresent 61% of patients who undergo surgery for posterior impingement.

In their analysis of 47 articles and 905 patients treated surgically, both with open and arthroscopic approach, Ribbans et al. reported 67–100% good or excellent results [37].

5.9.3 Open Debridement

A lateral approach between the long peroneal muscles and the FHL offers a more direct access to the posterolateral process with less risk for the medial neurovascular bundle. However, a medial approach let to treat easier the concomitant FHL pathology [29, 38].

5.9.4 Arthroscopic Debridement

Since 2000, the posterior arthroscopic approaches have gained in popularity, with the possibility of a faster return to sports and a lower rate of minor complications [39–41].

In their review of 16 studies, Zwiers et al. concluded that arthroscopic treatment has a lower rate of complications than open treatment (7.2% vs. 15.9%) and an earlier return to the sports activity (11.3 vs. 16 weeks) [42].

Posterior ankle arthroscopy is a challenging technique that requires excellent arthroscopic ability and an excellent knowledge of the posterior anatomy.

5.10 Arthroscopic Debridement: Step-by-Step Tricks to Avoid Pitfalls

5.10.1 Patient's Position (Fig. 5.6)

The patient is in the prone position, with the foot hanging on the edge of the table and perpendicular to the floor. The ankle must be free to be dorsiflexed or plantar flexed. The contralateral limb is lowered to facilitate handling of the instruments [11, 26].

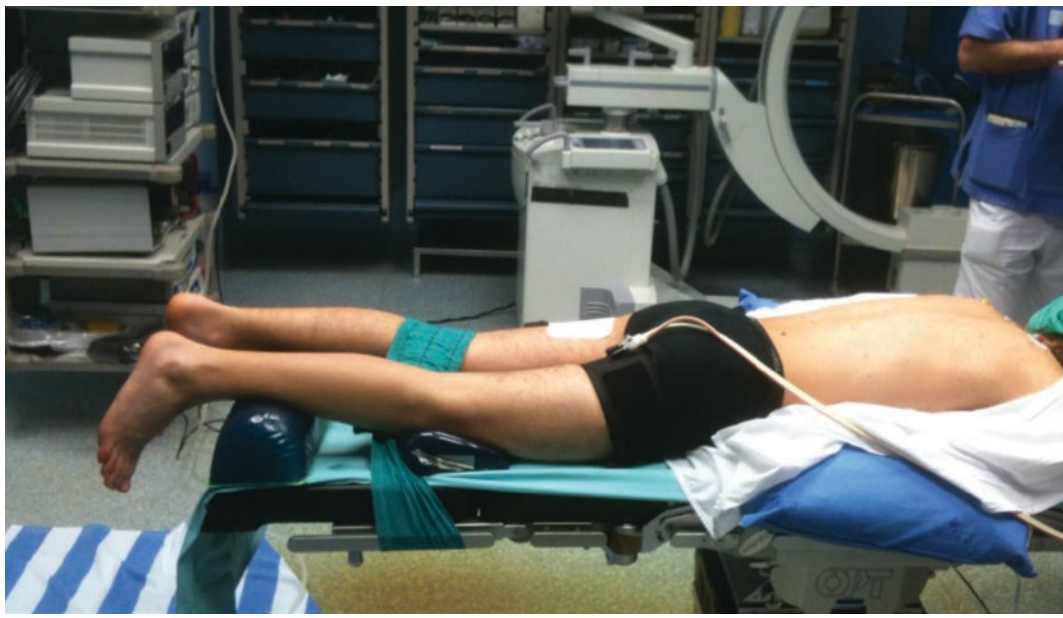

Fig. 5.6 Patient position

Several approaches have been proposed in the presence of simultaneous anterior and posterior impingement:

- Anterior supine arthroscopy, to then turn the patient over into the prone position for the posterior approach, or vice versa.
- Prone position: Perform the posterior arthroscopy and then flex the knee at 90° and perform the anterior arthroscopy, which can create confusion because the arthroscopic view of the joint is upside down. We believe that this option should be recommended to experienced arthroscopic surgeons only.

5.10.2 Arthroscopic Portals and Principles of the Posterior Arthroscopic Technique

The posterolateral and posteromedial portals are the most widely used and are performed just laterally and medially to the Achilles tendon. It is useful to mark the reference points before incising the skin. With the ankle in a neutral position, a line is drawn from the apex of the lateral malleolus to the Achilles tendon. The accesses are made just above this landmark line (Fig. 5.7).

The posterolateral access is made first. After skin incision, clamp and arthroscope are introduced through the lateral portal, pointing towards the second metatarsus until the posterior surface of the talus bone (Fig. 5.8). Next, at the same height as the posterolateral portal, the posteromedial portal is made. Also in this case, we will introduce a clamp that will point just anteriorly towards the Achilles tendon, ending up touching the sheath of the arthroscope [11, 43, 44].

Then the shaver is introduced at about 90° with respect to the arthroscope. The shaver is slid forward along the arthroscope access until it is displayed by the camera. In this phase, there will be no reference points, and we are surrounded by soft tissues, so it is crucial to initiate cleaning of the posterior articular space in safe conditions. The aspirator of the shaver is only used when the cutter is stopped to minimise the risk of "pulling" neurovascular structures into the cutter [45].

The space between the soft tissues is created from lateral to medial. To work safely, the strategy described by van Dijk is to display the FHL tendon since the beginning of procedure and work with the instruments always laterally to it. The FHL tendon is always posterolateral to the neurovascular bundle, so if the surgeon keep his instruments always laterally to it, there are no risks of injury to the noble structures of the bundle [11, 26]. When the FHL is isolated, the posterior articular space is to be cleaned by removing any loose bodies, the synovial hypertrophic tissue, and isolating the cause of the impingement, which is generally the os trigonum (Fig. 5.9).

5.10.3 Os Trigonum Removal

The edges of the os trigonum should be defined before resection. The resection must then begin at the posterior edge of the bone prominence to avoid free bone fragments in the retrocalcaneal space. The os trigonum is cleaned circumferentially from the surrounding tissues using the shaver and arthroscopic radiofrequency; the synchondrosis between the talus and os trigonum is interrupted using a lever manoeuvre with an osteotome that must be inserted into the cleavage plane between the two bone structures. Once completely free, the os trigonum is removed with forceps [27, 46, 47] (Fig. 5.10).

5.10.3.1 Postoperative Treatment

There is no need for immobilisation in a cast for either anterior or posterior bone conflict. Usually, a bandage is used for a few days. Partial load is allowed immediately, depending on the tolerance. Once the first postoperative phase is over, assisted physical therapy will begin, with the aim to recover the complete ROM, muscular strength and proprioception.

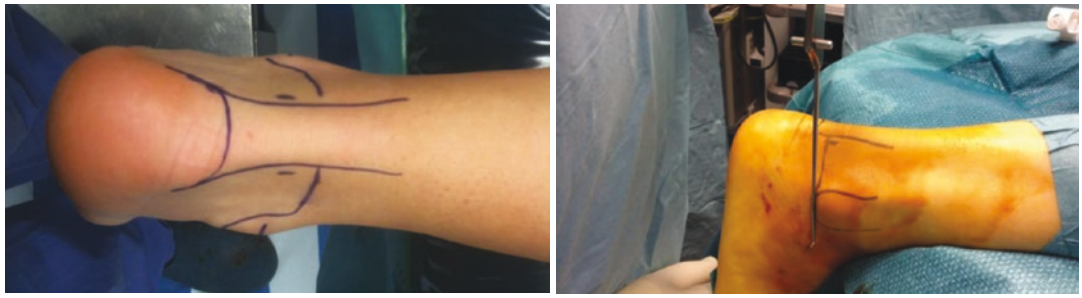

Fig. 5.7 Arthroscopic portals

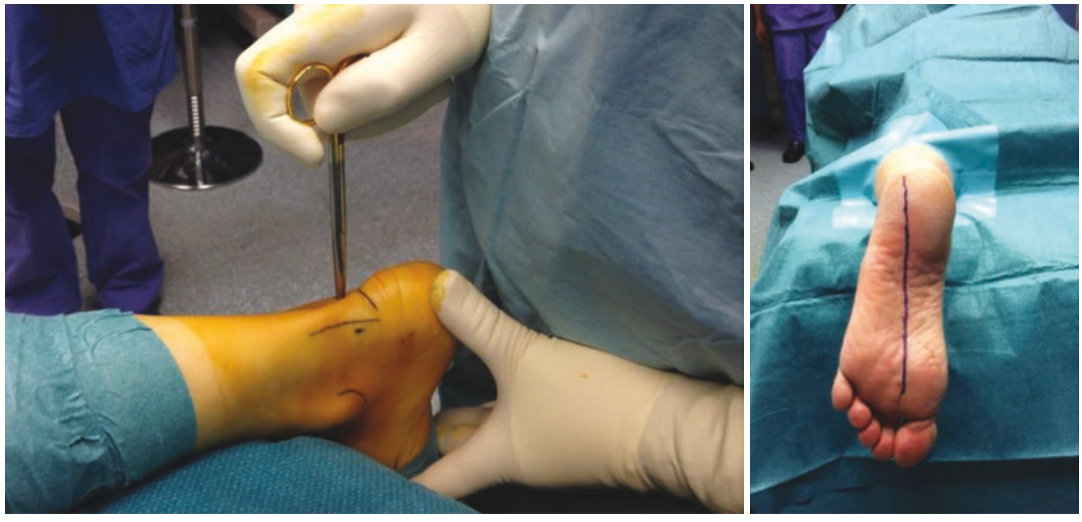

Fig. 5.8 Arthroscopic approach

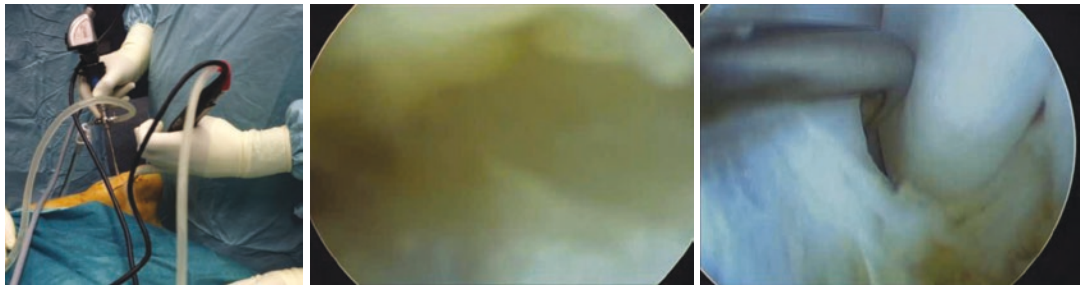

Fig. 5.9 Arthroscopic identification of FHL

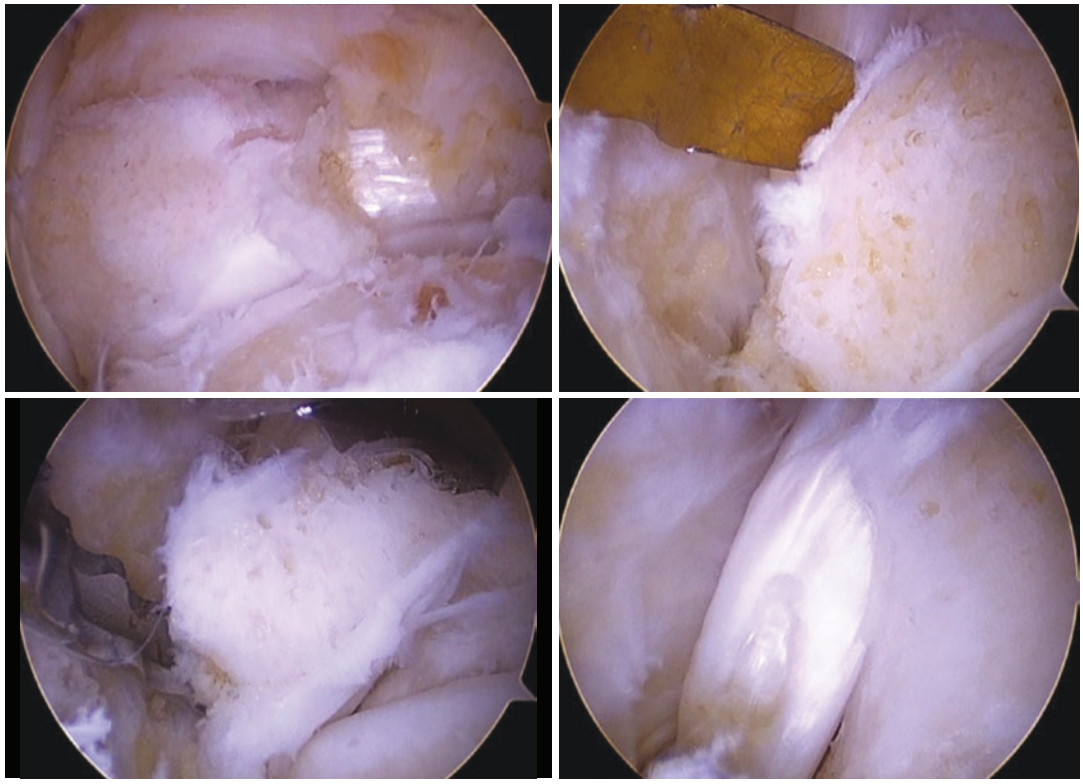

Fig. 5.10 Os trigonum removal and impingement resolution

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6 Soft Tissue Impingement: Etiology and Classification, Treatment, Arthroscopic Procedures. Pitfalls, and Tricks

Angelo Bertelli, Alberto Marangon, Elisa Facci, and Piergiuseppe Perazzini

6.1 Introduction

One of the most frequent causes of chronic ankle pain is impingement syndrome, which can be determined by bony prominences, known as osteophytes, located around the joint or soft tissue disorders. The painful condition is caused by friction in the joints.

A traumatic event, such as a sprain or a fracture lesion, may be the "primum movens" in the formation of intra-articular fibrous and synovitic tissue, responsible for pain and altered joint function.

Patients are generally young, often professional sportsmen/women with chronic ankle pain located all around the anterior gutter, in the back of the joint, or both locations, sometimes associated with a reduced range of motion (ROM).

Conventionally, impingement syndromes are divided into anterior (anterior-medial, central, and anterior-lateral) and posterior.

Over the years, arthroscopic surgery has become an increasingly common technique due to its indisputable advantages, especially for patients.

The surgeon has a duty to carefully select patients to treat and to be very precise in the indications and therapeutic choices to be adopted. Accurate differential diagnosis is vital.

6.2 Etiology and Classification

6.2.1 Anterolateral Impingement

The anterolateral region of the ankle is the area most vulnerable to soft tissue impingement, often resulting from sprains or repetitive microtrauma with the foot in the plantarflexion and supination position. This pathological condition takes a number of forms: meniscoid syndrome, chronic synovitis with or without associated ankle instability, or an anatomical thickening of the distal bundle of the anterior inferior tibiofibular ligament (AITFL).

The anatomy of the anterolateral gutter is bounded laterally by the fibular malleolus and posteromedially by the tibia; the anterolateral zone is bounded by the joint capsule and anterior tibiofibular ligament, talofibular anterior ligament, and calcaneofibular ligament.

After a sprain, with involvement of the joint capsule and ligamentous structures, hemorrhage may ensue with inflammation, hypertrophy, and irritation of the soft tissue, causing anterolateral impingement, even in the absence of significant ankle instability (Fig. 6.1).

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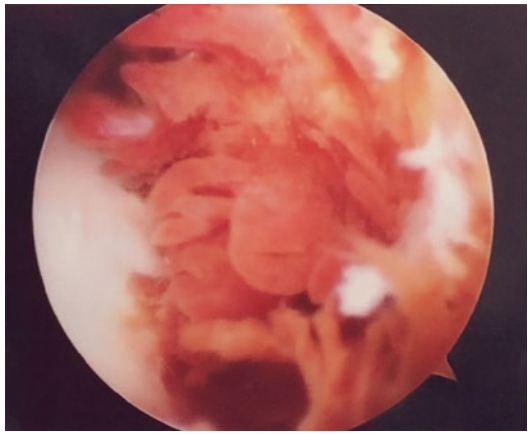

Fig. 6.1 Hypertrophy and irritation of the soft tissue

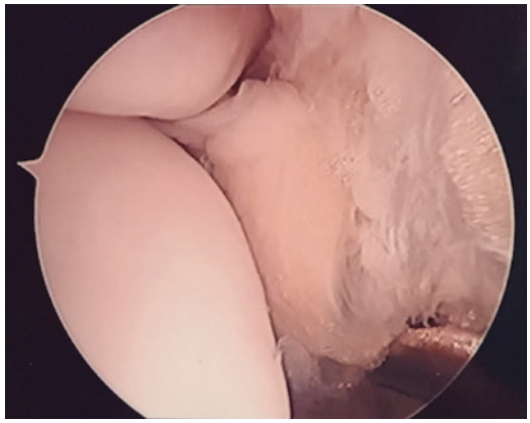

Fig. 6.2 The meniscoid syndrome of the anterolateral region

Secondarily, with injury to the lateral ligamentous component of the ankle, degeneration of the intra-articular tissue may ensue, particularly of the anterior talofibular ligament, giving rise to "meniscoid syndrome" (Fig. 6.2).

This syndrome, described by Wolin in 1950, is a post-traumatic painful condition of the anterolateral compartment of the ankle [1]. Over the years, this pathological condition has come to be associated with pain syndrome resulting from repeated trauma, typical in athletes [2].

Clinically, patients complain of sharp pain in the anterolateral part of the ankle during palpation, becoming stronger during movements for the pronation and supination of the foot.

Another cause of anterolateral impingement is chronic synovitis, again as a result of trauma in inversion or rheumatologic diseases. It should always be posited in cases of chronic post-traumatic pain, even in the absence of ligamentous laxity.

Ankle impingement by the distal bundle of the anterior inferior tibiofibular ligament (AITFL) is relatively unknown.

Anatomically, the distal part of the tibiofibular joint is known as a syndesmosis, stabilized by three ligaments: the interosseous, the posterior tibiofibular, and the anterior tibiofibular ligament. In the latter, it is sometimes possible to find a supplementary distal fascicle, i.e., Bassett's ligament.

Bassett et al. were the first to suspect AITFL as a cause of anterolateral impingement, while Nikolopuolos was the first to describe the anatomy of this supplementary ligament, following cadaveric dissection, in the belief that it was not part of the excess of the anterior tibiofibular ligament, but a genuine ligament, with a reported incidence in the literature of 21–92%.

Explaining the pathogenesis of this impingement, Bassett et al. state that in a lax condition after traumatic injury, dorsiflexion of the foot causes an anterior slip of the talus, conflicting with the distal bundle of the AITFL. The anatomical features and the size of the accessory bundle seem to be relevant too. These findings have been reported by several authors as a result of abundant arthroscopic experience [3, 4].

Anterior ligament impingement should always be posited in patients with chronic ankle pain, with a history of sprains even in the absence of radiographic evidence of pathological alteration.

The choice of treatment should always take into consideration the pathological mechanism of the trauma and its cause, even if the sprain is slight. In the absence of osteochondral lesions, treatment should be conservative for at least 6 months after the trauma.

The therapeutic decision is based on the clinical history of the patient and careful physical examination.

Arthroscopic treatment cleans out the synovitis and reshapes the distal bundle of the AITFL. Ligament reconstruction may be considered in cases of instability [5].

6.2.2 Anteromedial Impingement

The incidence of anteromedial impingement syndrome is much less frequent. It should be suspected in patients with chronic anteromedial pain following inversion sprains or fractures, even with a stable ankle.

The deltoid ligament comprises a superficial and deep layer. These layers, if injured by trauma, may be the reason for the impingement syndrome. The diagnosis is essentially clinical with localized pain in the anteromedial portion of the ankle, awakened by palpation and by dorsiflexion and inversion, due to the impact of the ligamentous component against the talus.

Much rarer are sprains by eversion of the foot, albeit possibly causing partial or complete rupture of the deltoid ligament. They are the cause of anteromedial impingement due to hypertrophy of the soft tissue, similar to lateral meniscoid syndrome (Fig. 6.3).

Injury to the ligamentous lateral complex, by repeated sprains in inversion, may result in pain associated with medial impingement. In high-speed trauma, as in sports involving running or jumping, significant tensile forces are exercised on the lateral compartment, resulting in ligament rupture with compression between the internal face of the medial malleolus and talus.

Following this, osteochondral lesions and especially synovitis, injuries of the deltoid ligament or capsule, and the fibrous reparative processes as a result of hemarthrosis are frequent [6].

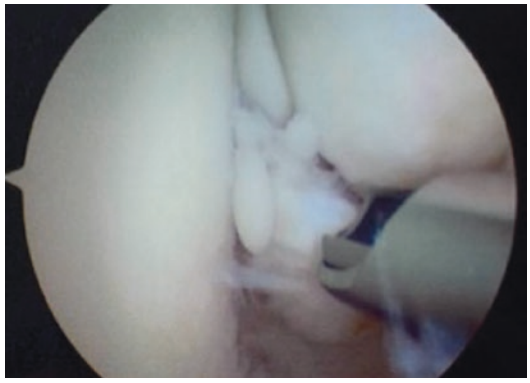

Fig. 6.3 The anteromedial impingement due to hypertrophy of the soft tissue

Recommended in addition to proper clinical evaluation is an MRI. X-rays, in different views, enable the physician to exclude the presence of osteophytes and hence bony impingement. Arthroscopic debridement usually eases pain and restores joint function [7].

6.2.3 Anterior Impingement

This is very common in athletes and is characterized by anterior chronic pain with limitation of dorsiflexion of the foot. Causes include soft tissue and bony impingement [8].

In repetitive microtrauma injuries of the talus against the tibia (e.g., in runners and jumpers), the formation of calcification inside the joint capsule or tibiotalar fibrous bundles is quite frequent (Fig. 6.4). Repeated forced movements of plantarflexion and dorsiflexion of the foot, typical of dancers, lead to painful impingement due to tissue hypertrophy resulting from persistent traction of the anterior joint capsule.

Arthrofibrosis is another cause of anterior soft tissue impingement, which may engage in toto the ankle joint. It may result from surgical treatment or articular fractures. This condition seems to occur in about 73% of patients treated surgically, even in the presence of an anatomical reduction of the fracture with full recovery. Arthrofibrosis is correlated directly with pain and joint dysfunction. The treatment of arthroscopic debridement is the best and least invasive solution with significant symptomatic and functional improvement [7].

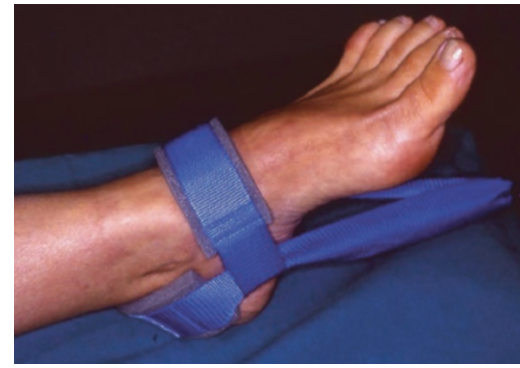

Fig. 6.4 Anterior tibiotalar fibrous bundle

6.2.4 Posterior Impingement

One of the most common causes of posterior impingement is the presence of a bone abnormality such as an os trigonum, a prominence of the posterior tibial profile, or a huge talar queue. The os trigonum, however, is rarely the sole cause of impingement. Frequently, it is associated with the presence of synovitis and hypertrophic tissue due to conflict between the os trigonum and the posterior edge of the tibia.

The condition is related to traumatic events or to repeated microtrauma injuries resulting in excessive plantarflexion of the foot, such as in dancers and football players.

The pain is deeper at the posterior ankle, untreatable by infiltration or physiotherapy, and is exacerbated by plantar hyperflexion movements of the foot with the knee flexed at 90°. Often, the patient experiences sharp pain on palpation along the flexor hallucis longus (FHL) tendon running behind the medial malleolus. Chronic synovitis of this tendon may also cause impingement and can lead to the formation of nodules, palpable with great toe mobilization.

Radiographic exams are performed to check for any bony impingement, but MRI is preferable to obtain a complete view of the soft tissues. The treatment in each case may be arthroscopic with two Achilles paratendineous portals. With arthroscopic surgery, it is possible to remove synovitis, clean out capsular tissue hypertrophy, and release the flexor hallucis longus (FHL) [9].

Very rare causes of posteromedial impingement are muscle abnormalities in the region, e.g., of the accessory muscle flexor digitorum longus, the accessory soleus muscle, the tibiocalcaneal muscle, and the peroneocalcaneus internus muscle [10]. These anatomical variants must be considered in cases of chronic posterior ankle pain even in the absence of trauma. Usually, pain is experienced during exercise and ceases spontaneously with rest. Physiotherapy and infiltrative treatments are ineffective. On examination with the patient in the prone position, it is possible to highlight a posteromedial or posterolateral bulge. The hyperflexion test of the foot with the knee flexed is negative. MRI reveals the presence of these anatomical muscle variations, treated by surgical removal [11].

6.3 Pertinent Imaging

Diagnostic imaging of the painful ankle helps the surgeon to understand if the morbid condition is due to bony impingement, a fracture, or soft tissue impingement. The first step is to perform an X-ray of both ankles and feet with different views under load in the anterior-posterior, lateral-lateral, and anteromedial impingement views (AMIs) to evaluate the structural alterations of the articular tibial and talus profile. In the latter view, the beam is tilted 45° in the cranio-caudal direction with the leg at 30° external rotation and the foot in slight plantarflexion in relation to the standard lateral X-ray position [12].

The second diagnostic step is to carry out an MRI to detect the presence and location of joint effusion, a synovitic reaction all around the ankle. This examination is important to exclude the presence of loose bodies or chondral/osteochondral lesions.

CT mainly defines bone changes such as osteophytes, staging the size of the osteochondral lesions with any cystic areas, and is useful to evaluate the degrees of the degenerative state of the arthritic joint.

6.4 Arthroscopic Procedures

6.4.1 The Anterior Ankle Arthroscopy Procedure

When carrying out anterior ankle arthroscopy, it is very important to consider the patient position and to create a working space that allows the surgeon to work and move around inside the joint in total tranquility.

A historical analysis of the positions described by surgeons shows that distraction of the ankle with a noninvasive system was the most common procedure until the introduction by the Dutch school of a new technique with the foot left free and in dorsiflexion [13–16] (Figs. 6.5 and 6.6). This method has a series of advantages because

forced dorsiflexion increases the working space, allowing larger arthroscope to be used, increasing saline inflow (Fig. 6.7). This enables treatment of the osteophytes present in the anterior part of the tibia and talus, synovectomy, the removal of loose bodies, and treatment of osteochondral lesions [17]. Another advantage is that the introduction of instruments is less likely to cause injury to the neurovascular bundle and cartilage present on the joint surfaces because the talus is concealed from the tibia; this is not possible with joint distraction.

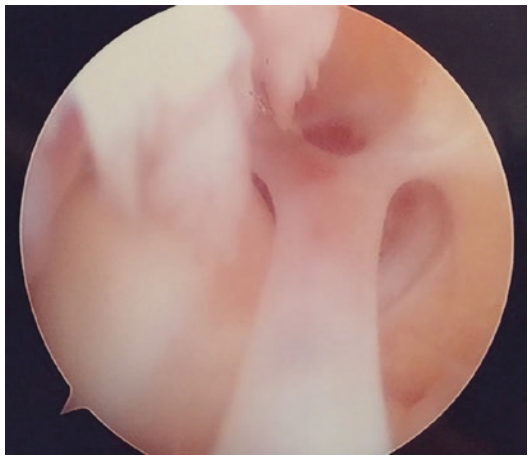

Fig. 6.5 Anterior approach with noninvasive distractor

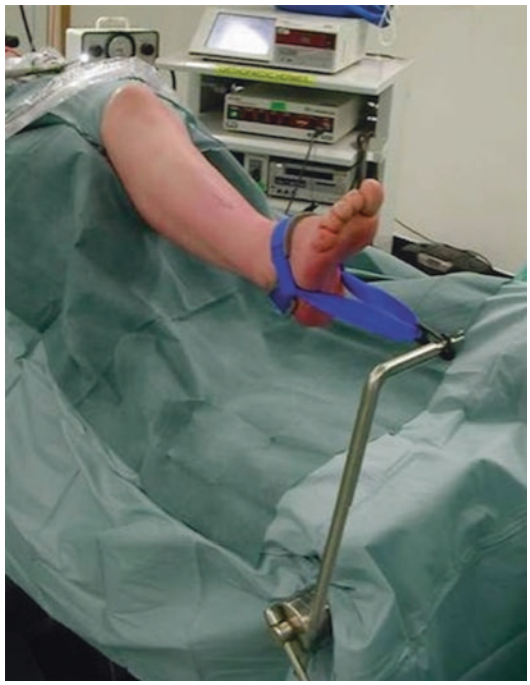

Fig. 6.6 Anterior approach with noninvasive distractor

The surgery is performed in outpatient care (outpatient surgery).

The patient is positioned supine on the table with the heel resting on the edge of the bed. The tourniquet is applied to the ipsilateral leg (250 mmHg). The anesthesia can be epidural or general.

The precise execution of the portals is critical. Before starting, it is always advisable to draw the landmarks on the skin such as the medial and lateral malleolus, the anterior edge of the distal tibia, the peroneus superficial nerve, the anterior tibial artery, and clearly the anteromedial and anterolateral portals.

For joint irrigation, a normal saline solution is used with inflow by a pumping device or gravity. A 4.6 mm arthroscope sheath with a 4.0 mm arthroscope with an inclination of 30° is routinely used for anterior ankle arthroscopy.

The anteromedial portal is created first since it is easy to access with the foot in dorsiflexion. It is located internal to the anterior tibial tendon where there is a soft spot (a depression point that can be palpated). With the foot kept in forced dorsiflexion, a small longitudinal incision is made with a scalpel (11); and subsequently, with a hemostatic mosquito, the subcutaneous tissue is released until the capsule is opened. The second step is to

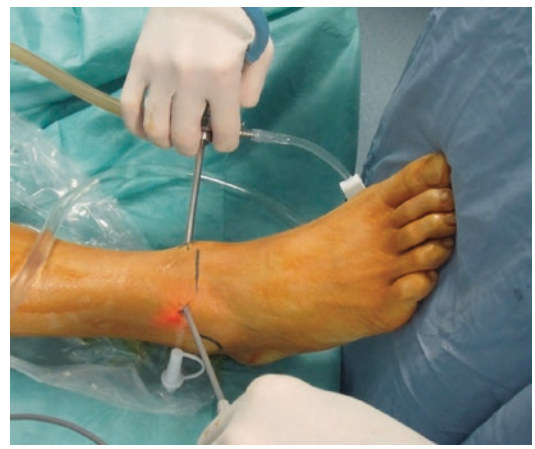

Fig. 6.7 Anterior approach without distraction

introduce the arthroscope shaft with the blunt trocar into the anterior compartment. Then the blunt trocar is removed and the arthroscope introduced.

The position of the second standard portal is located lateral to the common extensor and peroneus tertius tendon on the joint line and is created under direct vision by introducing a spinal needle (22 G) into the joint.

Take care to avoid the lateral superficial peroneal nerve, very close to this portal; it can be viewed by keeping the foot in forced plantarflex and supination [16]. To create the second portal, follow the same steps described for the first.

Sometimes it is necessary to make supplementary portals (anteromedial, anterolateral, and posterolateral) which should always be created with a direct view.

At the end of the procedure, it is important to control the bleeding by electrocautery.

The incisions are closed with 3.0 Ethilon, and a compression dressing is applied.

6.4.2 The Posterior Ankle Endoscopy Procedure

The patient is placed in the prone position with a support under the relevant lower leg. The foot must be left free to move. A tourniquet for hemostasis is placed on the thigh, and anesthesia may be epidural or general (Fig. 6.8).

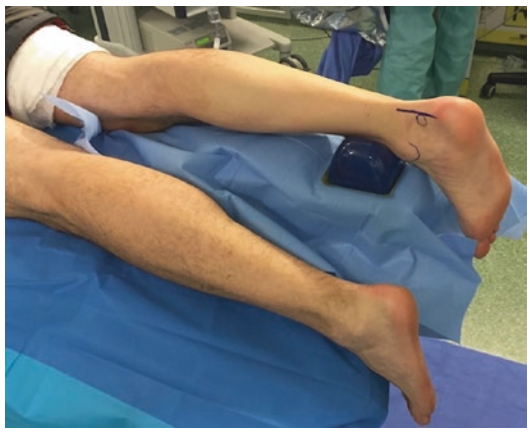

Fig. 6.8 Posterior approach—a tourniquet for hemostasis is placed on the thigh

For irrigation, a normal saline bag with gravity flow is used. The arthroscopic instruments are the same as for the anterior procedure. Alternatively, a 2.7 mm arthroscope, 11 cm in length with a 4.6 mm arthroscope sheath, may be used.

The first step is to mark the anatomical landmarks: Achilles tendon and medial and lateral malleolus. While looking for the two paratendinous portals, the foot should be kept at 90° to the lower leg and a line drawn from the tip of the lateral malleolus to the Achilles tendon, parallel to the sole of the foot. The posterolateral portal (the first) is placed above this line and close to the Achilles tendon, while the posteromedial portal is located precisely on the opposite side (Fig. 6.9).

A small longitudinal incision is made with a scalpel (11), and blunt dissection of the subcutaneous tissue is performed using a hemostatic mosquito. At this point, the arthroscope sheath (diameter, 4.6 mm) with a trocar is introduced into the posterior space of the ankle following the direction of the first intermetatarsal space. Once at the back of the ankle joint, the blunt trocar is removed and the arthroscope introduced. Unlike anterior ankle arthroscopy, visibility is initially greatly reduced by the presence of abundant fatty tissue in the space.

The previously marked posteromedial portal is created with the same scalpel and the soft tissue dissected with the mosquito. The arthroscope

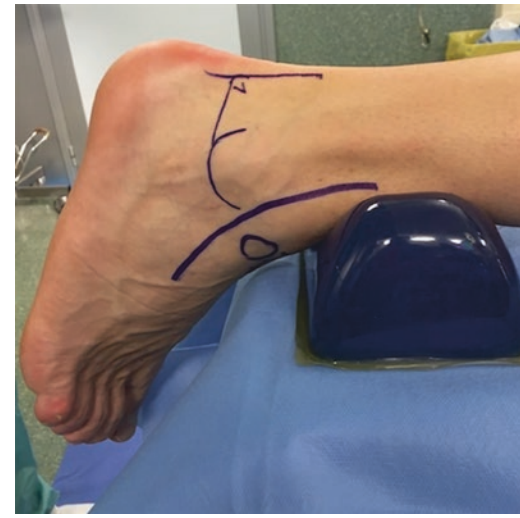

Fig. 6.9 Posterior approach, portals

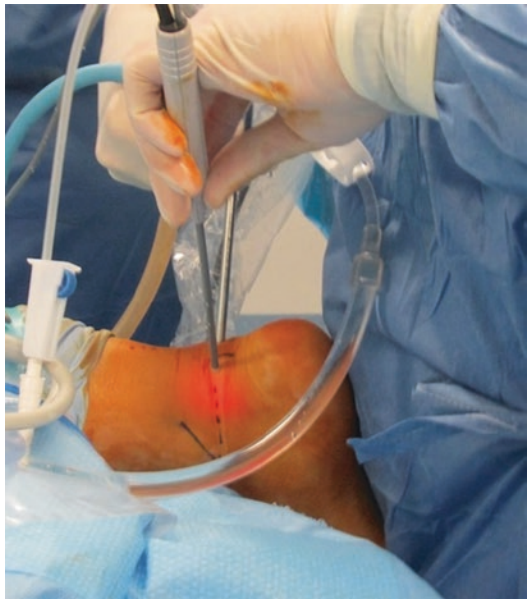

Fig. 6.10 The right position of the arthroscopic instruments

shaft, in the other portal, is used as a guide; and the hemostatic mosquito, at 90° to the shaft, is then made to slide toward the tip until it comes into view. At this point, the mosquito is replaced with a shaver to clean out the soft tissue present all round (Fig. 6.10). The crural fascia and Rouvière ligament are now visible. After removal of the thin joint capsule, the subtalar joint is identified with the posterior talar process. The most important landmark for the surgeon to find out at the beginning of endoscopy is the flexor hallucis longus (FHL) tendon protecting the neurovascular bundle located on the medial side. It is important never to go over the FHL to avoid creating damage to the neurovascular bundle that is around the corner [9].

For the last step, the same procedure as for anterior arthroscopy is followed, controlling bleeding, carrying out sutures, and applying a sterile compression dressing.

6.5 Postoperative Protocol

The patient can usually be discharged the same day as the surgical treatment, without complications.

For anterior and posterior ankle arthroscopy, generally weight-bearing is allowed with the use of crutches for 4/5 days. The sterile dressing is kept for 7 days, and the stiches are removed after a week. Postoperatively, the leg is held high with an ice bag on the ankle for a few hours each day. The patient is immediately able to actively move the ankle to regain the dorsiflexion/plantarflexion.

Physiotherapy is usually started very soon and maintained until the patient returns to the pre-injury or pre-surgery level of functioning.

6.6 Potential Complications

One of the most common complications in anterior ankle arthroscopy is injury to the branch of the superficial peroneal nerve, a sensitive nerve, estimated in 1.9% of cases [18].

Using noninvasive distraction, the risk of nerve damage is directly related to time and the force applied to the ankle.

Another risk is the extravasation of the saline fluid in the subcutaneous tissue during the procedure due to the faulty positioning of the instruments (cannula). The cannula must be positioned properly to prevent compartment syndrome.

DVT is rare. Routine overall prophylaxis is required for patients genetically prone to clotting.

In the treatment of posterior impingement, the highest risk is lesion to the neurovascular bundle on the medial side of the ankle. It can be avoided by following the above recommendations, making proper portals and moving the instruments carefully during endoscopy.

6.7 Pitfalls and Tricks

- Imaging techniques such as MRI and CT are particularly helpful in the diagnosis and treatment of synovitis, loose bodies, and chondral lesions, showing the location and size of the lesion. Preoperative planning is essential.
- Arthroscopic staging is the best way to decide treatment.

- Before starting an anterior or posterior arthroscopic procedure, position the patient correctly (prone or supine).
- Mark the anatomical landmarks. Care and time should be taken to avoid nerve injury and ensure appropriate portal positioning, providing a clear intra-articular view.
- Use dedicated small-joint arthroscopic instruments.
- Place the arthroscope correctly to avoid extravasation of the fluid and compartment syndrome.
- The shaver blade and other arthroscopic instruments must always be in arthroscopic/endoscopic view.
- In hindfoot endoscopy, the posteolateral portal is made first. It is located above the intersection between a horizontal and a vertical line that run the first one parallel to the foot sole, from the tip of the fibula to the Achille's tendon and the second one on the edge of the tendon.
- The FHL tendon is an important landmark to avoid injury to the neurovascular bundle, so remember to stay to the side.

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Part IV

Chondral and Osteochondral Ankle Joint Defects

7 Etiology, Classifications, Mechanism of Action

Alberto Gobbi and Graeme P. Whyte

7.1 Introduction

Osteochondral lesions of the ankle are a significant cause of pain and functional disability, affecting people over a wide range of ages and of all activity levels. There is high variability in reported rates of these lesions, given the frequency of ankle injury in the general population and inconsistent use of imaging modalities that would be expected to have a high sensitivity in identification of such injuries.

The understanding of osteochondral injuries and available treatment modalities developed to address this pathology has expanded greatly since the initial descriptions of these lesions. In 1888, König described mechanisms by which loose bodies could arise in the knee joint, one of which was described as osteochondritis dissecans, which was attributed to a lesion of spontaneous development, without prior trauma [1]. Later, in 1922, Kappis described osteochondral lesions specifically related to the talus [2].

The ankle mortise is a complex anatomic structure that is sensitive to incongruity leading to chondral disruptions. Although lesions may affect the articulating surfaces of the distal tibia or fibula [3], it is talar lesions that are most commonly identified and are the primary focus of diagnostic imaging and treatment. Injury to the osteochondral unit of the talus may lead to progressive chondral damage and dysfunction. Timely diagnosis and accurate characterization of the lesion should be a priority.

7.2 Epidemiology

The true incidence of traumatic and atraumatic osteochondral lesions of the talus has been difficult to accurately determine, given that many cases of pain and dysfunction related to the ankle joint are treated without assessment specifically designed to identify osteochondral damage. Lateral ankle sprains are a frequently described source of chronic ankle pain, and it is estimated that a third of cases continue to be symptomatic more than 2 years after the instability event [4]. The presence of osteochondral pathology should be considered in cases of persistent pain after such an injury, and appropriate diagnostic imaging should be performed to avoid incorrect diagnoses [5, 6]. Without advanced imaging or direct visualization of the articulating surfaces within the ankle, osteochondral lesions may be missed, even in cases of chronic symptomatology that undergo frequent medical assessment.

Komenda et al. described arthroscopic findings from evaluation of 55 ankle joints with chronic lateral ankle instability undergoing

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surgical stabilization. Chondromalacia was identified in 22% of these procedures, while osteochondral lesions were identified in 16% [7]. In patients treated with lateral ankle ligament reconstruction to treat chronic instability, DiGiovanni et al. reported osteochondral injury to the talar dome in 23% of cases [8].

Arthroscopic findings in patients undergoing open reduction and internal fixation of ankle fractures were analyzed by Leontaritis et al. [9]. Chondral lesions were reported in over 70% of cases [9]. Sorrento et al. inspected the lateral talar dome in patients undergoing open reduction and internal fixation of type IV supination-external rotation injuries and reported osteochondral injuries in 38% of cases [10]. No significant difference in likelihood of lateral talar dome injury was identified between lateral malleolar, bimalleolar, and trimalleolar fracture types.

7.3 Etiology

There are a variety of mechanisms by which osteochondral lesions of the talus may arise. Trauma and ischemia are often cited etiologies, although associations have been identified with endocrine disorders, and familial associations suggest a genetic contribution in certain cases. In regard to osteochondritis dissecans of the talus, it is unclear what proportion is related to a previous traumatic injury [11].

Traumatic etiologies have been described a great deal in the literature, and such mechanisms have been confirmed through cadaver studies and with intraoperative findings. It is important to note that attributing the occurrence of prior traumatic events to the development of osteochondral lesions of the talus is often based on patient-reported history, which may be problematic, given the high frequency of reported ankle injuries in the general population.

Roden et al. proposed in 1953 that osteochondritis dissecans lesions that were located laterally on the talar dome had a traumatic etiology, whereas medial lesions were associated with an atraumatic etiology. It was also reported that laterally based lesions were associated with increased symptomatology [12]. In a retrospective review of ankle injuries, Canale et al. reported that all osteochondral lesions located to the lateral talar dome were associated with a traumatic incident [13]. Medial sided lesions were reported to have both traumatic and atraumatic etiologies. Furthermore, it was found that although laterally based lesions were more superficial in nature, there was greater risk of persistent symptoms compared to cases of medially located lesions. Similarly, Alexander et al. and Flick et al. reported that trauma was more likely to be associated with osteochondral lesions located laterally on the talar dome, compared to medially [14, 15]. With respect to location of osteochondral lesions in the sagittal plane, laterally based lesions have been reported to occur more often anteriorly, with medial lesions more frequently located posteriorly on the talar dome [16].

7.4 Classification Systems

A number of classification systems have been developed with the purpose of diagnosing the severity of osteochondral injury to the talus and guiding the management of this condition, although there continues to be a lack of agreement in the literature regarding ideal surgical treatment [17]. These classifications have employed a variety of assessment tools and include staging systems based on plain radiographic imaging, CT imaging, MRI, and arthroscopic findings.

Berndt and Harty [18] originally described a classification based on plain radiographic findings. Osteochondral lesions were categorized into four stages of injury to the talar dome, with stage I described as a compressed lesion and stage IV indicating detachment and displacement of the osteochondral fragment (Table 7.1) [18]. This classification was later modified to include

Table 7.1 Berndt and Harty's staging of osteochondral lesions of the talus based on plain radiographs [18]

Stage I	Compressed lesion
Stage II	Chip avulsion, remains attached
Stage III	Complete detachment, undisplaced
Stage IV	Detached and displaced

a fifth stage by Loomer et al. in 1993 in order to incorporate findings related to radiolucency of osteochondral lesions [19].

Accurate characterization of osteochondral lesions may be difficult on plain radiographs. To better visualize pathology of the subchondral bone and to assess fragmentation and displacement of osteochondral lesions, CT imaging provides great detail. Ferkel and Sgaglione developed a classification based on CT imaging that lists stages of pathology from I to V (Table 7.2) [20]. Stage I describes subchondral cystic changes with intact overlying cartilage, while stage V corresponds to a detached and displaced fragment.

The optimal imaging modality to identify and characterize osteochondral lesions of the talus is widely considered to be MRI. This imaging format allows for detailed evaluation of subchondral bony involvement as well as assessment of the overlying cartilage, although evaluation of edematous changes within bone may lead to an overestimation of lesion size [21]. MRI is particularly useful in early stages of osteochondral injury, where plain radiographs do not yet demonstrate visible abnormality. Hepple et al. developed a classification system in 1999 to stage lesions based on MRI findings (Table 7.3). This method stages lesions based on signal changes consistent with edema of overlying cartilage as well as subchondral bone and includes both lesion displacement and subchondral cyst formation in the classification [22]. Dipaola et al. later described a classification system to stage osteochondral lesions of the talus on MRI that correlated well with findings on arthroscopic examination [23]. Following this, Mintz et al. developed a staging system that focused on MRI findings related to pathologic changes in overlying articular cartilage (Table 7.4) and proposed that this type of evaluation may be useful in surgical decision making [24].

There is debate regarding the usefulness of these staging classifications, particularly when using plain radiography to guide management of osteochondral lesions affecting the talus. Anderson et al. evaluated staging of osteochondral injury to the talus using plain radiographs, CT imaging, and MRI, finding that many lesions were undiagnosed when relying solely on plain radiography [25]. Pritsch et al. found that arthroscopic evaluation of osteochondral lesions did not correlate well with the characterization of these lesions on preoperative plain radiographs and subsequently published a classification system in 1986 based on arthroscopic findings (Table 7.5), also recommending treatment based on this grading system

Table 7.2 Ferkel and Sgaglione's staging of osteochondral lesions of the talus based on CT imaging [20]

Stage I	Cystic lesion within dome of talus, intact roof on all views
Stage IIA	Cystic lesion with communication to talar dome surface
Stage IIB	Open articular surface lesion with overlying non-displaced fragment
Stage III	Non-displaced lesion with lucency
Stage IV	Displaced fragment

Table 7.3 Hepple et al.'s staging of osteochondral lesions of the talus based on MRI [22]

Stage I	Articular cartilage edema
Stage IIA	Cartilage injury with underlying fracture and surrounding bone edema
Stage IIB	Stage IIA without surrounding bone edema
Stage III	Detached but non-displaced fragment
Stage IV	Detached and displaced fragment
Stage V	Subchondral cyst formation

Table 7.4 Mintz et al.'s staging of osteochondral lesions of the talus based on MRI [24]

Stage 0	Normal
Stage 1	Hyperintense cartilage surface, morphologically intact
Stage 2	Fibrillation or fissures not extending to bone
Stage 3	Flap present or bone exposed
Stage 4	Loose non-displaced fragment
Stage 5	Displaced fragment

Table 7.5 Pritsch et al.'s staging of osteochondral lesions of the talus based on arthroscopic findings [26]

Stage I	Intact overlying cartilage
Stage II	Soft overlying cartilage
Stage III	Frayed overlying cartilage

[26]. Analysis of plain radiographic, CT, and MRI findings examined by Ferkel et al. in 2008 demonstrated poor correlation of staging based on all diagnostic imaging modalities and clinical outcomes after arthroscopic treatment. Clinical outcomes did correlate with lesion staging based on arthroscopic findings, and so Ferkel expanded the arthroscopic classification system originally proposed by Pritsch [27].

7.5 Mechanism of Action

The location of osteochondral lesions about the talus has been described in association with specific traumatic mechanisms at length in the literature. To better standardize the localization of osteochondral lesions affecting the talar dome and to analyze the frequency by which areas are affected, Elias et al. created a grid system to categorize the location of pathology. There were nine separate zones described, numbered sequentially from anteromedial to posterolateral. Zone 1 corresponds to a lesion anteromedially, while zone 3 would represent an anterolateral lesion. Zone 7 would indicate a posteromedial lesion and zone 9 a posterolateral lesion. This work was based on analysis of 428 osteochondral lesions on MRI. Contrary to previous reports in the literature of a preponderance of lesions being located laterally on the talar dome, Elias et al. reported that lesions were located medially 62% of the time and laterally 34% of the time. The most frequently affected area was zone 4 (medial/central), which was affected in 53% of cases. This was followed by zone 6 (lateral/central), which was affected 26% of the time. Furthermore, medial sided lesions had increased surface area and depth than those located laterally [28]. Using MRI and following the grid system, Hembree et al. supported these findings that the medial/central area of the talar dome was most frequently affected by osteochondral lesions (54.5%), with the lateral/central zone being the second most frequently involved area (31.2%) [29].

In the work published by Berndt and Harty in 1959, the sequence of traumatic injury was reported to begin with compression of the lateral talar dome against the adjacent fibula as the foot inverts [18]. In 1980, Canale et al. reported that an inversion or inversion-dorsiflexion mechanism was responsible for traumatic laterally based osteochondral lesions of the talus [13]. Interestingly, however, using a cadaver model and pressure transducers, Bruns et al. [30] found that peak pressures were identified at the medial aspect of the talar dome during supination under various conditions of lateral ankle ligament transection. Even prior to ligament disruption, these pressures were measured to be highest medially [30].

Examining cases of ankle fractures treated with open reduction and fixation, arthroscopic findings have demonstrated that those injury mechanisms described as a type IV supination-external rotation and pronation-external rotation were at greatest risk of suffering two or more osteochondral lesions to the talus [9].

7.6 Summary

Osteochondral lesions of the ankle are increasingly being identified as a significant cause of pain and dysfunction related to the ankle joint. A variety of etiologies have been described, although the majority of lesions likely arise secondary to trauma. The clinician should have a high index of suspicion for such lesions when persistent symptoms are reported in order to ensure early diagnosis and treatment. Recent literature suggests that medial osteochondral lesions of the talus may be more common than laterally based lesions. While plain radiographic examination remains an important component of clinical assessment in cases of ankle symptomatology, MRI should be made a routine component of diagnostic imaging to facilitate accurate diagnosis and characterization of osteochondral lesions, when such pathology is suspected. Early diagnosis and accurate staging of osteochondral lesions will assist the treating clinician to determine the appropriate treatment in order to optimize outcomes. New technologies including the use of mesenchymal stem cells and tissue-engineered scaffold showed good results at medium-term follow-up (Fig. 7.1).

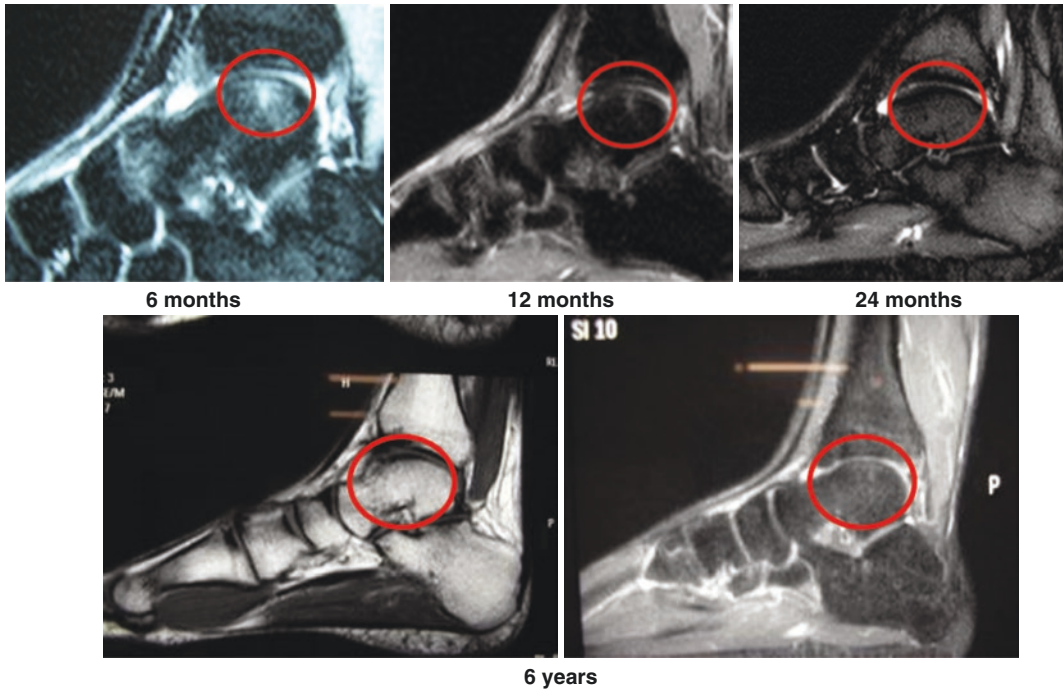

Fig. 7.1 Osteochondral lesion of the talus treated with HA-BMAC technique—result at 6 years

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8 Repair by Microfractures and Perforations

Antonio Zanini, Manuel Bondi, Pierfrancesco Bettinsoli, and Andrea Pizzoli

8.1 Introduction

Microfractures and microperforations are still the most common technique showing the best results for the repair of chondral lesions with diameter less than 15 mm [1].

The micro-traumas, as a result of excessive stress, may lead to the formation of hemarthroses; but, even as a result of synovial hypertrophy, micro-traumas may provoke the formation of fibrous tissue at joint level that can become a rigid structure with cell degeneration, causing fissures on the chondral surface and thinning of the subchondral bone [2].

The synovial fluid, which is often in excess to compensate for the disease, produces pressure on the chondral surface; and it can lead to joint damage with fissures or flaps up to subchondral cysts and chondral fractures. In the latter case, the applied forces can extend the lesion from the cartilage layer to the subchondral bone, and this process occurs frequently [2–4].

There are also non-traumatic causes of chondral lesions: chronic ankle instability, endocrine and metabolic factors, bad joint alignments, idiopathic avascular necrosis, joint degeneration, systemic vascular disease, and genetic predispositions [1, 5–12].

In case of cartilage injury, mainly acute, the formation of a communication between the cartilage and the underlying trabecular bone may occur, allowing a reparative spontaneous response resulting in the lesion filling [13].

8.2 Literature Overview

In 1959, Pridie [14] proposed the concept of therapeutically induced bleeding at subchondral bone level below the articular cartilage damaged regions. The technique involved the removal of unstable cartilage fragments, including the underlying bone, and then the stimulation of the defect healing through the formation of 3–4 cm^2 holes with 1.5–2.0 mm diameter and a depth of 2 cm.

Several studies have shown the efficacy of this technique that is still considered the gold standard in the case of injuries mainly characterized by cartilage damage with minimal involvement of the subchondral bone [15–17].

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In 1982, Johnson [18] modified the chondro-abrasion technique (already proposed by Haggary in 1940) claiming that the abrasion must be performed at the "tide-mark" level and it should not reach the cancellous bone, thus reaching a depth of 1–2 mm.

In 1992, Steadman [19] developed the microfracture technique that consisted in drill holes at a distance of 3–4 mm and a depth of 4 mm on the articular surface of the lesion. This procedure, through perforations of smaller diameter with respect to Pridie's technique (about 0.5–1.0 mm to 1.5–2.0 mm), allowed a minor disturbance to joint biomechanics [19]. This treatment, initially reserved for knee chondropathy, was then extended to the cartilaginous lesions of the ankle joint.

Different recent studies showed good results for these surgical technique [15, 17, 20–22].

8.3 Indications

The arthroscopic surgical procedure of microperforations or microfractures for chondral lesions' repair must consider several variables.

The key issue is to consider whether the etiology of the lesion is due to either an acute injury or multiple traumas. The depth is equally important, also considering the possible presence of subchondral cysts or avascular necrosis zones or bone infarcts. The defect can then be either circumscribed or not with mono- or multipolar sites. The ligamentous integrity of the ankle should also be considered, as well as the alignment (varus or valgus axis deviation) and any previous treatments.

The X-ray imaging is critical in assessing the joint space and the eventual arthritic component (osteophytes or cysts), while the magnetic resonance allows to evaluate the depth of the lesion of the bone bruise and of the avascular necrosis.

We can therefore claim that the main indications for this type of surgery are injuries from 0.5 to 2 cm^2, age of the patients from 15 to 50 years, and the absence of alignment and instability deficits.

The contraindications that we can consider as relevant are overweight, joint stiffness, axial deviation, instability, age according to the clinical case, severe osteoarthritis, inflammatory disease, and rheumatoid arthritis.

8.4 Surgical Technique

Once the clinical and instrumental diagnosis has been implemented and the characteristics of the lesion and its evolution stage have been identified, the choice of the treatment for a chondral defect of the ankle depends on whether it is measured in the acute phase or in the next phase.

In the acute phase, the chondral defect must be fixed by the use of metal or absorbable synthesis devices. In case of small lesions, the chondral defect is compacted. If the injury is chronic and in an accessible location, we proceed to the removal of any free fragment, treating it as a moving body, implementing a revitalizing treatment of the subchondral bone with perforations or microperforations.

Most of the lesions treated with microfractures take advantage of an arthroscopic anterior access with the ankle in full plantar flexion. This access allows the treatment of lesions located from the middle third ahead of the talar dome and also of posterior lesions partially accessible, thanks to new devices [23].

Surgical treatment with multiple perforations proposed by Ferkel via transtalar and transmalleolar aims at making the perforations, with the aid of a compass, in hardly accessible areas or areas not reachable arthroscopically (Fig. 8.1a, b). To the articular chondro-abrasion, executable through the classic arthroscopic access, are added the perforations performed with motorized tool; but this is a method in disuse because it can pierce the tibia or the talus.

Compared to procedures involving a mini-open creation, arthroscopy shows less invasiveness and a lower morbidity; moreover, it offers an advantage in terms of early rehabilitation and a quicker return to work and recreational activities.

For this type of surgery, which we normally execute without stretch of the ankle joint, sometimes it is necessary to apply a traction sling that allows the articulation diastasis in order to enable a more simple transition of the tools, especially in central and posterior zones. In cases of

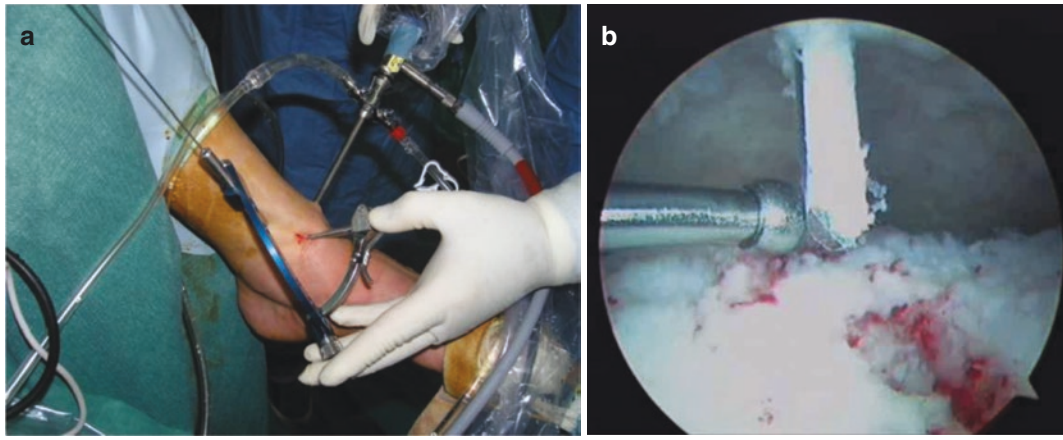

Fig. 8.1 (**a**) Use of the Ferkel compass for talar perforations. (**b**) Perforations through Ferkel compass via transtibial and transmalleolar

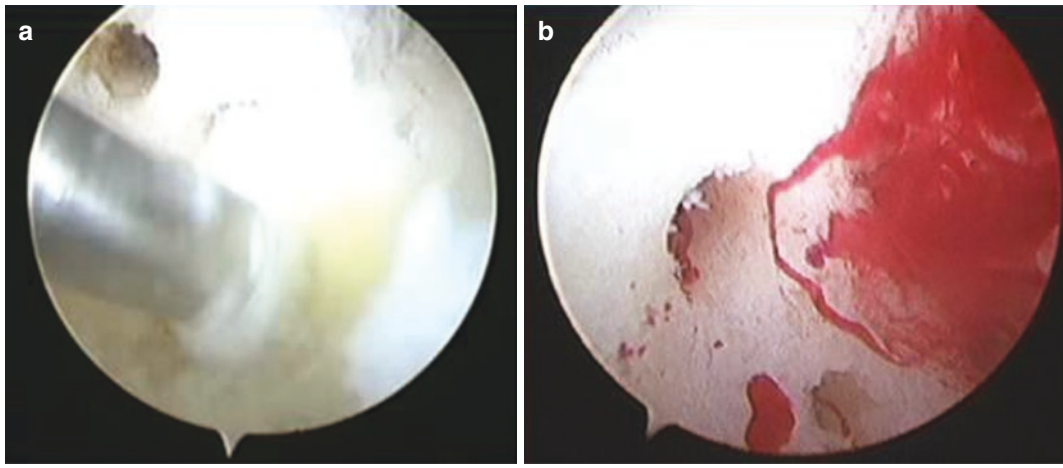

Fig. 8.2 (**a**) Subchondral bone perforations with Kirschner wire. (**b**) Subchondral bone fracture layer after perforations with a K-wire

clamped joints, the use of the external fixator to allow arthrodiastasis can be decisive.

The subchondral bone can be perforated with the aid of a milling cutter or with a Kirschner wire of 1.5–2 mm diameter (Fig. 8.2a, b) [24, 25].

The microfracture technique, proposed by Steadman [26], has been improved with the use of special perforators that allow greater accessibility to the articular environment, thanks to the extremity curve following the shape required by the articular anatomy (Fig. 8.3a, b) [27].

One possible complication of this procedure occurs at the time of instruments' removal due to the creation of free endoarticular bone fragments that, if they are not removed, can cause a future articular block or cartilaginous damage for loose bodies [28].

The use of a microperforation osteochondral drill (microOCD) has been recently introduced. The main difference between the microfractures and microOCD is determined by the fact that the microfractures, presenting a spinky conformation, cause a chondro-compaction in the deep section which can prevent or reduce the blood flow due to the chondral wall developed in front of the hole that acts as a stopper. In the most superficial portion of the cone, a good cancellous bone is developed, but we do not know if it is

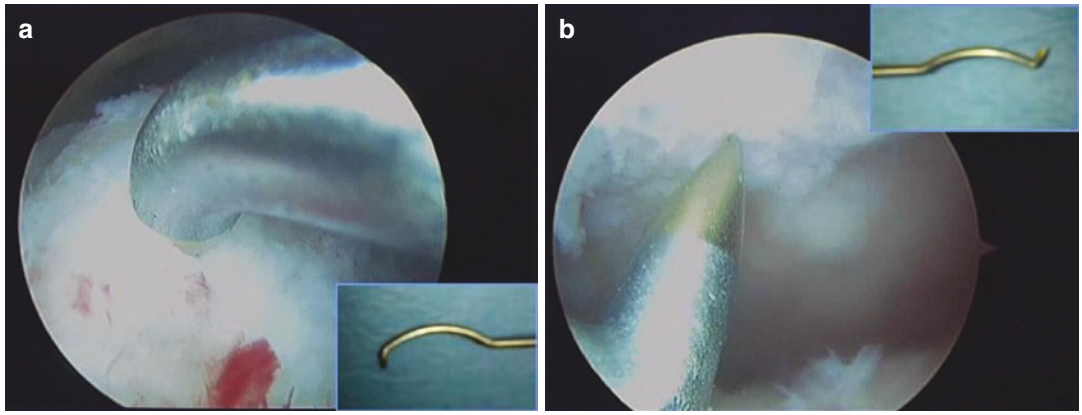

Fig. 8.3 (**a**) New drills that allow better accessibility to articular talus environment. (**b**) New drills that allow better accessibility to articular tibial environment

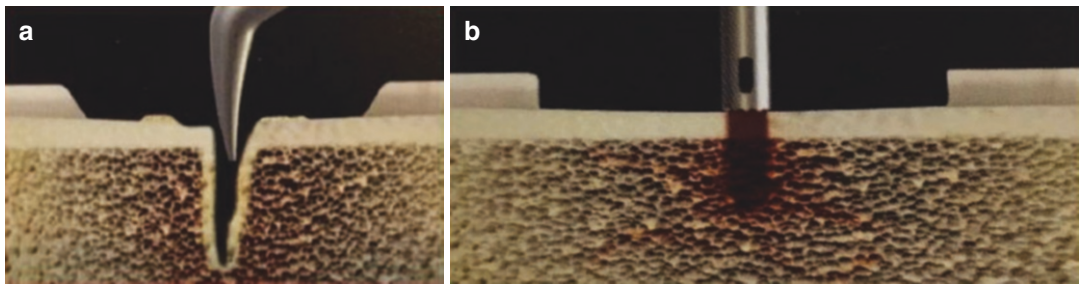

Fig. 8.4 (**a**) The microfractures, with a spiky conformation, cause a chondro-compaction that can prevent or reduce the blood flow due to the chondral wall developed in front of the hole. (**b**) The microfracture perforates the subchondral bone with the same diameter throughout its length causing a less compaction of the bone

well vascularized. The area affected by the lesion is often accompanied by edema; thus, the vascularization may be sufficient but not abundant due also to the low depth.

The microperforations, however, do not shrink the bone (Fig. 8.4a, b); but they perforate with the same diameter throughout its entire length. This technique allows to reach a greater depth, and it induces the bleeding from all points of the perforations, but it remains the negative element represented by the rotation speed of the perforating tip.

After creating a stable cartilaginous wall around the lesion with the specific curettage (Fig. 8.5), we proceed with a drill on the perforations to be performed at various points with the angle as close as possible to 90°. Moreover, in order to slide the flexible drill with 4.6 mm diameter, it is necessary to use rigid cannulated guides with different inclination angles. The depth of the hole can vary from 4 to 7 mm (or more) and is controlled by the guides characterized by millimeter-graduated caps that are inserted directly on the handgrip of the guides (Fig. 8.6a, b).

The perforation with the drill remains a limit of this technique because it may necrotize the tissue. But the small diameter of the tip and the chance of modulating the speed can reduce this possibility. In addition, another limit to be considered is the movement in a tight joint such as the ankle, which is possible only in anterior portions.

Unlike the knee, in which the articular range ensures an easier access to the chondral lesions and allows also an excellent treatment of patellar injuries that are difficult to treat with other methods, the narrowness of the ankle joint does not always allow to reach

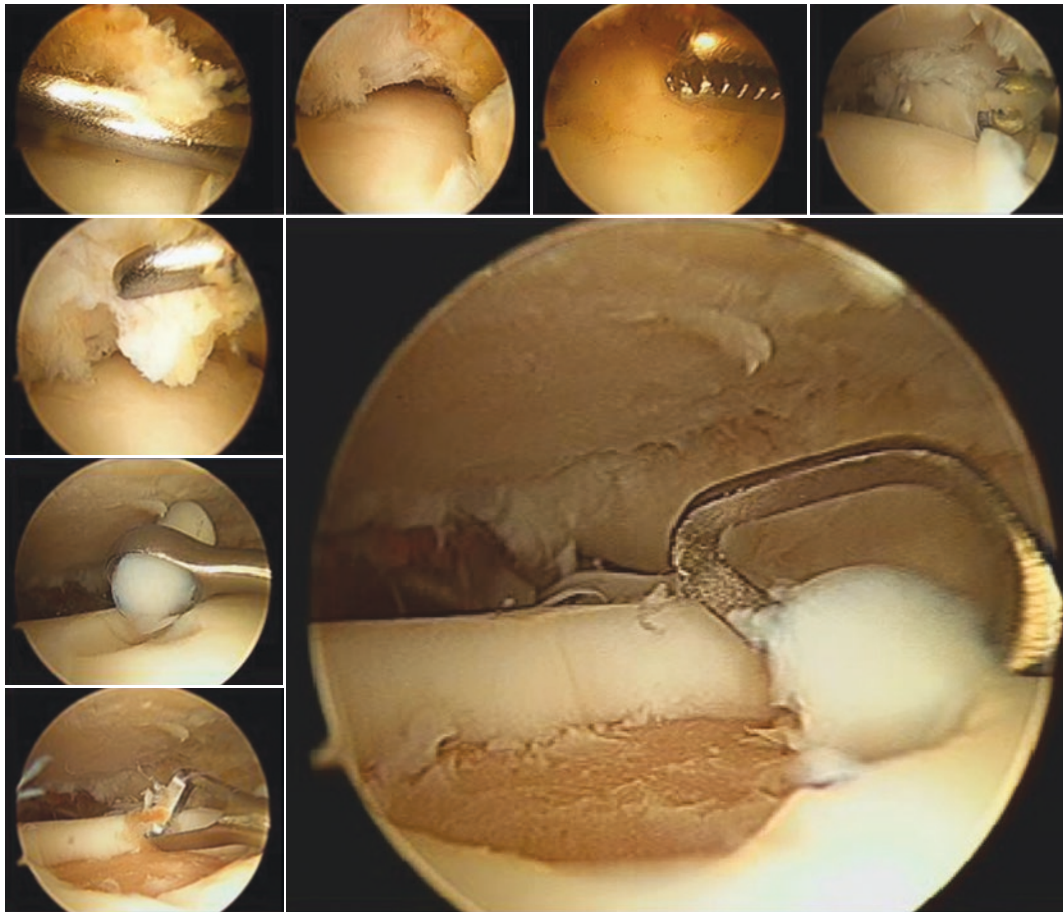

Fig. 8.5 Preparation of the articular surface for the microfractures

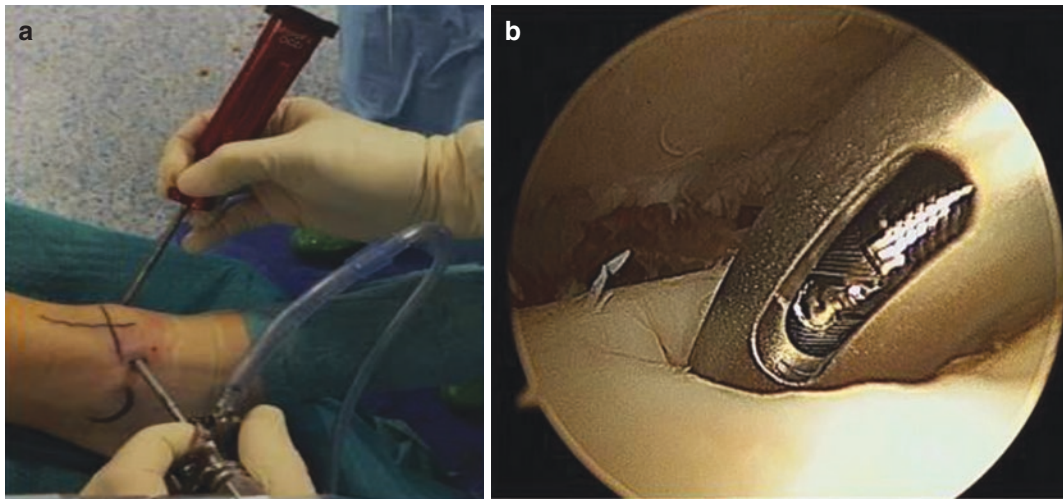

Fig. 8.6 (**a**) Use of microfractures during ankle arthroscopy. (**b**) The microfractures have graduated caps directly assembled on the handgrip of the guide allowing the choice of the depth to be reached by the perforation

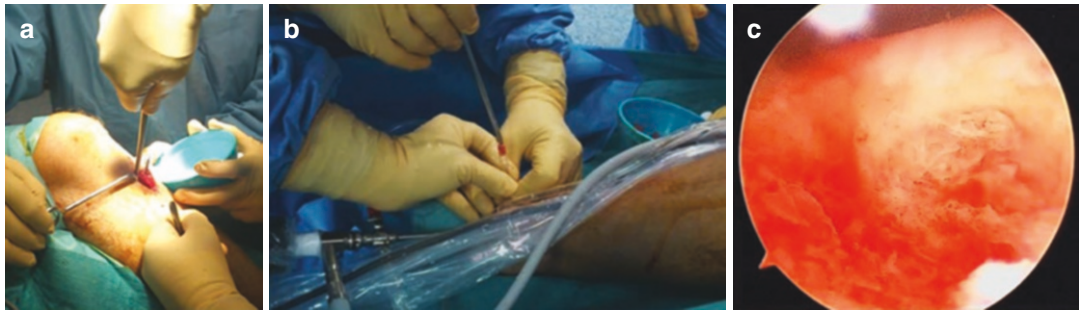

Fig. 8.7 (**a**) Extraction of cancellous bone for the treatment of an osseous cyst. (**b**) Application of cancellous bone through the cannula of the spinal needle. (**c**) Filling of the talar cysts through the cancellous bone taken from the proximal ipsilateral tibia

all the pathological localizations and perforate them with perpendicular angle. Indeed, in some cases, we perform a mixed treatment consisting in microfractures and microperforations that may be, if confirmed by the follow-up, a surgical solution for access to all the areas affected by the disease. The achievement of these areas of the posterior talar dome is difficult even with the use of a medium-angle cannula, and often the use of a straight cannula is required even if it does not ensure the perpendicularity on the articular surface.

The use of PRP, after performing microperforations, favors the development of semicartilaginous tissue allowing a reduction of the patient's symptomatology.

This technique concerns small chondral lesions with little bone involvement.

For large chondral defects at subchondral bone depth, after performing microfractures, we prefer to fill the bone gap with cancellous bone taken previously from the proximal third of the ipsilateral tibia through a small skin incision (Fig. 8.7a–c).

We perform an incision of about 2–3 cm in the proximal third of the leg; after achieving the osseous plane, using a saw and small chisels, we create an access cortex that allows us to reach the underlying cancellous bone, which is taken through a serrated spoon.

The bone removal is introduced into the lesion with the aid of a small cannula (often we use the covering plastic of the spinal needles); then it is pressed with the blunted trocar of the arthroscope. At the end, we complete the procedure with a spatula to level and control the stability of the cancellous bone. The same procedure is also performed on the knee pathologies.

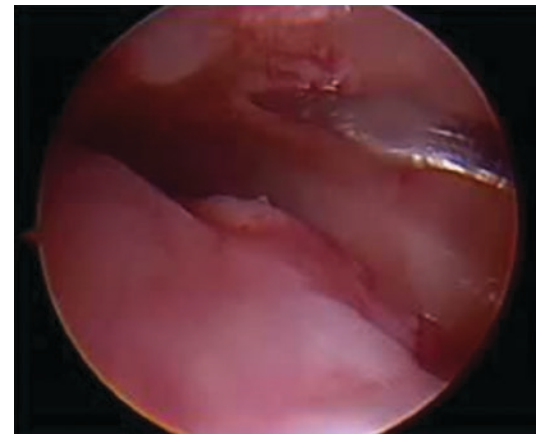

Fig. 8.8 Application of the bio-collagen in semi-liquid structure with high biocompatibility and ability to be colonized [33]

The bone graft may be coated with swine origin biomaterials (ACIC) that allow, through their matrix structure, to stabilize and maintain the blood cells in situ (Fig. 8.8). These blood cells are released through the microfractures, and they favor the cartilaginous regeneration where it is necessary. New liquid matrices allowed to use this technique arthroscopically, because the injected biological collagen polymerizes in the site where it is positioned [29–31], allowing an immediate rehabilitation.

In a recent publication [29], the cytocompatibility of a bio-collagen (CartiFill) and its ability to be colonized by cells stimulated by the microfractures was analyzed. The study showed a significant decrease in pain and a good increase of the AOFAS score after 6 months.

Actually, there has been a shift from an average AOFAS score of 53.8–86 at follow-up. The same VAS decreased from 6.6 to 1.6. The majority of patients of the study returned to perform athletic activity 6 months after surgery. It therefore appears to be a one-step functional and less expensive (hospitalization, surgery, and biomaterial) method with respect to the two-step procedures, which are still used nowadays [1, 32].

All the lesions offer a repair of the defect with fibrocartilage by covering the talar gap in direct proportion to its size: the smaller the defect, the better the clinical and radiographic results.

Görmeli et al. [33] have analyzed 40 patients who took part of the microfracture procedure; and, during the same surgical procedure, 13 patients have received a PRP infiltration, 14 patients a hyaluronic acid infiltration, and 13 patients a physiological infiltration. The authors reported a better AOFAS score at follow-up for patients treated with microfractures and PRP, suggesting that this method is better compared to the use of hyaluronic acid, also due to the well-known analgesic effect in the early stages of rehabilitation.

8.5 Rehabilitation

Our experience allowed us to decrease the discharge times to which the patient has to undergo after surgery. In fact, we reduce the time proposed by Steadman (10–12 weeks) [19] allowing the patient partial weight-bearing after 4–6 weeks and gradually increasing in the last 2 weeks. During the postoperative period, protection of the axial load and the tangential forces must be ensured; moreover, the pain and inflammation must be controlled, and it is needed to regain the movement and the muscle strength.

Starting from the 4th–5th postoperative day, the passive motion of the ankle in flexion-extension that it is gradually increased according to the patient's tolerance during the first 15–20 days begins.

The patient starts to ambulate with the aid of Canadian canes with no weight-bearing on the operated limb for 3 weeks, and then the patient begins the rehabilitation in water always in discharge.

The functional and antalgic results have been more than encouraging, thanks to the rehabilitation in water and to the stabilometric platform Delos, started from the eighth week [34–36].

The clinical data expect a good functional recovery ensuring a marked improvement in operated patients, but it is evident that the perfect filling and coverage of a lesion can be independent from the disappearance of pain or from the functional recovery.

8.6 Considerations

According to the literature, the combination of debridement and medullary stimulation probably represents the best available and less expensive treatment to treat cartilaginous injuries of the ankle [36–38].

In orthopedic language with the term "debridement," we mean the cleaning of the damaged cartilaginous surface.

Microperforations and microfractures are the first step in the treatment of symptomatic osteochondral lesions inferior to 1.5 cm^2; in fact, these are too small dimensions to be considered for a stabilization of the fragment [39, 40]. The advantage of this method is the execution of the procedure through a mini-invasive approach, without needing dedicated tools that would increase the costs, resulting in limited iatrogenic tissue damage. In the case of a deep localized defect exceeding 15 mm in diameter, it is advisable to associate perforations to the application of a cancellous bone graft, positioned into the defect site after a proper cleaning of the bone bed [41].

Nasaka and colleagues [42] have successfully used the method of microfractures even in rheumatoid patients, thus extending the surgical indications.

We believe that the stimulation of fibrocartilage with microfractures is an unpredictable biological reaction and that it is not possible nowadays to know how the injury will react to the treatment, also due to the different thickness of the cartilaginous talus dome and to the poor vascularization. Certainly, the neoformed fibrocartilage will have a limited duration inducing a progressive reduction of the effectiveness of the treatment.

8.7 Conclusions

Despite the relative technical difficulty of the surgical technique of the cartilaginous mantle recovery, arthroscopy is a valuable method to achieve the repair of articular cartilage, with all the advantages that a mini-invasive method can offer compared to an alternative open surgery.

The important consideration is that the neoformed fibrocartilage after microfracture and nano- or microperforations is partly biomechanically valid, but it may face a subsequent deterioration, reducing the effectiveness of the procedure over time.

It is essential to explain to the patient that he will not get the healing of his articulation with this arthroscopic treatment, but a reduction of the symptomatology with an operational restoration. In fact, the certain success of this surgery consists in the length of the patient's benefit.

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9 Restoration by Autologous Osteochondral Transplantation

Yoshiharu Shimozono and John G. Kennedy

9.1 Introduction

Operative treatment for osteochondral lesions of the talus (OLTs) can be divided into two broad categories: reparative procedures, including bone marrow stimulation (BMS), and replacement procedures, including autologous osteochondral transplantation (AOT). Despite successful outcomes following BMS for OLT with small lesions in the short to medium term, deterioration of fibrous cartilage repair tissue is inevitable over time, with clinical outcomes deteriorating in the same way. Conversely, AOT is a replacement technique that substitutes the lesions with viable hyaline cartilage and subchondral bone by insertion of a cylindrical autologous osteochondral graft. The goal of this procedure is to reproduce the similar mechanical, structural, and biomechanical properties of native hyaline cartilage.

9.2 Indications

Current indication to either proceed with BMS or AOT is primarily based on the lesion size, but a definitive indication between BMS and AOT is still controversial. It is traditionally considered that lesion size greater than 150 mm^2 in area or 15 mm in diameter should be treated with AOT and smaller lesions are treated with BMS [1, 2]. However, a recent systematic review endorsed by the International Congress on Cartilage Repair of the Ankle (ICCRA) demonstrates that smaller lesions >100 mm^2 or 10 mm are regarded as the optimal indication size [3]. In addition, AOT is indicated in the treatment of OLTs that have large cystic subchondral defects or that have failed previous BMS [4, 5].

Contraindications include rheumatoid disease, infections, or posttraumatic ankle osteoarthritis with grade II or above. Ankle malalignment should be corrected before AOT. A relative contraindication is a case of preexisting patella femoral joint arthritis, which may deteriorate by graft harvesting.

9.3 Preoperative Preparation/ Positioning

Specially designed surgical instruments for AOT procedure, including a recipient sizer, recipient harvester, donor harvester, and tamp, are necessary. While several instruments are commercially available, the authors prefer to utilize the Osteochondral Autograft Transplant System (OATS; Arthrex, Naples, FL). The utilized core sizes are 6, 8, and 10 mm. Standard surgical instruments for the tibial osteotomy and exposure of the ankle joint are

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required. Kirschner wires (K-wires), a cannulated drill set and screws (3.5/4.0 mm), and fluoroscopy are used for fixation of osteotomy.

Under general or spinal anesthesia, the patient is placed in the supine position. The authors prefer to use a thigh tourniquet to get clear visualization in a bloodless field.

9.4 Operative Technique

The surgical approach is determined by the location of the OLT. If the lesion is located in the anterior aspect of the talar dome, an arthroscopic technique or anterior arthrotomy can be applied. However, the central and posterior lesions require an osteotomy to expose the defect because a perpendicular approach is critical for graft implantation.

9.4.1 Tibial Osteotomy

For medial talar lesions, a medial malleolar osteotomy is usually required because medial lesions are commonly located in central or posterior position. A longitudinal skin incision is made over the medial malleolus. The posterior tibial tendon is identified and retracted with extra care to avoid neurovascular damage. A provisional K-wire is inserted to determine a precise osteotomy line under fluoroscopy (Fig. 9.1). After the decision of the osteotomy line is completed, two parallel fixation holes are predrilled in the medial malleolus for later anatomic screw fixation. An osteotomy is performed using an oscillating saw and osteotome. The authors prefer to utilize a chevron-type osteotomy to provide stability after fixation, larger surface area for healing, and greater exposure. At the level of the subchondral bone, the saw is stopped; and the final cut at the articular cartilage is completed with a sharp osteotome, which minimizes the articular cartilage damages. The osteomized malleolus is retracted in the plantar direction on the deltoid ligament hinge, and adequate visualization of the medial aspect of the talar dome is achieved (Fig. 9.2).

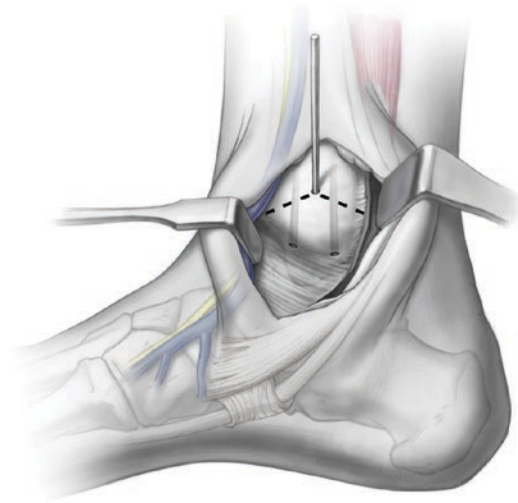

Fig. 9.1 A provisional Kirschner wire is placed to visualize the osteotomy site fluoroscopically. A chevron-type osteotomy is made in the medial malleolus

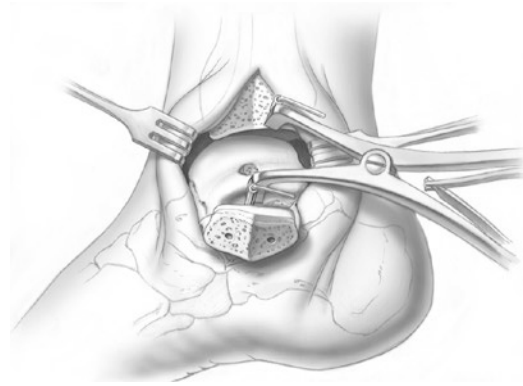

Fig. 9.2 Plantar displacement of osteotomized medial malleolus

As lateral lesions are commonly located in the anterior aspect of the talar dome, anterolateral arthrotomy with plantarflexion of the ankle joint can expose the anterolateral lesions sufficiently without an osteotomy. However, in cases of central or posterior lateral lesions, a tibial or fibular osteotomy can be required. Centrolateral and posterolateral lesions can be accessed with an anterolateral tibial trapezoid osteotomy (Fig. 9.3) [5]. The advantage of this osteotomy over the fibula osteotomy is to avoid the lateral ligament reconstruction which is required after fibular osteotomy. Similar to the medial osteotomy, the

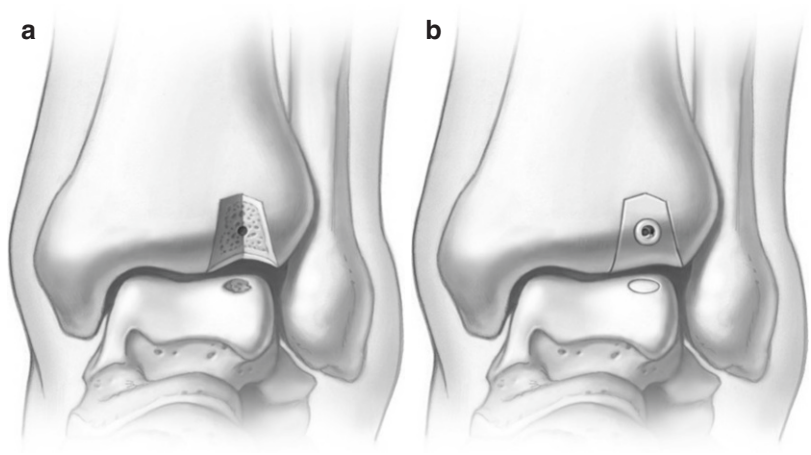

Fig. 9.3 Anterolateral tibial osteotomy (**a**) and fixation of osteotomy (**b**)

site of proposed osteotomy should be predrilled for subsequent fixation with a 4 mm titanium screw. In cases requiring a fibular osteotomy, anterior talofibular ligament or/and calcaneofibular ligament release and subsequent reconstruction are typically required.

9.4.2 Preparation of the Recipient Site for the Insertion of the Osteochondral Graft

Debridement of the damaged unstable cartilage is first performed using a curette and a rongeur. The defect should be surrounded by a stable and viable cartilage rim. The lesion size is measured, and the required graft size and number are determined with a recipient sizer. The appropriate-sized trephine is placed into the talus defect, and the lesion is removed to 10 mm depth. To reconstruct a smooth articular surface, it is crucial to maintain the recipient-harvested trephine perpendicular to the lesion. If necessary, eversion or inversion may improve access. The base of the graft recipient site is then overdrilled by further 1–2 mm with an acorn-shaped drill tip, yielding a graft depth of 11–12 mm. The graft should be shaped in a bullet fashion to fit with the recipient site. This provides the graft stability and sustains articular congruity [6]. Then a 0.045-in. K-wire is used to create multiple small holes in the walls of the recipient site to facilitate the graft and native host tissue integration.

If the lesion requires two grafts, a figure-eight "nested" technique where two cylindrical grafts are placed side by side producing a figure-eight configuration is performed (Fig. 9.4) [5]. This minimizes the empty space that would be filled with fibrocartilage.

9.4.3 Harvesting Osteochondral Graft from the Ipsilateral Femoral Condyle

Donor osteochondral graft plugs are most commonly harvested from the ipsilateral knee. The authors prefer the non-weight-bearing portion of the lateral femoral condyle. This area provides topographic variations that match the articular surface of the talar dome. A mini-open arthrotomy with a lateral parapatellar approach is used. Once the appropriate site and orientation of the graft which match the talar defect as closely as possible are determined, the donor trephine is placed perpendicular to the chondral surface, and the orientation is maintained during harvesting. The donor trephine size should be 1 mm larger than the recipient trephine size for a precise press fit.

After osteochondral graft harvesting, the graft is measured and trimmed in a bullet shape to correspond to the depth of the recipient site using a rongeur. The authors prefer to soak the graft in concentrated bone marrow aspirate (CBMA) or platelet-rich plasma (PRP) at this time in order to

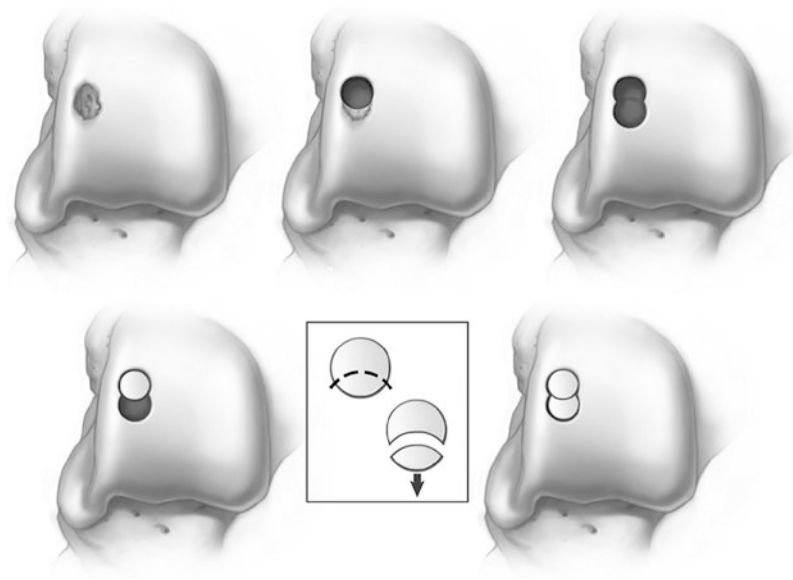

Fig. 9.4 Figure-eight "nested" technique. The two plugs are contoured to each other

potentially improve integration between the graft and native cartilage.

The knee donor site is backfilled with a synthetic bone plug(s) or allograft to prevent hemarthrosis, which may inhibit obviating postoperative scar formation. It also promotes fibrocartilage and bone infill in the donor site. The authors used the OBI TruFit plug (Smith & Nephew, Andover, MA) but currently use allograft because OBI TruFit plug was removed from the market in the United States.

9.4.4 Insertion of Osteochondral Graft into the Recipient Site

Prior to implantation, 1.0 ml of CBMA or PRP is injected into the recipient site. The osteochondral graft plug is then transferred to the recipient site. At this stage, the graft congruency is of paramount importance because even if there is a mere 1.0 mm of graft protrusion above the level of native host cartilage, the contact pressure on the graft surface is increased by almost sevenfold [7]. In order to achieve a flush graft surface, the highest point of the graft is marked with a surgical marking pen as well as the highest point of the surrounding talar cartilage, and then the graft is implanted in the defect gently with a mosquito snap so that both marks are in line (Fig. 9.5). When the graft is in acceptable alignment, the graft is gently tapped into a final appropriate position (Fig. 9.6). Care should be taken not to repeat tapping too much or with great force as these affect the cell viability of the graft [8]. If the graft is sunk, a K-wire is inserted into the graft and used to elevate like a joy stick.

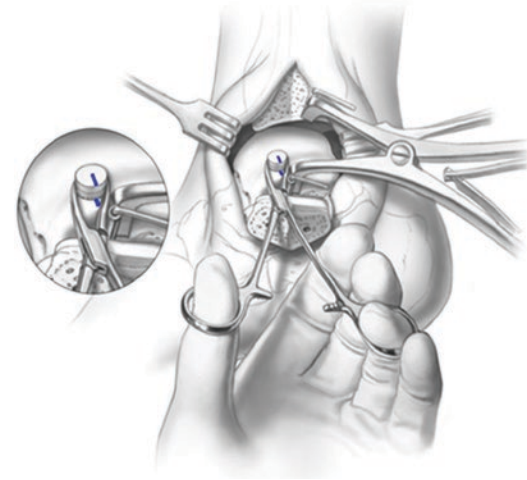

Fig. 9.5 Osteochondral graft is implanted into the created recipient site so that the marks are in line using a mosquito snap

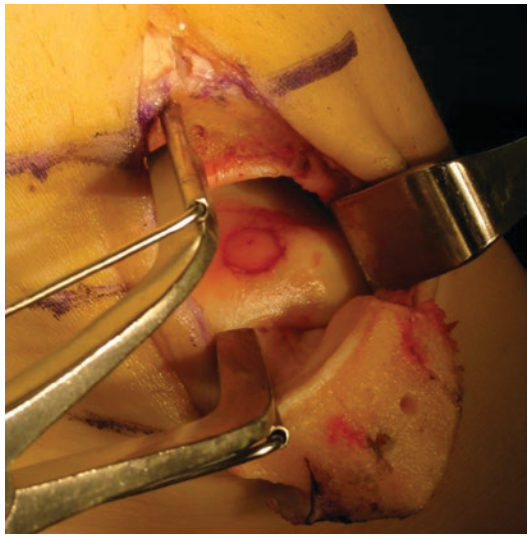

Fig. 9.6 Final graft position with flush graft surface

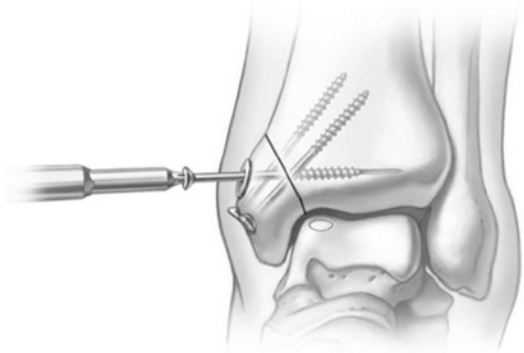

Fig. 9.7 Medial malleolar fixation with two partially threaded cancellous screws and a transverse screw

9.4.5 Fixation of Osteotomy Fragment

The medial malleolus is reduced to the anatomical position and fixed using the predrilled holes as a guide with partially threaded cancellous screws. As the use of only these two screws has the potential to allow the medial malleolus to migrate superiorly, therefore, a third transverse screw with a washer should be placed to maintain anatomical fixation (Fig. 9.7).

9.5 Postoperative Management

The ankle is immobilized in a postoperative splint for 2 weeks. At 2 weeks postoperatively, the splint is switched to CAM Walker boot with continued non-weight-bearing. At this point, the patient is encouraged to start range of motion exercises of the ankle to promote cartilage metabolism and prevent excessive cicatrization in the anterior ankle joint. At 4 weeks postoperatively, the patient is allowed to put down 10% of their body weight and increase by 10% each day. Full weight-bearing is achieved at approximately 6 weeks after surgery. If there are sufficient signs of healing at the osteotomy site, the patient begins a physical therapy program, including balancing and proprioception training. Normal daily activity can be achieved at 8–10 weeks. At 10 weeks postoperatively, sports-specific physical therapy is commenced. From 3 months, the patient may begin to return to sports activity. It usually takes 5–6 months to return to high-demand competitive sports activity.

9.6 Complications

Knee pain after AOT is a concerning possible complication. While the rates of donor site morbidity vary from several studies, most recent studies with a large series of cases demonstrate less than 5% incidence with good functional outcomes [9]. This is in contrast to earlier studies that showed an increased incidence of knee pain in small case series. Donor site knee pain does not appear to be an apparent cause for concern particularly in facilities in which more than one or two of these cases are performed per annum. The key to prevent patella femoral maltracking is to avoid overtightening the lateral retinaculum when repairing the arthrotomy incision. Bone cyst formation after AOT is another concern and has been reported in up to 75% of patients. However, the clinical influence of cyst around grafts was not found to be significant in short- to mid-term follow-up, and most cysts resolve

over time [10]. To prevent this, the authors utilize CBMA to facilitate the integration at the graft and host interface.

Other minor complications include mal-/nonunion at the osteotomy site and soft tissue impingement in the anterior aspect of the ankle joint. A chevron-type osteotomy can reduce the potential risk of mal-/nonunion more than a straight-cut osteotomy. A retrospective case series study showed that only 5% of patients had pain at the osteotomy site and almost all had satisfactory healing and fixation with fibrous cartilage tissue evident on MRI evaluation [11]. To prevent excessive cicatrization around the ankle joint, range of motion exercises should be started at 2 weeks postoperatively.

9.7 Outcomes

Approximately 85% of patients have good to excellent clinical outcomes in short- and mid-term follow-up, and the results do not appear to deteriorate over time [4, 5]. A retrospective case series of 85 patients who underwent AOT demonstrated that the mean Foot and Ankle Outcome Score (FAOS) was improved from 50 to 81 at mean 47 months follow-up and the mean magnetic resonance observation of cartilage repair tissue (MOCART) score was 86 at mean 25 months follow-up [12]. In the athletic population, a recent study reported that American Orthopaedic Foot and Ankle Society (AOFAS) scores were improved to 89 at 24 months follow-up and 90% of athletes successfully resumed pre-injury sports activity [13].

9.8 Summary and Conclusions

AOT replaces the osteochondral lesions with hyaline cartilage and subchondral bone. The technique is indicated for large lesions (>100 mm^2 or >10 mm) or cystic lesions and provides good to excellent clinical outcomes in short- to mid-term follow-up. There are several tips and pearls when performing AOT. Adequate exposure of OLT through a malleolar osteotomy for a perpendicular access to the lesions is the key to achieving a successful procedure. The graft site and the end of the graft should be shaped in a bullet fashion to maintain graft congruency, and it is crucial that the implantation of the graft should be flush with surrounding native host cartilage. Donor site morbidity from the ipsilateral knee is a potential complication, but it does not appear to be a significant issue. Most symptoms resolve within 1 year.

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10 The Use of Aci/Maci to Restore Osteochondral Defects in the Ankle

Sean Haloman and Richard Ferkel

10.1 Introduction

Cartilage injuries of the talus can be challenging to treat. In the USA, 250,000–300,000 patients with symptomatic cartilage injuries undergo surgical treatment, most commonly in the knee. Though the majority of ankle osteochondral lesions can be managed with debridement, microfracture, and/or drilling with good results, many, especially those >150 mm^2, have poorer outcomes [1–3]. In fact, that number may be even smaller as Raponi et al., reported in their recent systematic review. They found that lesions of 107.4 mm^2 might be the maximum size for drilling or microfracture for good outcomes [4]. For larger lesions, there are other treatment options available. These include osteochondral allografts, osteochondral autografts (single plug or mosaicplasty), autologous chondrocyte implantation (ACI), juvenile allografts, micronized cartilage matrix, and resurfacing procedures (Fig. 10.1). Factors influencing choice of surgical procedure include: size of

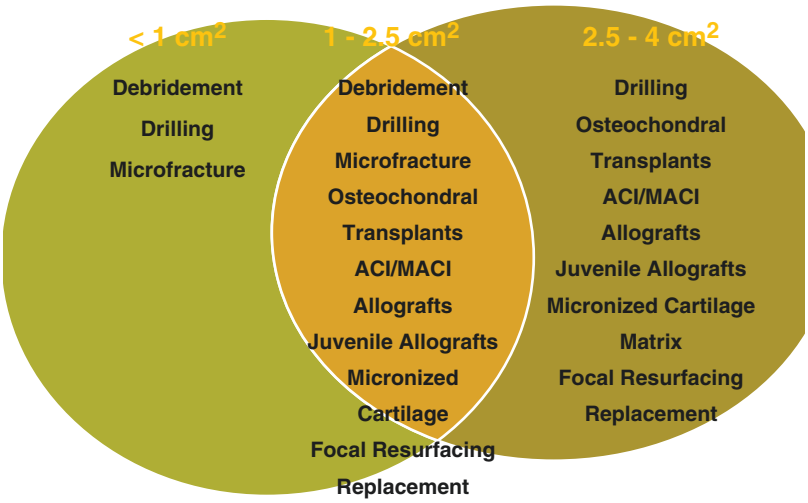

Fig. 10.1 OLT treatment options based on size of the lesion

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lesion, location of lesion, containment, associated subchondral cyst, status of cartilage cap, associated pathology, and patient preference.

Autologous chondrocyte implantation (ACI) was developed almost 20 years ago as an option to restore native hyaline cartilage. The process involves harvesting autologous cartilage tissue from either the non-weight-bearing portion of the knee, the non-weight-bearing posteromedial portion of the talus, or from the cartilage lesion [5]. This tissue is then sent to a laboratory where the chondrocytes are isolated and grown. Those cells are either implanted under a periosteal patch (ACI) or membrane (second-generation ACI), or placed on to a biodegradable membrane (matrix-/membrane-induced chondrocyte implantation (MACI)) and then implanted into the defect.

The goals of cartilage repair are the following: (1) Restore the articular cartilage surface. (2) Match biochemical and biomechanical properties of normal hyaline cartilage. (3) Improve patient symptoms and function. (4) Prevent or slow progression of focal chondral injury. The advantages and disadvantages of ACI are listed in Table 10.1a, b.

10.2 Workup and Decision Making

It is imperative to obtain a comprehensive medical history and to perform a thorough physical exam. One must be very suspicious of patients who have a history of acute or repetitive ankle trauma. Further workup would include weight-bearing radiographs, followed by CT scan and MRI. Issues with ankle joint alignment and stability need to be identified so that necessary steps can be taken to correct the associated pathology if necessary. Indications and contraindications for ACI in the ankle are listed in Table 10.2a, b.

10.3 Technique

10.3.1 Stage 1

Currently, autologous chondrocyte implantation is performed as a two-stage procedure. Stage 1 consists of a diagnostic ankle arthroscopy and harvest of chondrocytes from either the knee or the ankle. Giannini et al. have recommended harvesting the damaged cartilage, but the results have been mixed as some studies have demonstrated decreased viability of chondrocytes [5]. Matricali et al. recommended harvesting cartilage from the posteromedial aspect of the ankle [6]. The advantage would be similar chondrocytes as well as incisions only around the ankle. There are some theoretical advantages to using ankle chondrocytes as well. Compared to knee cartilage, ankle cartilage is stiffer, thinner, and less permeable. Ankle chondrocytes make more glycosaminoglycan and do not express MMP-8, which makes ankle cartilage more resistant to progressive degeneration [7]. Though there are theoretical advantages of harvesting ankle chondrocytes, knee cartilage is easily accessible with minimal morbidity. Therefore, one of the more common techniques is to perform arthroscopy of the knee and harvest of 200–300 mg of cartilage tissue from the non-weight-bearing portion of the intercondylar notch.

The native chondrocytes are then typically sent to a lab (in the USA, the chondrocytes are sent to Vericel Corporation (Cambridge, MA)) for isolation and proliferation. The process takes 2–3 weeks after which the patient may return to the operating room to receive the cells.

10.3.2 Stage 2

An open osteotomy technique is needed to perform the ACI procedure. An appropriate incision is made on the medial or lateral aspect of the ankle depending on the location of the OLT. The dissection is carried down to the bone. The level of the osteotomy is determined prior to cutting the bone by inserting a Kirschner wire through the bone at an oblique angle directed just past the lateral extent of a medial lesion or medial to the extent of a lateral lesion. This is verified under fluoroscopy and is imperative to ensure adequate exposure of the OLT. We recommend predrilling screw holes for osteotomy site fixation prior to making the bone cut (Fig. 10.2). This will ensure anatomic alignment after fixation. The appropriate osteotomy is then made with an oscillating

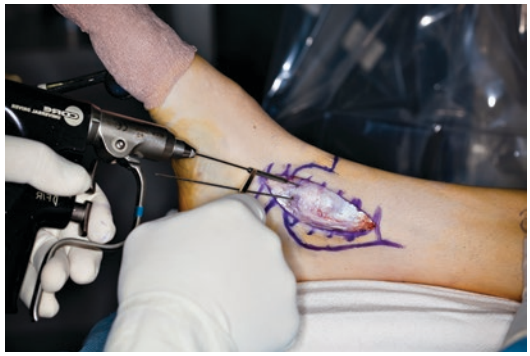

Fig. 10.2 Prior to making the medial malleolar osteotomy, the medial malleolus is predrilled with cannulated 4.0 mm screws to facilitate easier reattachment at the end of the case

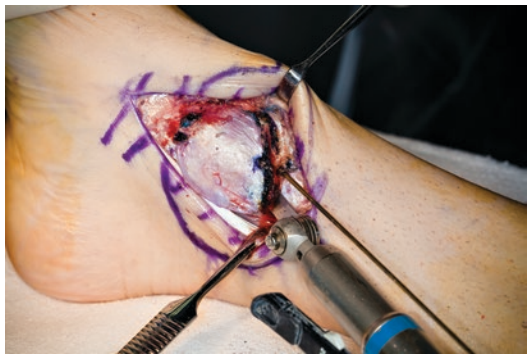

Fig. 10.3 Medial malleolar osteotomy in a right ankle. This osteotomy is performed by inserting a guide pin lateral to the osteotomy and then cutting along the guide pin(s) with an oblique osteotomy, using an oscillating saw. The osteotomy will be finished carefully using an osteotome

saw and finished carefully with an osteotome (Fig. 10.3). The osteochondral lesion is then debrided with care to preserve the subchondral plate. This will prevent bleeding into the defect, which has been shown to contaminate the cultured chondrocyte population. The walls of the chondral defect should be debrided to vertical sidewalls. Once the debridement is complete and the lesion optimally exposed, a template for the size of the lesion can be obtained. An impression of the lesion can be made using foil, which can then be carried to the back table to template the cut of the periosteal patch or other graft tissue (Fig. 10.4).

If a periosteal patch is used to cover the chondrocytes, it can be harvested from either the

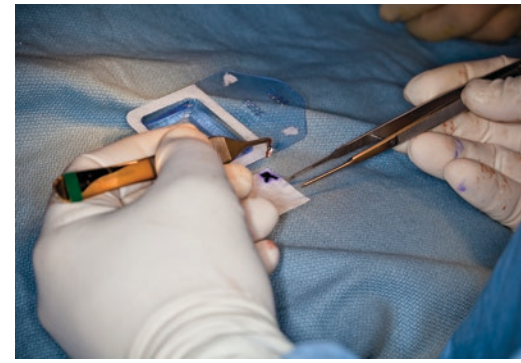

Fig. 10.4 The autologous chondrocytes are loaded onto the collagen type I/III membrane prior to insertion onto the osteochondral defect. This requires careful measurement of the appropriate size of the membrane prior to insertion

proximal or distal tibia. The most efficient method is to harvest from the distal tibia about 2–3 cm above your osteotomy site. Sharp removal with a 15 blade should be employed and electrocautery should be avoided to prevent necrosis of the periosteal flap. Care should be taken to make a periosteal graft that is 1–2 mm larger than the actual defect. On the back table, the soft tissue should be debrided off of the graft and the noncambium layer marked. Store the graft in a sponge to prevent graft shrinkage.

The periosteal membrane is thin and consists of an outer fibrous layer and an inner cambium layer that is osteogenic and nonpermeable. The graft is then placed into the prepared talar defect with the cambium layer facing toward the bone. It is secured into place using 5–0 or 6–0 vicryl sutures. The sutures are placed about 3 mm apart and the knot should be placed on the graft side leaving a hole for chondrocyte insertion. The edges are sealed with fibrin glue. Next an angiocatheter is introduced into the hole in the periosteum layer and a watertight seal is verified by injecting a very small amount of saline. All of the saline is then removed, and the harvested chondrocytes are injected. The hole in the periosteal patch is closed with a 6–0 vicryl, and fibrin glue is used as a final sealant (Fig. 10.5).

For cystic lesions, a sandwich technique can be employed. The base of the lesion is debrided thoroughly. We routinely use curettes and high-speed burrs for adequate debridement and drill

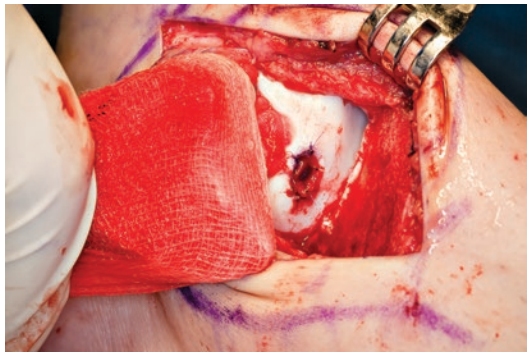

Fig. 10.5 Completed attachment of the type I/III collagen membrane with the cells injected underneath and the construct sealed with fibrin glue

the base as needed. Autologous bone graft from the iliac crest, proximal tibia, or calcaneus is then utilized to fill the cystic defect to the level of the subchondral bone. Because of the exposed bone graft, two periosteal grafts are needed with the chondrocytes in between forming a sandwich. After bone grafting, the first periosteal graft is placed with the cambium layer facing the articular surface and secured with 6–0 vicryl sutures and reinforced with fibrin glue along the periphery. The second periosteal patch is then placed over the defect, with the cambium layer facing the cambium layer of the previous patch.

If a type I/III porcine cartilage patch (biogide) or membrane/matrix bilayer (MACI) is available, then this would be the preferred vehicle for coverage and delivery of the chondrocytes. These patches don't require harvesting of the periosteum, can be cut to fit the size of the defect, and have lower rates of graft hypertrophy. The surgical technique is the same as outlined above. In the USA, there is no collagen membrane that is currently approved by the FDA for cartilage repair. The Chondro-Gide (Geistlich Biomaterials, Wolhusen, Switzerland) has been used in the USA off-label. A similar membrane (called Biogide) is currently approved in the USA for dental procedures only and also used off-label as a periosteum substitute. However, MACI® (Vericel Corporation, Cambridge, MA) has recently been FDA-approved in the USA and is a membrane-induced chondrogenic delivery system, which will likely supplant previous generations of ACI. The major advantage of the MACI system is that the membrane contains harvested and expanded chondrocytes and eliminates the need to inject the cells under a membrane or periosteum. It reportedly can also be placed with fibrin glue alone; however, many surgeons still prefer to place sutures for stability.

Once the OLT has been filled, the osteotomy can then be reduced and fixated according to basic AO principles. Predrilling the fixation holes will ensure anatomic osteotomy reduction. The medial malleolus is fixed with two 4.0-mm AO-cannulated screws placed obliquely up the medial malleolus and one transverse screw. The lateral malleolus osteotomy is stabilized with two 3.5 mm lag screws and a one/third tubular neutralization plate. The soft tissues are closed in layers, and the patient is placed into a well-padded splint.

Over the last 15 years, much has been learned about performing the ACI procedure. Great attention to detail is critical to achieve a successful outcome in this technically demanding operation (Table 10.3).

10.4 Postoperative Care

Patients are placed into a well-padded short-leg splint and are placed on a strict non-weight-bearing protocol for the immediate postoperative period. They return to clinic at two weeks, and sutures are removed if appropriate. If wounds need longer to heal, then they are kept immobilized and non-weight-bearing. Once wounds are healed, the patient is transitioned into a CAM walker and TED hose and are advanced to 30 lb. partial weight-bearing. They are allowed to start home range-of-motion exercises at this time. At 6 weeks postoperative, they are encouraged to start a progressive weight-bearing program to full weight-bearing and formal physical therapy is initiated. We have a 4-phase rehabilitation protocol: early (<8 weeks), transition (8–12 weeks), mid-phase (3–5 months), and final phase (6–12 months). Phase 1 goals are to focus on range of motion so that full motion is achieved by 8 weeks. Stationary bike without resistance is a

good modality during this phase. Phase 2 involves proprioceptive exercises, isometric exercises, followed by eccentric strengthening, and closed-chained presses. Phase 3 focuses on increasing walking distance and speed and load training is increased. Phase 4 involves the initiation of sport-specific training [8, 9].

10.5 Results

As ACI becomes more prevalent, more studies are being produced. Recently Baums et al. reported their results on 12 patients treated with ACI [10]. Mean follow-up was 63 months. Outcome scores as measured by the Hannover ankle score and AOFAS mean score increased from 40 to 86 and 43.5–88.4, respectively. All patients previously involved in competitive sports returned to full activity.

These findings were similar to Nam and Ferkel who published their findings on 11 pts. AOFAS scores improved from 47 to 84 [11]. Perhaps more interestingly, in their study, they performed second-look arthroscopy on 10 patients. All lesions were covered by "cartilage-like" surface. They noted that the firmness of the cartilage increased with longer follow-up. That study was further enhanced by Kwak and Ferkel adding an additional 18 patients to the original 11 [12]. Average follow-up was 70 months and average lesion size was 198 mm^2. Second-look arthroscopy was performed on 90% of those patients, and results were similar to the original study. AOFAS scores improved from 50.1 to 85.9 on average.

Giannini et al. have reported long-term follow-up [13]. Their results on 10 patients showed improvement in the AOFAS score from 37.9 to 92.7 at 10 years after surgery. MRI was also completed and showed restoration of the articular surface.

There are some reported issues with the periosteal patch as reported in the knee. Reoperation rates have been as high as 50%. Most frequently, reoperation is a result of graft hypertrophy, delamination, failure, and insufficient regenerative cartilage [14].

10.6 Second-Generation ACI

Second-generation techniques of chondrocyte delivery involve the use of absorbable porcine type I/III collagen membrane as a substitution for periosteal graft. This reduces the risk of graft hypertrophy and periosteal donor site morbidity, while also providing an opportunity for a minimally invasive approach. Collagen-covered autologous chondrocyte implantation (CACI or ACI-C) is currently not approved in the USA by the FDA. However, the Chondro-Gide patch (Geistlich Biomaterials, Wolhusen, Switzerland) has been used off-label by many surgeons. Its application in the ankle has not been published, but results in the knee are promising [15]. The major trend in the literature is that the risk for reoperation for graft hypertrophy is significantly decreased. This is highlighted by the retrospective review by Gomoll et al. [16]. They looked at 100 ACI-C and 300 ACI-P procedures at 1-year follow-up: 25% of patients treated with ACI-P required reoperation for graft hypertrophy versus 5% with ACI-C.

10.7 Matrix-Induced Autologous Chondrocyte Implantation (MACI)

Third-generation technology has allowed implanting ex vivo expanded autologous chondrocytes onto biodegradable scaffolds. This obviates the need for a periosteal patch and introduces the possibility of performing the case through minimally invasive or arthroscopic methods.

Giannini et al. reported on 46 patients treated with Hyalograft C (Fidia Advanced Biopolymers, Abano Terme, Italy), which is a product made from HYAFF-11 benzylic ester of hyaluronic acid [17]. They had follow-up of 12 and 18 months and showed a mean AOFAS score increase from 57.2 to 86.8 at 12 months and 89.5 at 18 months. Good-to-excellent results were achieved in 82 patients.

MACI® (Vericel, Cambridge, MA) was recently approved by the FDA in the USA. Their product uses expanded chondrocytes implanted

on a porcine I/III collagen membrane. Its approval was based on the results of the SUMMIT trial in the knee, which demonstrated improved outcomes from MACI procedures over microfracture [18].

Results of use for OLTs have been promising. Guillen and Abelow reported on their first 50 cases of MACI, eight of which were OLT measuring 2–6 cm [19]. Good-to-excellent results were appreciated in six of these patients. Schneider and Karaikudi found statistically significant improvement in AOFAS from 60 to 87 in 20 patients who underwent MACI. Mean size of lesion was 233 mm^2 and follow-up was 21.1 months. Only two patients clinically failed [20]. Magnan et al. treated 30 patients with MACI [21]. The mean OLT was 2.36 cm^2, and follow-up was 45 months. They found an AOFAS score increase from 37 to 84, but only 50% returned to previous sport.

Kreulen et al. recently reported 7-year outcomes on nine patients. Preoperative AOFAS scores improved from 65 to 85 postoperatively. These patients also demonstrated significant improvements in social and physical functions based on SF-36 outcome scores [22].

Giza et al. prospectively collected data on ten patients who had previously failed microfracture of the talus [23]. These patients were treated with MACI procedure and followed for 2 years. AOFAS and SF-36 showed statistical improvement in all patients [24]. Anders reported follow-up on 21 patients who underwent MACI for the talus and similarly demonstrated improvement in AOFAS from 70 to 95.

Bartlett et al. have reported the results of MACI versus ACI-C [25]. They performed a prospective randomized controlled trial on 91 patients with symptomatic chondral defects of the knee. There were no statistical differences in patient outcomes and rate of patch hypertrophy was 9% versus 6% in ACI-C and MACI groups respectively.

10.8 Future Direction

ACI/MACI has shown good results thus far, but newer investigations may lead to alternative techniques, requiring only one stage for treatment. One such technique is utilizing particulated juvenile cartilage. The surgery can be completed in one stage and done all-arthroscopically. We prefer the arthroscopic technique to remove the OLT and perform microfracture and drilling. The particulated cartilage can then be added to a fibrin glue bed. If a cyst is present, bone grafting can be performed prior to the placement of the particulated cartilage. Recently Coetzee et al. reported on the results of particulated juvenile cartilage used for OLTs and found good-to-excellent results in 78% of patients [26].

One-stage techniques using a collagen membrane as a patch and platelet-rich plasma to deliver mesenchymal stem cells are intriguing, but not yet fully studied. A newer material called micronized cartilage matrix has been studied for its ability to form a new articular surface over an OLT. It consists of allograft cartilage extracellular matrix that contains type II collagen, proteoglycans, and additional cartilage growth factors. It is dehydrated and micronized and has a shelf life of 5 years. Unfortunately, there is little published evidence to support its use at this time.

Recently, a newer one-stage technique has been developed using the patient's own cartilage. CartiOne (Orteq Sports Medicine, London, UK) is a one-stage procedure that utilizes autologous chondrocytes harvested same day and combines those cells with bone marrow aspirate. This theoretically allows the mononuclear cells to potentiate the harvested chondrocytes. The cells can then be planted onto a wide variety of scaffolds/membranes and then placed onto the OLT defect. The obvious advantage is that there is no need for a two-stage procedure. CartiOne is not FDA-approved in the USA.

10.9 Conclusion

It is imperative to thoroughly work up patients with suspected OLTs. Weight-bearing radiographs, CT scans, and MRIs are required for a thorough evaluation. Larger lesions, or those that fail microfracture, may require a cartilage restoration procedure. ACI/MACI has shown good

clinical results in a number of studies. It is important to remember potential complications of these procedures including graft hypertrophy, donor site morbidity, and the current requirement for two procedures. Future investigations will likely reveal a method to procure and implant chondrocytes in a single stage with excellent cell viability and hyaline cartilage restoration.

Appendix

Table 10.1 Advantages and disadvantages of ACI

a	b
Advantages of ACI	Disadvantages of ACI
• Autologous tissue • Hyaline-like tissue formation • Minimal donor site morbidity • Can treat larger lesions	• Technically difficult • Staged surgery required (for now) • High cost • May need to bone graft • Extended recovery

Table 10.2 Indications and contradictions for ACI in ankle

a	b
Indications for ACI in the Ankle	Contraindications
General • Age 15-55 • Focal Defect >1cm^2 • Unipolar Lesions (Only Talus) • Contained • Edge Loading • Failed Previous Surgery • Large lesions, especially those with subchondral cystic changes in primary cases	Absolute • Osteoarthritis • Uncorrected Malignment • Uncorrected Instability Relative • Bipolar Lesions • No previous surgery • Early degenerative joint disease

Table 10.3 ACI technique pearls

ACI Technique Pearls
• Adequate osteotomy • Excise all diseased tissue • Bone graft deep cysts • When suturing the periosteum or membrane, grasp 4-5mm into normal cartilage • Seal graft completely • Correct associated instability • Rigid fixation of osteotomy

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11 Repair by Autologous Collagen-Induced Chondrogenesis (ACIC) Technique

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11.1 Introduction

Articular cartilage does not contain vascular, nervous, and lymphatic tissues. The absence of trophic support and the lack of access to reparative and humoral factors that promote tissue healing determine a limited repair potential of cartilage lesions [1, 2]. Due to this limited ability of regeneration in response to injury and the scarcity of therapeutic options, even minor articular cartilage lesions may lead to progressive damage and joint degeneration [3, 4]. For all these reasons, chondral lesions have always been considered difficult to treat [5]. Many therapeutic strategies were set up over the last years. Nonoperative treatment is useful only for symptoms relief and it should represent the first approach to the patient. When surgery is needed, treatment includes one-step procedures, such as cartilage debridment [6], microfractures, and osteochondral autograft transfer system (OATS) [7], and two-steps approach, including chondrocyte implantation (ACI) [8] and matrix-induced autologous chondrocytes trasplantation (MACI) [9].

The purpose of orthopedic surgeons, as seen in the last year, was to achieve satisfactory structural and biomechanical restoration of the articular cartilage exploiting the potential of cell-based therapy, mainly based on the recruitment of mesenchymal stem cells (MSCs) from bone marrow. Proliferative potential and multilineage differentiation of MSCs constitute the principle of regeneration of chondral tissue, by directly replacing damaged cells. Furthermore, they have an important role in supporting the articular homeostasis, by secreting a variety of cytokines with anti-inflammatory activities.

As known, the blood clot deriving from microfractures of the subchondral bone contains progenitor cells. A recent therapeutic approach, called matrix-assisted microfracture technique, combining microfractures with the use of biological matrix has been proposed [10, 11] to stabilize and maintain the blood clot in the lesion site.

Among these approaches, autologous collagen-induced chondrogenesis (ACIC) technique might be quite effective; the main innovation is represented by the composition of the matrix, which is composed by injectable atelocollagen, which is able to polymerize and allow performing the entire procedure arthroscopically. We conducted a study, already published [12], to

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assess the cytocompatibility of the collagen gel-based matrix (Cartifill™, Regenerative Medicine System) using human bone marrow mesenchymal stem cells (BMSCs) and human chondrocytes (HCs) and the preliminary outcome of five patients affected by an osteochondral lesion of the talus treated with the arthroscopical ACIC technique.

11.2 ACIC Treatment [12, 13]

Our indication for the ACIC technique treatment are chondral lesions of the talus from 2 to 4 cm^2, grade III–IV according to International Cartilage Repair Society scale technique (Figs. 11.1 and 11.2).

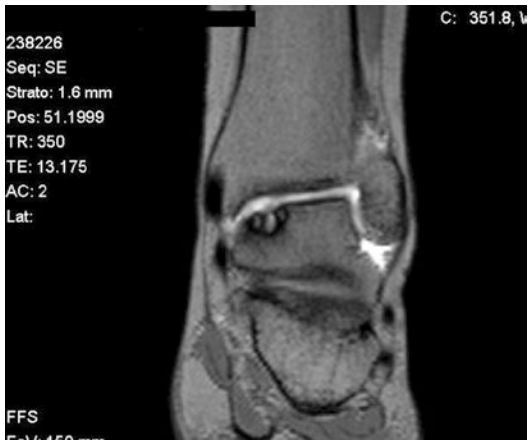

Fig. 11.1 Coronal ankle MRI of a patient included in the study

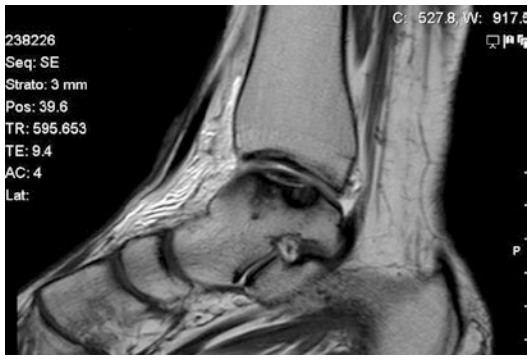

Fig. 11.2 Sagittal ankle MRI of a patient included in the study

Our exclusion criteria were arthritis of the ankle joint, "kissing" lesions, untreated ankle instability or malalignment, tendon tears of ankle joint, previous surgery at the same ankle, rheumatoid arthritis, or any other inflammatory arthropathy.

All procedures have been carried out using a single arthroscopic step, through standard anteromedial and anterolateral approach for the ankle and thigh tourniquet for all patients at 250 mm/Hg. We evaluated the lesions and then we debrided the defect down to the subchondral bone using a shaver and then a curette.

It's very important to obtain a stable "shoulder" at the margin of the defect.

Microfractures were performed using a 4–5 mm angled chondral pick, placed 3–4 mm apart from each other (Figs. 11.3 and 11.4). Then, tourniquet was removed, water was replaced by carbon dioxide (CO_2) in order to maintain the same joint distension and avoid the dilution of the injectable matrix (Fig. 11.5). It's important to make the joint completely dry, residual water in the joint was aspirated using a suction tube. As reported in the manufacturer's sheet, Cartifill™ was combined with human fibrin glue in 1:1 ratio to allow its polymerization. The mix of these two components was guaranteed by a prefilled double chamber syringe connected with a needle and that was introduced into the joint using the arthroscopic portal.

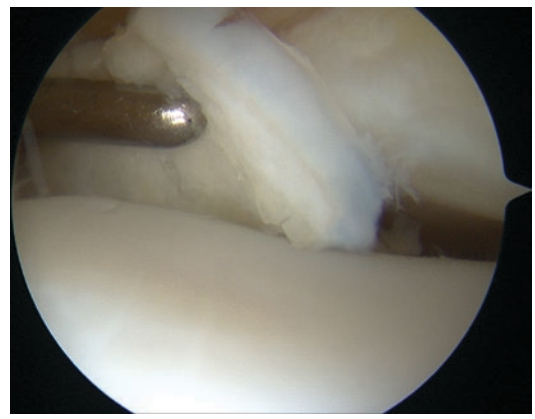

Fig. 11.3 Arthroscopic view of chondral lesion of the talus

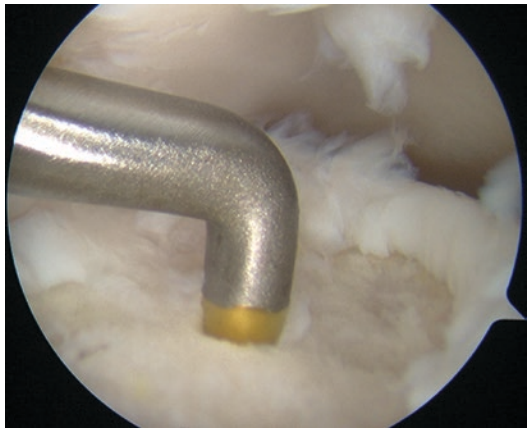

Fig. 11.4 Arthroscopic view of the chondral pick used for microfractures

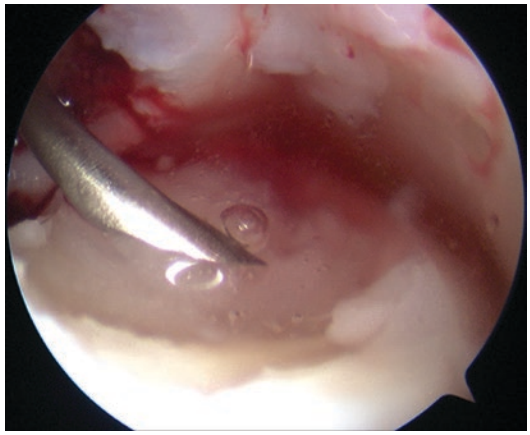

Fig. 11.5 Covering of the lesion with Cartifill™

Under arthroscopic vision, the defect was filled with Cartifill™. Once the first layer of Cartifll, we waited for 1 or 2 min and then we filled up the defect with more gel over the first layer, to achieve full-defect filling.

After being introduced and spread on the lesion, Cartifill™ polymerized within 5–8 min (Fig. 11.6).

Once Cartifill™ is well dry, it is important to check the stability of the implant by moving the ankle through a full range of motion several times. CO_2 was switched off, and the joint was insufflated with normal saline under pressure.

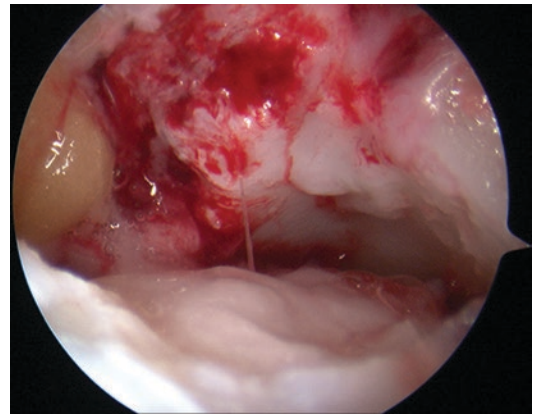

Fig. 11.6 Arthroscopic final view

The skin was closed with sutures.

We discharged the patients the day after surgery without weight-bearing for 3 weeks and 3 weeks with partial weight-bearing without any use of cast or walker boot.

11.3 Results

11.3.1 In Vitro Analysis Results

After 7 days of culture of both BMSCs and CHs, histological evaluation of cell-scaffold construct was performed (Fig. 11.7). Hematoxylin-eosin revealed that both BMSCs and CHs seeded on presolid atelocollagen scaffold, independently from the cell concentration used, were able to grow and colonize the whole sphere, from the surface to the core (Fig. 11.7a, b). On the contrary, BMSCs and CHs seeded on solid scaffolds were not able to migrate within the scaffold but just grew on the surface; similar results were observed for both the two cell densities 0.5×10^5 and 10^5 (Fig. 11.7c, d). Morphologically, CHs cells appeared to have a smaller size (approximately 15–20 μm in diameter) and rounded, while hBMSCs were spindled to stellate. No GAGs or collagen II deposition was found in the examined samples, probably due to the too short period of culture.

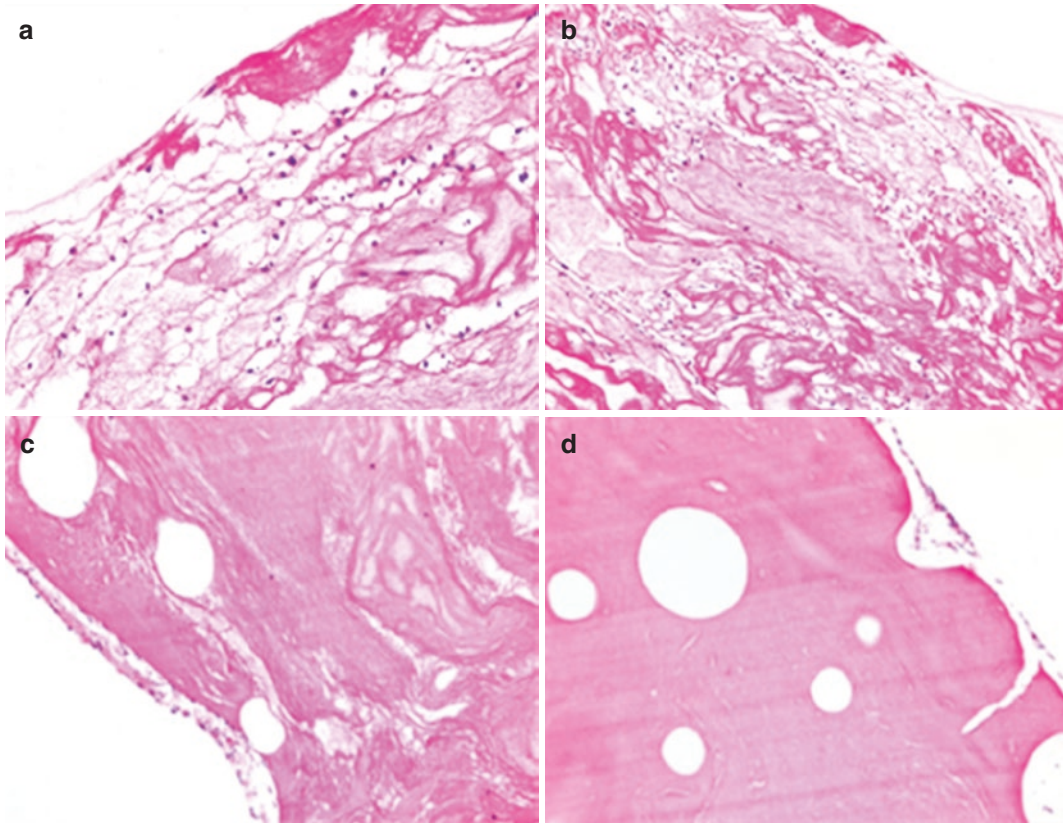

Fig. 11.7 Histological evaluation colored with hematoxylin-eosin of atelocollagen scaffold at presolid state (**a**, **b**) and after polymerization (**c**, **d**), seeded with BMSCs (**a**, **c**) and CHs (**b**, **d**) at a concentration of 10^5

11.3.2 In Vivo Clinical Results

All the patients had a rapid recovery after surgery. Nobody suffered from joint swelling or had severe pain after surgery; moreover, no infections occurred in the postoperative period.

A significant improvement in AOFAS and VAS scores was observed in all patients treated with ACIC technique.

A general and marked improvement was observed in VAS pain scale.

As regards AOFAS score, the difference between presurgical values and follow-up results is statistically significant.

After 6 months from surgery, the VAS scale for pain improved significantly.

When measured against Tegner activity scale, we got ambivalent results: in some patients we recorded a decline. Other two patients showed an improvement of their activity level and finally in one case, we observed the same presurgical level at the 6-month follow-up. Regarding AOFAS, we found improvements in all the patients compared to preoperative situation. We clinically monitored the patients at 2 weeks after surgery and we did not record any adverse events or complications such as infections, nerve palsies, synovitis, and allergic reactions.

11.4 Discussion

In our in vitro experiment [12], both BMSCs and CHs were found to be able to grow and colonize the surface and the core of the presolid atelocollagen scaffold. This didn't happen when the solid atelocollagen scaffold was used, independently from cell density. In this case, cells seeded on

scaffolds remained confined on the edge and they were not able to move on toward the center of the scaffold.

When GAGs were evaluated at 7 days, they were negative in all samples. Probably this period of time is insufficient to allow the production of an in vitro collagenic matrix. It would be useful, to evaluate the quality of the matrix produced, to coculture cells for a longer period of time.

However, these results seem to suggest that atelocollagen is capable to cover the lesion efficiently, forming a homogenous covering matrix; moreover, the in vitro findings suggest that to achieve the best results Cartifill™ should be applied before its complete solidification after fibrinogen addiction in order to allow a homogenous cellular distribution.

ACIC technique proved to be safe, and it does not add any potential risk to a common ankle arthroscopy. It is a feasible and easy technique to be completely performed arthroscopically. This feature makes ACIC technique very suitable for joints characterized by difficult surgical access such as the ankle and especially in the treatment of chondral lesions of the talus, which often still require open surgical procedures.

We did not observe a significant extension of surgery time in relation to isolated microfractures. This technique is also performed on other joints such as the knee with good results [13]. Our clinical results are similar to those obtained by Van Dijk et al. [14]. that reported 14% of unsatisfactory results in patients affected by osteochondral lesion of the talus treated with microfractures technique, and to those obtained by Gobbi et al. [15] that reported 20% of persistent pain in a similar population after microfractures of the talus.

It is difficult to compare clinically ACIC with other techniques because of the lack of RCT. We believe, as already stated, that more patients need to be studied and included in further studies.

Tegner score values slightly decreased after the surgical procedure. However, since Tegner score evaluated the sport activity level, this decrease can be explained by the fact that usually patients return to sports after 6 months from this kind of surgery. Indeed, two patients had very high preoperative values (score 9) and both of them, at the time of evaluation, had not returned to their original activities yet. In particular, one of them, a basketball player in the Italian lower division, was in the middle of physical training in order to come back to official basketball games. In the other three cases (with medium or low Tegner starting value), we assessed an improvement or, at least, the same result compared to preoperative Tegner scores.

Cartifill™ proved to be cytocompatible and in the presolid state, it allows a more uniform cellular distribution both for BMSCs and CHs. The microfractures technique associated with the use of injectable matrixes in the treatment of IV grade chondral lesions might be a remarkable advantage, because it is less invasive, especially in the talus, as it is a fully arthroscopic technique.

11.5 Tips and Tricks

Clean and debride the lesion very well in order to obtain a well-defined “shoulder”
Remove the tourniquet before applying the gel to optimize growth cells
Fill the lesion well with Cartifill™ (twice)
Check the stability of the system well

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Restore by Mesenchymal Cells

12

Roberto Buda, Luca Perazzo, Matteo Baldassarri, Andrea Pantalone, and Danilo Bruni

12.1 Characteristics of Mesenchymal Cells (Bone Marrow-Derived Cell Transplantation)

Mesenchymal Stem Cells (MSCs) are multipotent stromal cells that can differentiate in several lineages responding to local environmental stimuli such as cytokines and growth factors, which are released in response of tissue injury [1, 2]. Recently, MSCs, because of their ability to differentiate into various cellular lineages, including osteoblasts and chondrocytes [3, 4], were proposed as a new option for the treatment of articular cartilage defects [5].

MSCs differentiation in the desired direction may be achieved as a result of environment, mechanical stimulation, and growth factors capable of stimulating cells toward osteogenesis and chondrogenesis [6–8].

Although the use of MSCs as a pure cell lineage has been advocated [5], the key role in the described technique is played by the surrounding microenvironment (or niche) obtained by centrifugation of the bone marrow aspired. The bone marrow-derived cells transplantation (BMDCT) in fact allows the implantation of MSCs together with all the mononuclear cells and high regenerative potential factors present in the bone marrow [9–11]. Furthermore, autologous bone marrow contains not only stem cells and precursor cells, but also accessory cells that support angiogenesis and vasculogenesis by producing several growth factors. According to this rationale, it is possible to implant into the cartilage lesion the entire regenerative potential of the niche, without the need to select and expand the MSCs in a lab phase. A one-step procedure is therefore permitted [12].

Moreover, an autologous platelet-rich fibrin gel (PRF) is used in order to provide directly in situ additional growth factors. The platelet gel is a very effective "accelerator" for healing processes [13]. The secretory granules of platelets, the α granules, contain platelet-derived growth factors AA, BB, and AB; transforming growth factors β1 (TGF-β1) and β2; platelet-derived epidermal growth factor; platelet-derived angiogenesis factor; insulin growth factor 1 and platelet factor 4, which influences bone regeneration [14]. Moreover, the platelet-rich fibrin (PRF) is richest in fibrin and is able to coagulate faster than platelet-rich plasma, providing an additional stability of the implant due to its jelly consistence.

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The combination of MSCs, biological scaffold, and platelet-rich concentrates is fundamental to improve chondrogenic and osteogenic differentiation and cell growth, tissue regeneration, and the control of the articular environment. All of these components generate a bioactive construct completely integrated with the joint microenvironment and the surrounding tissues [15]. This technique should be reserved to young and active patients with focal osteochondral defects and, in selected cases, concomitant mild or moderate OA.

12.2 Surgical Technique

12.2.1 Indications

The treatment is indicated for focal osteochondral lesions of the talar dome classified as grade 3–4 according to International Cartilage Repair Society classification (ICRS) (area of the lesion $\geq$1.0 cm^2, depth of the lesion <5 mm) [16]. For lesions of depth higher than 5 mm, autologous bone grafting is needed. Patients younger than 14 years or older than 60 years, patients with osteoarthritis or kissing lesions of the ankle, and patients with rheumatoid arthritis should not be treated with this technique. Malalignment of the lower limb and the presence of joint laxity are considered relative contraindications to be corrected if present.

12.2.2 Surgical Procedure

The surgical technique for the BMDCT consists of several phases, all to be performed during the same surgical session.

12.2.2.1 Platelet-Rich Fibrin Gel Production

The PRF is produced with an automatic system (The Vivostat® systems) the day before the operation or the same day: 120 ml of venous blood is harvested with a needle size 16 connected to a bowl previously prepared with the anticoagulant solution. The bowl is then inserted inside the spinner and processed. At the end of the machine cycle, a syringe containing 6 ml of PRF is extracted to be stored at −35 °C or either used immediately. In case of storage, the PRF needs to be slowly heated to room temperature 30 min before to its use.

12.2.2.2 Bone Marrow Aspiration (Video 12.1)

The bone marrow is aspirated from the posterior superior iliac crest after preparation of a sterile surgical field with the patient lying prone and already under spinal or general anesthesia. The equipment for the bone marrow harvesting, concentration, and implant is part of a dedicated kit for osteochondral regeneration developed by Rizzoli Orthopaedic Institute (Bologna, IT) and Novagenit (Mezzolombardo TN, Italy).

By insertion of a needle size 11 G to a depth of about 3 cm in the iliac crest, a total of 60 ml of bone marrow is harvested as a result of subsequent aspirations. Needle and syringe should be previously heparinized. The bone marrow harvested is placed in a bag preloaded with 500 UI of sodium heparin (EPSODILAVE) in 10 ml. The harvesting is made in little steps on different locations on the crest in order to maximize the collection of stromal cells useful for the regeneration and minimize the diluting effect coming from the aspiration of the peripheral blood (Fig. 12.1).

12.2.2.3 Bone Marrow Concentration (Video 12.2)

The previously extracted bone marrow volume is then reduced by eliminating the plasma and the red cells, therefore increasing its stem cells concentration. This procedure is performed directly at the end of the aspiration phase in the operating room using a cells separator-concentrator and its related sterile and disposable kit. In 12 min, 6 ml of concentrated cell, containing the mesenchymal stem cells and other cell populations, which constitute the nucleated bone marrow microenvironment, is obtained.

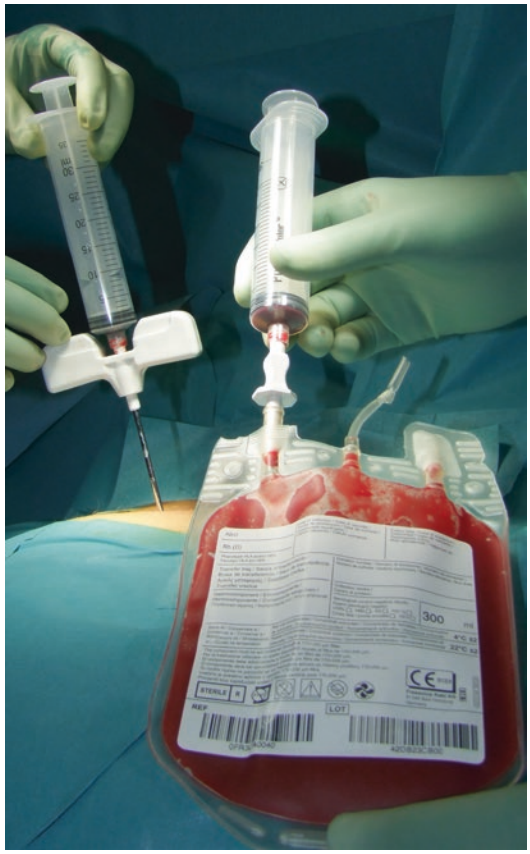

Fig. 12.1 Surgical field showing aspiration of the bone marrow from the posterior iliac crest

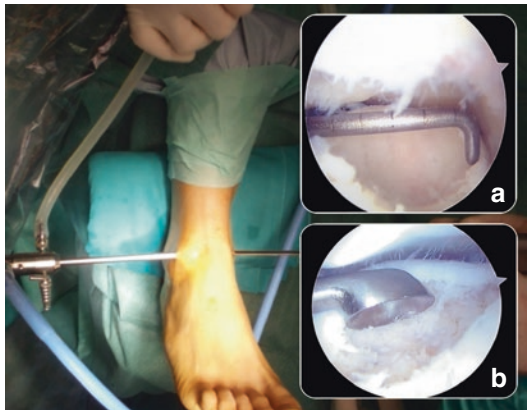

Fig. 12.2 A standard ankle arthroscopy is performed and the lesion is inspected (**a**) and shaved until healthy subchondral bone is reached (**b**)

12.2.2.4 Surgical Procedure (Video 12.3)

The patient is positioned in supine decubitus with a tourniquet at the leg to be operated. A standard ankle arthroscopy is performed through anteromedial and anterolateral portals.

The lesion is inspected: articular fibrosis, intra-articular loose bodies, or osteophytes are to be removed. The osteochondral lesion is detected and shaved, until healthy subchondral bone bed is reached (Fig. 12.2).

The size of lesion is measured with the aid of a graduated probe and recorded. The biomaterial to be implanted is then prepared in the same size and shape of the lesion. Borders of the lesion need to be regularized in order to obtain vertical shaped edges.

A scaffold (highly hydrophilic collagen membrane) is used for cells support. Following the size and the dimension of the lesion (previously measured using the sizers provided by a special instrument set) the biomaterial is accurately clipped. Approximately 2 ml of bone marrow concentrate is loaded on the scaffold and fastly absorbed. A dedicated open cannula is then inserted through the arthroscopic access closer to the lesion (Fig. 12.3). The fluid is completely removed from the joint. The previously prepared biomaterial is applied in the window of the cannula and guided to the edge of the lesion. At this point, the cannula is removed and the biomaterial is made to adhere perfectly to the lesion using a flat probe (Fig. 12.4).

Finally the PRF is applied to cover the lesion by means of a spray pen in no-air modality, in order to provide a high concentration of growth factors and to further promote the stability of the implant due to coagulation of the PRF. The implant stability is checked by performing ankle flexion/extension movements. The skin accesses are closed with a 3–0 absorbable suture wire covered by a bandage and replaced the following day by a flat medication.

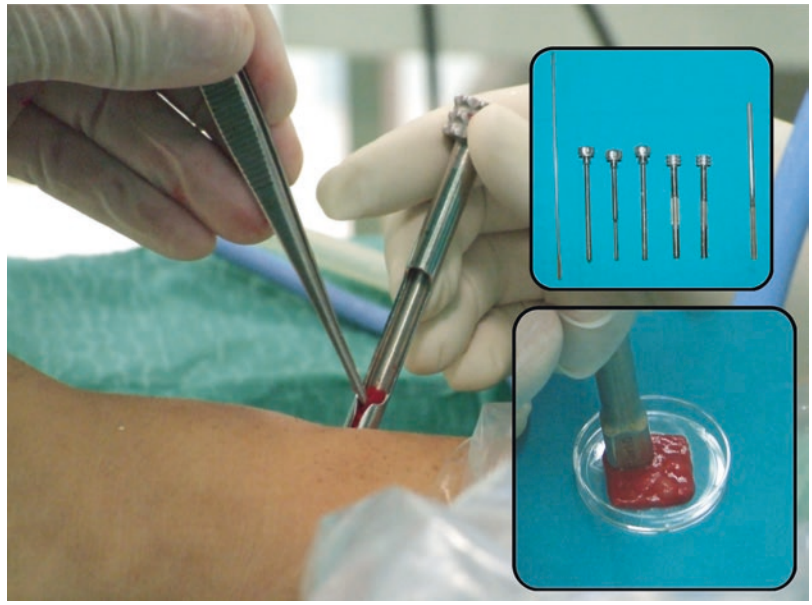

Fig. 12.3 Surgical Instruments to cut scaffold and deploy it into lesion site

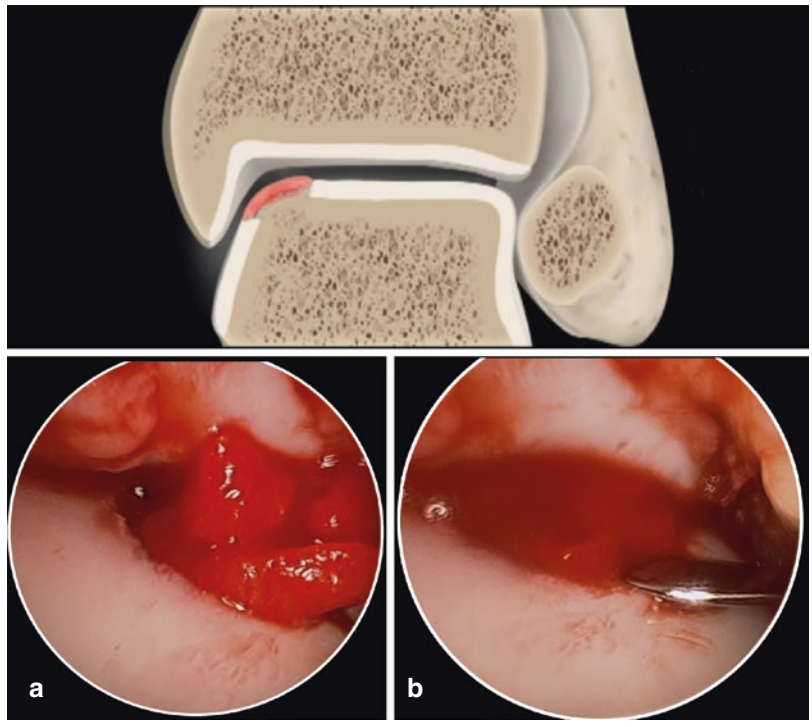

Fig. 12.4 The biomaterial is positioned (**a**) and adhered to the lesion site using a flat probe (**b**)

12.3 Rehabilitation

A team evaluation (Orthopedist, Physiatrist, and Physiotherapist) should personalize rehabilitation treatment for each patient. We can distinguish three phases that can vary depending on the patient's clinical status.

12.3.1 Early Postoperative Phase: From 0 to 6 Weeks After Surgery

In the immediate postoperative the patient performs:

- Continuous passive mobilization (CPM) by means of a motorized device: this movement, through compression and joint decompression, facilitates mesenchymal cells proliferation and their differentiation in the sense of chondrocyte, stimulates the synthesis of molecules of the cartilage matrix, and reduces the risk of adhesions inside the joint.

  Initially, the CPM is performed at slow speed (1 cycle per minute) for 6–8 h a day. Joint excursion must be adjusted according to the pain, seeking movement of flexion and extension.
- Mobilizations carried out passively by the therapist: on all the joints of the foot and ankle in order to retrieve the full movement of all the components of the foot.
- Connective tissue massage, to avoid scar fibrous retractions.
- Stretching exercises for the muscles that tend to be shortened.
- Muscle reinforcement for all foot muscles with particular regard to ankle stabilizers.
- Electrotherapy, especially useful in patients with muscular deficit.
- Lymphatic massage and vascular gymnastics for the reabsorption of edema.
- In-water rehabilitation can be performed starting from 15 days after surgery (after stitches removal) and is indicated at all stages of treatment to improve joint movement and tropism and to overcome the long period during which the patient has no full weight bearing on the operated foot. Furthermore, the in-water therapy allows an intense stimulation of the receptors not only of the affected segment but of the whole body.

For the first 6 weeks after surgery, the patient walks with partial load (20% of body weight) using two crutches.

12.3.2 Late Postoperative Phase: From 6 Weeks to 4 Months After Surgery

At this stage, exercises are performed with manual contrast or with 1–2 kg weights.

Exercises involving muscles of the entire lower limb such as bike riding can be associated.

By the sixth week, the patient may progressively load on the operated foot up to full weight bearing using one crutches. At 8 weeks also the second crutch can be abandoned.

From the eighth week, if the full range of motion and tropism have been recovered, and in the absence of pain, proprioceptive exercises should be started. Proprioceptive rehabilitation is initially carried out without load and from the tenth week bipodalic and then monopodalic load is allowed. The rehabilitation program proceeds with customized exercises in terms of intensity and difficulty based on the clinical picture and on the type of patient. Three months after surgery, isokinetic exercises can be introduced in the rehabilitation treatment.

12.3.3 Physical and Sport Activity Resumption: From 4 Months After Surgery on

Around the fourth month, intense workout and aerobic training in athletic patients can be performed for recovery of adequate fitness. From the sixth month, the run on the field with figure eights and rapid direction changes are allowed together with sport-specific rehabilitation and return to a low-impact sports. Twelve months after surgery, high-impact sports such as football and tennis can be practiced.

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The Use of Allograft

13

Fabrizio Cortese, Domenico Mercurio,
Alessandro Santandrea, and Leonardo Puddu

13.1 Osteochondral Transplant from Donor Talus (Allograft)

Indication for osteochondral transplants is restricted to lesions larger than 15 mm and deeper than 8 mm. The therapeutic principle is to restore the hyaline cartilage-subchondral bone functional unit. The advantage that osteochondral transplants have over the cartilage regeneration techniques is supplying the subchondral bone-hyaline cartilage functional unit with stability due to the osteointegration of the transplant, while the cartilage regeneration techniques aim to regenerate a layer of fibrocartilage covering the subchondral bone [1–3]. Compared to OATS, the advantages consist of no morbidity of the donor site, no limits in diameter and the better matching with the shape of the lesion. In our experience, the indication can be also extended to deep lesions (usually cystic) with a diameter smaller than 15 mm.

The homologous osteochondral transplant (*Allograft*) resolves many of these limitations so that osteocartilage unit can be inserted into the lesion in order to reproduce the shape of the damaged bone. The transplantation is harvested from a donor talus whose size must be as close as possible to the patient bone. During the intra-operative phase, the precise size and shape of the lesion is determined and the graft is taken in the same position and in the same shape to create maximum compatibility. The possibility to use a single transplant instead of several osteocondral cylindrical grafts like in OATS reduces the risk of fibrocartilage forming between the grafts [4, 5].

The allograft can be fresh or frozen. The advantage of fresh transplant is the presence of chondrocytes in the cartilage, which reduces in direct proportion with the time elapsing between harvesting from donor and the implant. Literature report that at 4 weeks, 84% of the chondrocytes are still present and viable in the graft [6] at 44 days this percentage is reduced at 67% [7]. The freezing process kills the chondrocytes, and causes tears and delaminations of the cartilage that can lead to breakage of the articular surface of the transplant. This evidence makes the fresh allograft better indicated for this procedure and suggests reducing the time between the harvesting from the cadaver and the implant. The potential problems associated with this procedure are: high costs, the risk of transmitting infectious disease, the risk of immunisation, transplant rejection and the risk of non-osteointegration. The

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role that the surface antigens (HLA) play has not yet been clarified. Literature does not report problems with histocompatibility for osteochondral transplants of either the knee or the ankle, and this type of screening is not recommended. On the other hand frozen allograft is more easily available, has lower risk of disease transmission and transplant rejection and, despite its poor cellularity, report good long distance results at follow-up. Probably the graft in theese cases act like a scaffold which is colonized by the recipient's cells [8].

13.2 Surgical Technique

The treatment of osteochondral lesions with allograft is usually an open procedure and arthroscopy can be performed before the open approach to confirm the site and the size of the lesion.

13.3 Arthroscopic Technique

The operation can be also performed with totally arthroscopic technique in case of a small anteromedial or anterolateral lesion (max. 10 mm in diameter, usually cystic lesions). In this case, it is possible to use the OATS instruments to prepare the lesion site and harvesting of the graft. To prepare the receiving site, a secondary access is usually made so that the instruments can be introduced perfectly perpendicular to the articular surface. Van Dijk's group studied arthroscopic accessibility of the talar articular surface with the foot in plantar flexion. The study is based on CT images made with the foot in maximum plantar flexion. According to this study, 48.29 ± 6.7% of the talus dome medially and 47.8 ± 6.5% laterally can be reached with arthroscopic technique. This parameter depends on the articular ROM, and particularly on the plantar flexion of the patient (Fig. 13.1) [9].

To prepare the receiving site and the graft, the same surgical steps that we will describe for the open techniques are followed.

13.4 Open Techniques

13.4.1 Surgical Approach

Anterolateral arthrotomy with or without release of the anterior talofibular ligament is indicated for lateral lesions. Osteotomy of the peroneal malleolus is also described, but it is necessary only on rare occasions.

The surgical approach is usually arthrotomic for anteromedial lesions; in theese cases an osteotomy of the medial malleolus is generally required.

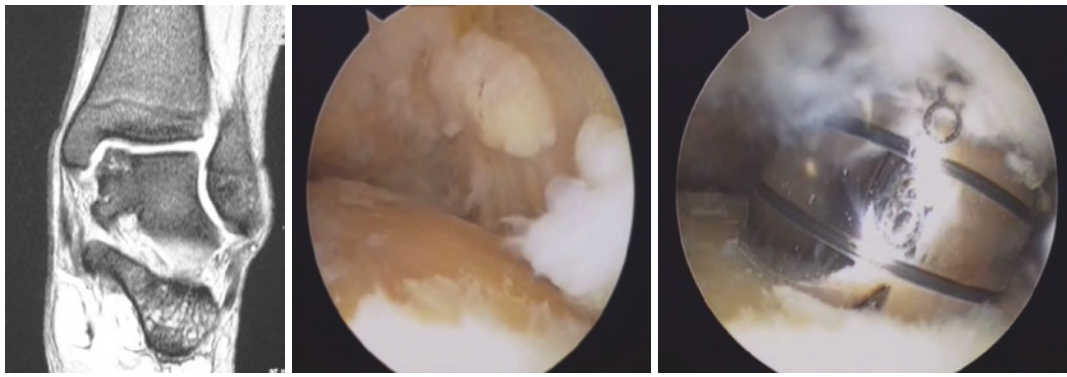

Fig. 13.1 Surgical arthroscopic steps (RM: antero medial lesion; arthroscopic view of the lesion; preparation of the lesion with OATS instruments)

13.4.2 Tricks and Tips of Osteotomy of the Medial Malleolus

A longitudinal surgical incision centred on the medial malleolus is performed. The periosteum is exposed and using a Homan retractor, the posterior tibial tendon is protected posteriorly. It is recommended to drill one or two holes for the screws that will be used for the final synthesis of the malleolus, before performing the osteotomy. This step offers two advantages: it makes the subsequent anatomical osteosynthesis easier and lets to use the holes on the medial malleolus to stabilise it with a Kirschner wire on the talus after the osteotomy is performed, to optimally expose the talar dome (Fig. 13.2).

The osteotomy is performed with an oscillating saw and completed with osteotome at 30° slant as to the vertical plane. We recommend performing the procedure under scopic control after positioning K-wire that serves as a marker. Osteotomy of the medial malleolus is performed horizontal or with self-stabilising 'inverted V-shape'. We recommend using the 'inverted V-shape' configuration, because it considerably helps the subsequent osteosynthesis. The osteotomy should reach the articular surface at the passage from the tibial plafond to the medial malleolus. The top of the 'V' is represented by the entry point of the K-wire. The osteotomy will end at the anterior and posterior edges of the malleolus. At the end of the procedure on the talus, the synthesis will take place with one or two cannulated screws using the pre-drilled holes (Fig. 13.3).

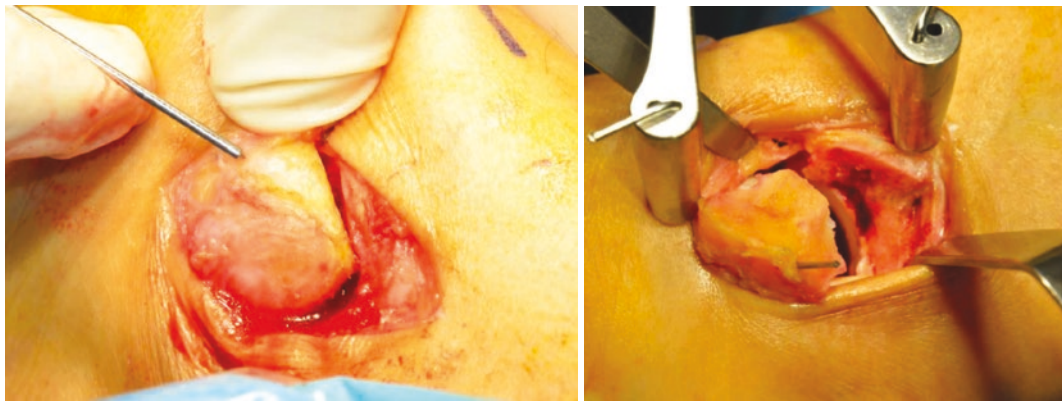

Fig. 13.2 'Inverted V-shape' osteotomy of the medial malleolus, use of the screw hole for the temporary stabilisation of the malleolus on the talus bone

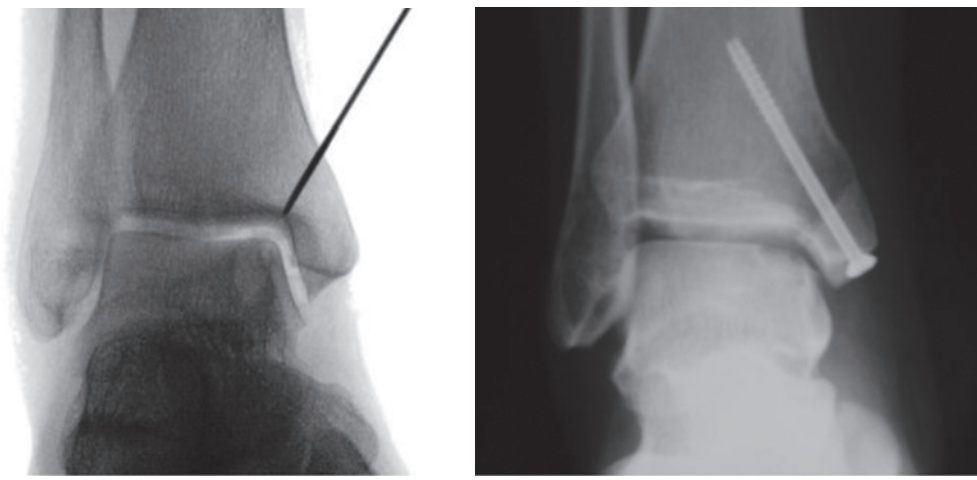

Fig. 13.3 Wire marking the osteotomy of the medial-malleolus; synthesis with cannulated screw

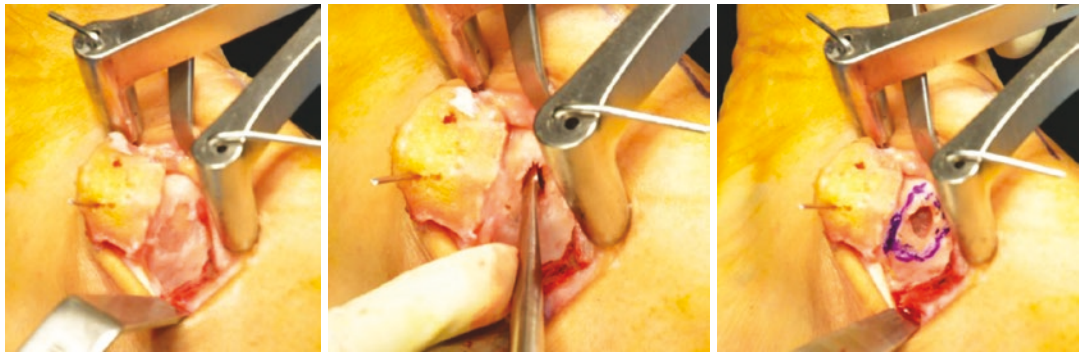

Fig. 13.4 Exposure of the lesion with Hintermann retractor; lesion preparation

13.4.3 Preparing the Lesion

To optimise exposure of the lesion, an Hintermann's retractor that allows distraction of the tibiotalar joint is very useful. The lesion must be prepared by removing the pathological osteocartilage with curettes and osteotomes until the vital subchondral bone is exposed and making the edges of the lesion as regular as possible to facilitate the subsequent positioning of the transplant. It is advisable to create a graft site delimited by four walls in order to obtain, if possible, a primary press-fit stability.

After the lesion is prepared, the next step is its measurement (Fig. 13.4).

13.4.4 Preparing the Allograft

13.4.4.1 Small 'Contained' Lesions

The depth of the transplant must be no more than 10 mm to optimise the probabilities of osteointegration. If the lesion is small in size and does not involve the 'shoulder', then an OATS type instrument can be used to prepare the receiving site and for the harvesting procedure of graft. OATS Arthrex® instruments allow specimens up to 10 mm in diameter to be taken. Corers of the same measurement as those used to prepare the lesion site are used. The depth of the harvesting site must be 1–2 mm less than the depth of the receiving site in order to prevent the graft from protruding into the joint. The receiving site is enlarged by about 0.1–0.2 mm with dedicated instruments (cylindrical hammer) to help the subsequent positioning performed with press-fit fixation (Fig. 13.5) [10, 11].

13.4.4.2 Marginal Lesions Involving the Shoulder or Larger in Size (>10 mm)

The main indication for using the allograft is the presence of larger lesions involving the shoulder of the talus. To improve the articular congruency we suggest to harvest the graft in the same site of donor talus corresponding to that of the original lesion, it will reproduce the most similar anatomy of damaged area. The size of the graft must be equal to that of the receiving site in order to permit a good press-fit at the time of positioning whereas the graft's depth should be 1–2 mm less in order to prevent the graft from protruding from the surrounding cartilage surface (Fig. 13.6).

13.4.5 Transplant Fixation

The transplant fixation techniques require perfect congruency and therefore a press-fit implant is always preferable. In case of marginal transplants, a subsequent fixation with re-absorbable pins or compression screws is often necessary, in these cases we prefer re-absorbable means of fixation to avoid their removal. When metallic fixation devices are used, X-Ray fluoroscopy is useful to check their correct positioning (Fig. 13.7).

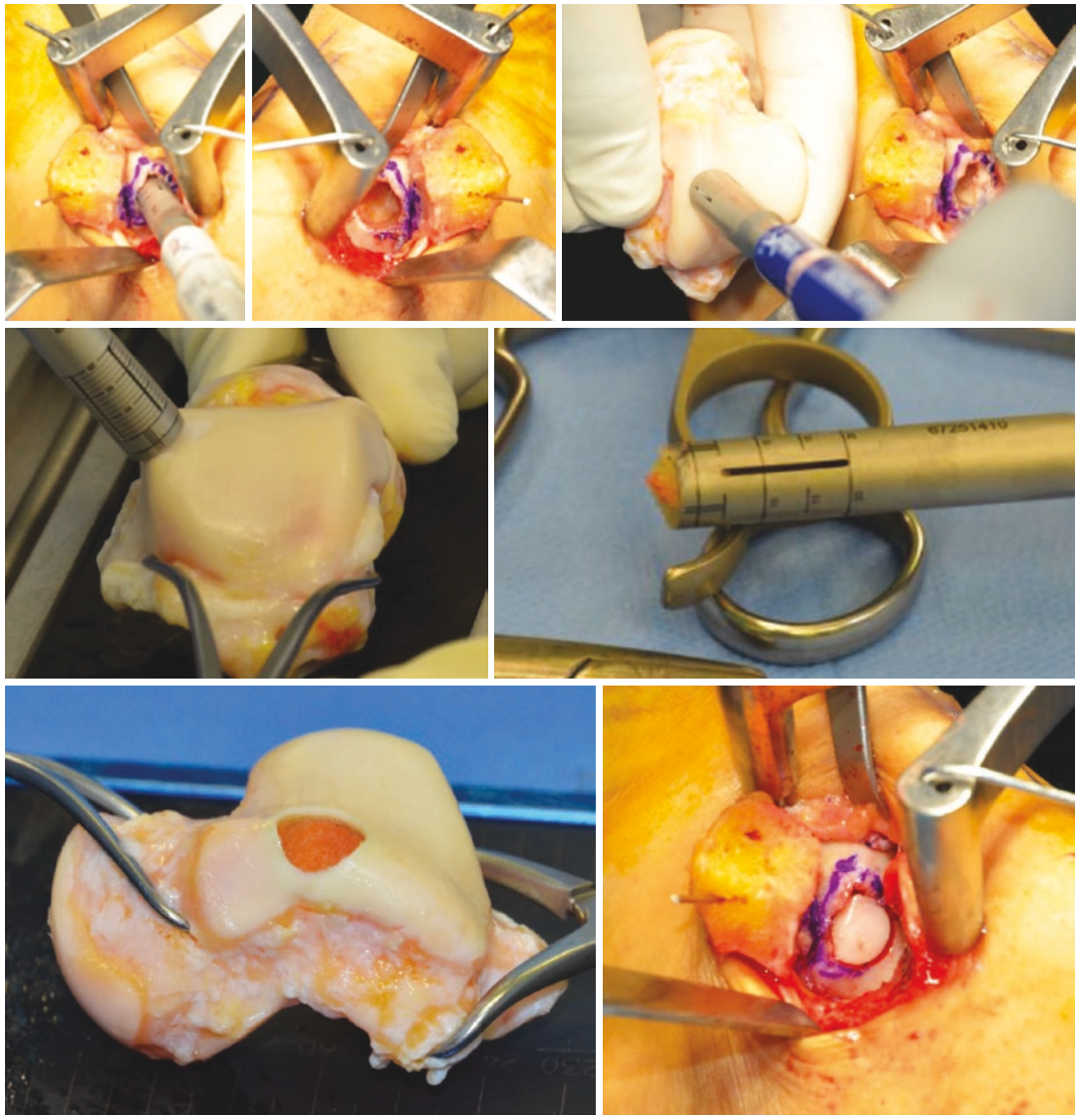

Fig. 13.5 Intraoperative images of *allograft* with OATS instruments for cylindrical lesion

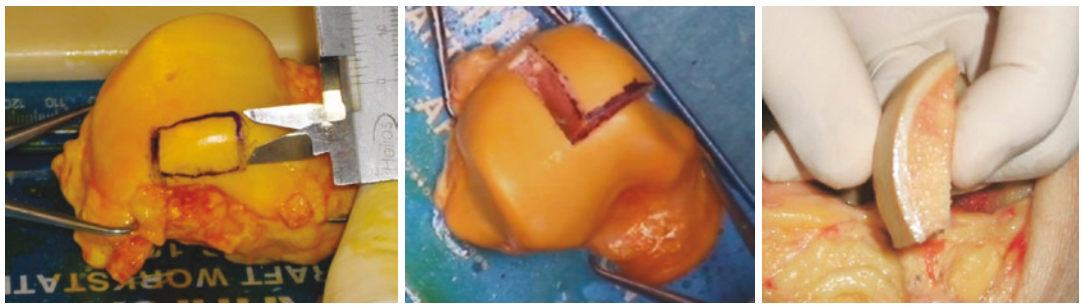

Fig. 13.6 Intraoperative images of *allograft* preparation; for the marginal lesions, the transplant is prepared in the site and of the shape corresponding to the lesion

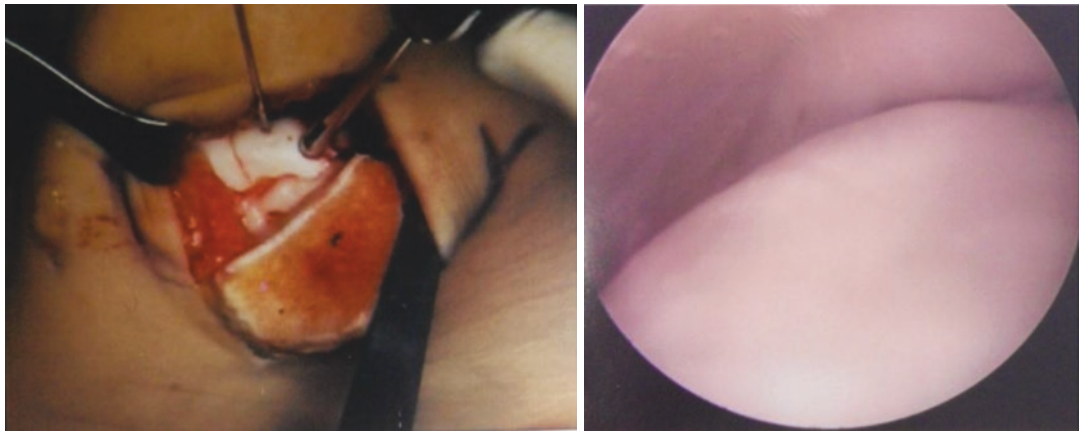

Fig. 13.7 Fixation of the transplant with re-absorbable pins; second arthroscopic look 18 months later

13.4.6 Post-op Treatment

Deambulation without weight bearing for 4 weeks in position valve; 6 weeks in case of osteotomy of the medial malleolus. Partial weight bearing for further 3 weeks, with walker boot. From week 4 exercises to resume use of joints will be suggested. From week 8, has to be performed fisiokinesitherapy to recover proprioceptivity. Return to sports is suggested only after 12 months from the grafting procedure.

13.5 Results in Literature

Many studies report the results of use of similar osteochondral transplants from fresh cadavers at knee level. [5, 12–16]

The studies that examine the results of the *allografts* at talar bone level are fewer and refer to a limited number of patients. The results are generally good, with functional improvement [17–19]. Berger et al. show not only an improvement in quality of life, but also the actual osteointegration with the MR control 2 years later [19].

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Part V

Instability of Ankle and Subtalar Joint

14 Classification, Treatment, and Arthroscopic Procedures

Alberto Branca, Marco Merlo, Clara Terzaghi, and Cecilia Pasquali

Ankle sprains represent one of the most frequent injuries occurred during the participation in sport activities. In the United States of America, 23,000 ankle sprains occur every day, with an annual injury rate of 12.8 per 1000 inhabitants [1].

The pain with conservative treatment decreases progressively with full functional recovery and pain remission of 36% in 2 weeks and 85% after 36 months [2].

Reports show great variability with subjective instability values varying between 5% and 33%.

The ligaments in the lateral compartment are more often involved; however, they often have medial lesions in the deltoid ligament or anterolateral injury in the bifurcate ligament [3].

These types of lesions, which are connected to trauma mechanisms and to the forces of the trauma, often exist in complex laxity of the ankle, and have the tendency to have more complications than simple lateral instabilities.

During the study of an unstable ankle, the predisposing factors related to the instabilities and other associated lesions must be taken into consideration: anterior and posterior impingement, osteochondral lesions, or lesions of the peroneal tendons.

14.1 Introduction

Ankle sprain represents one of the most frequent injury, which occurs during sporting and nonsporting activities, with a frequency of 23,000 daily events in the United States of America, with an annual injury rate of 12.8 per 1000 inhabitants [1, 2].

In regards to people with a sedentary lifestyle, this problem may be scarcely relevant, while for patients with an elevated functional request such as sport or certain types of employment, it could be a cause of important inability. The lesions can be classified based on the ligament compartment affected: medial ankle sprain, lateral ankle sprain, or the tibio-peroneal syndesmosis lesion.

The anatomical structures of the lateral compartment are represented by the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL).

The most frequent traumatic mechanism is the internal rotation of the plantar flexed foot.

In fact, due to the particular morphology of the ankle joint (the anterior diameter is greater than

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the posterior diameter), during this movement, the trochlea is free to tilt within the tibio-fibular mortise, giving this joint the most instability.

In addition, the conical form of the articular talar surfaces with the medial apex and the lateral base, favors during the supination, the anterior positioning of the articular surfaces accentuating the movement of inversion even more. The lateral compartment is progressively involved in this dynamics.

The ATFL is the first ligament injured, tightly connected to the capsule, less frequently the CFL, while the PTFL is rarely injured [4, 5].

14.2 Classification

A widely used classification to describe the lateral lesions is that of Hamilton, which distinguishes three levels of gravity:

- 1° grade: ATFL elongation without talar tilt and negative anterior drawer test
- 2° grade: ATFL lesion, CFL elongation with talar tilt and positive anterior drawer test (1–2° grade)
- 3° grade: ATFL and CFL lesion with important talar tilt and very positive drawer test [5]

Other classifications are present in literature with different applications [6].

One which is often used, however, is the Lanzetta classification and distinguishes four levels of gravity 0–1–2–3. This classification is based on the relationship between the morbid-pathological damage and the X-rays under stress (Table 14.1).

The anatomical structures of the medial compartment are represented by a triangular-shaped ligament, commonly known as the deltoid ligament, which originates from the malleolar apex and expands in two layers: the superficial one and the deep one.

The superficial ligament is attached to the calcaneus with the most robust component and to the talus. The deep ligament separated from the superficial one and has an anterior and a posterior component.

Conceptually, the superficial layer provides stability to the naviculocalcanear complex and resists external rotation of the talus relative to the tibia.

The deep layer assists in stabilizing the talus and resists posterior translation of the talus, valgus angulation of the talus, and lateral displacement of the talus from the medial malleolus.

The lesions of the deltoid ligament have been classified by Myerson in four degrees:

- 1°: Superficial deltoid ligament distraction
- 2°:Superficial deltoid ligament rupture and deep deltoid ligament distraction
- 3°: Superficial and deep deltoid ligament rupture
- 4°: Superficial and deep deltoid ligament rupture and posterior tibial tendon rupture [7]

The peroneus muscles assist the ankle ligaments action as static stabilizers, and they contrast the traumas in inversion.

The severity of the damage depends on varying factors such as the type of trauma (high and low impact), the ankle position at the moment of trauma (supination or pronation, dorsiflexion or

Table 14.1 Lanzetta classification

Degree	Signs and symptoms	Dynamic X-ray in Varus stress	Dynamic X-ray anteropulsion, mm	Pathological damage
0	Mild tenderness and swelling, malleolar pain	10°	5	No ligament lesions
1	Moderate pain and swelling with cracking, hematoma, pain with weight bearing	10–15°	8	Isolated ATFL lesion
2	Important hematoma, lateral tilt, claudicating walking	20–25°	10–15	ATFL, CFL, PTFL lesion
3	Swelling and Hematoma, pain in varism, Drawer test +, severe positivity of clinical stress examination	30°	15	ATFL, CFL, PTFL and syndesmosis lesion

plantarflexion), predisposing anatomical factors, or functional factors.

Scranton cites as other instability factors the proprioceptive deficits, insufficient function of the peroneus muscles, the varus of the tibial plateau or the varus of the foot, and the posterior position of the fibula [8].

The chronic sprain syndrome takes place after many ankle sprains, which, over time, bring chronic pain and persistent swelling, ankle stiffness, and gait instability, particularly on uneven terrain.

Intermittent episodes of sprain frequently reoccur over shortening periods of time and with functional recovery becoming longer and disappointing. Sporting activities are given up entirely or carried out at an inferior level. Walking on steep terrain becomes difficult, not infrequently the improper walking leads to knee pain, hip pain, or lumbar pain.

14.3 Treatment

14.3.1 Patient Evaluation

The best therapeutic choice can only be made after a through clinical assessment of the patient and after having collected a detailed medical history focusing on the number of events, with a perception of a "crack," on sporting activities done, or not done.

The examination includes the observation of the static and the dynamic ankle assessment.

During inspection, it is necessary to evaluate the presence of alterations in the anatomical profile, the presence of bruising, ematoma, or retromalleolar edema.

By palpation, it is possible to look for marker points on bone or ligaments, tendons, and joint.

It is important to check the medial and lateral malleolus, the distal tibio-peroneal joint, the distal tibial epiphysis, the neck of the talus with the insertion of the capsula, the sinus tarsi, the deltoid ligament, the posterior talofibular ligament, the anterior talofibular ligament, the peroneal tendons, and the peroneus brevis tendon insertion.

In the instability assessment of the ankle, dynamic trials are fundamental, the execution of which is often extremely difficult in acute trauma. The tests of the lateral ligament complex more often used in the clinical practice are the anterior drawer test, wherein the ankle joint is held in 10° plantar flexion and the clinician presses the heel forward while holding back the tibia; the talar tilt test, wherein the ankle joint is held in neutral position, the heel is held stable while inverting the talus and the calcaneus on the tibia (Fig. 14.1).

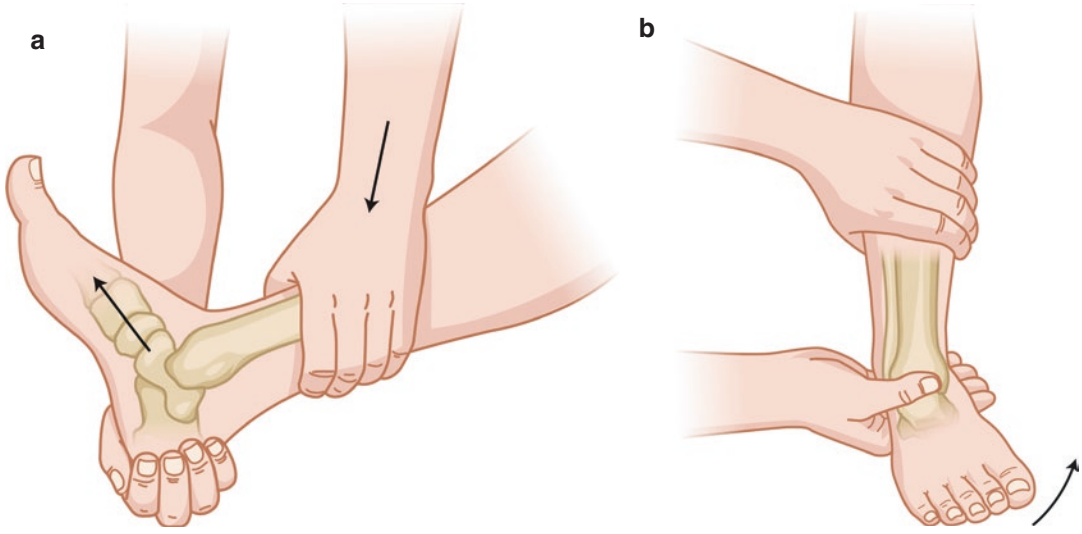

Fig. 14.1 Dynamic ankle assessment: (**a**) the anterior Drawer test for the ATFL (**b**) the talar tilt test for the ATFL and CFL. Illustration by Tomaso Baj

The external rotation test, the "squeeze test" and the cross-leg test are used to evaluate the sindesmosys.

After an appropriate clinical assessment, according to Ottawa Ankle Rule (OAR) follows the imaging with standard X-ray in frontal and lateral position and with a 15° external rotation of the foot [9].

Second-level image includes the Computerized Tomography (TC), and the Nuclear Magnetic Resonance (RMN), the first one identifies small bony and osteochondral fragments, and the second one indentifies in a more accurate way, ligament capsule lesions, as well as chondral and tendons tears (Fig. 14.2).

Treatments after the correct diagnosis can be the following:

- Conservative-functional treatment
- Surgical treatment
- Arthroscopic treatment

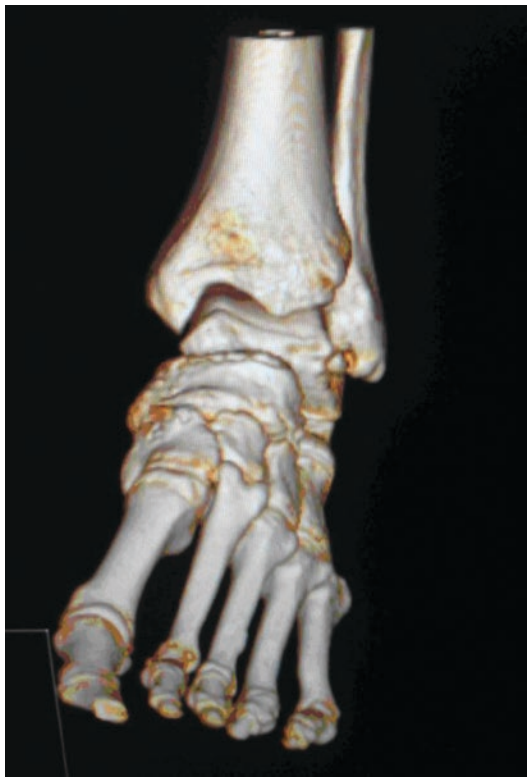

Fig. 14.2 Three-dimensional reconstruction in computed tomography

Reports state that in 80–90% of cases there are positive results using the conservative treatment; in 10–20% of cases, important symptoms remained with possible development of osteoarthritis in the most severe cases.

Second- and third-degree lesions, if not adequately treated, can leave anatomical and functional outcomes predisposing chronic ankle instability.

Some authors, since the 1990s, proposed the arthroscopy in acute ankle sprain [10–14].

In a recent review of the findings, Chaudhry et al. have stated that a significant difference has not been found between conservative treatment and surgical treatment by way of costs and the possibility of complications, as the eventual secondary reconstruction of the ligament rupture has the same results as the primary one [15–17].

In reference to reports, our protocol in the use of arthroscopy in acute ankle sprain includes:

– Young and sporty patients
– 2°/3° sprain
– Reassessment of the ankle 7/10 days after the trauma

Aim:

– Washing of the hemarthrosis and blood clots (Fig. 14.3)
– Views of ligament and capsular lesions
– Identification and treatment of osteochondral lesions (Fig. 14.4)

14.4 Arthroscopic Procedures

Technical details for surgery:

- The patient is in supine position, no traction, irrigation to maintain sufficient intra-articular pressure can be obtained using saline or Ringer solution using a gravity drip at a height of approximately 2 m; positioned on the patient's head; the irrigation occurs due to gravity or in rare cases, by using an arthroscopic infusion pump, in which case the pressure must be between 40 and 50 mm.

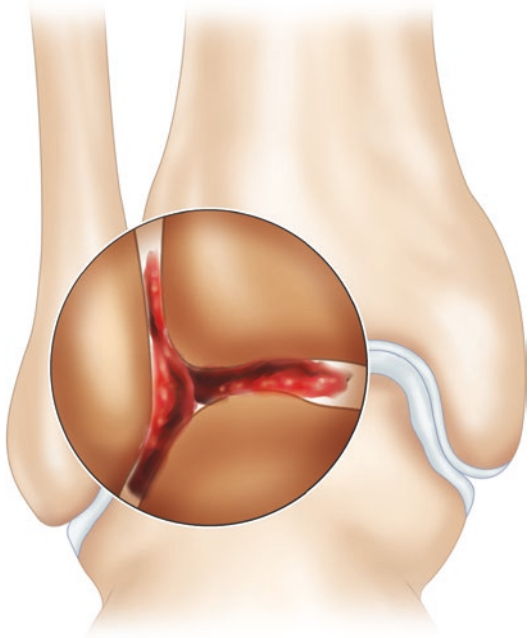

Fig. 14.3 Hemarthrosis

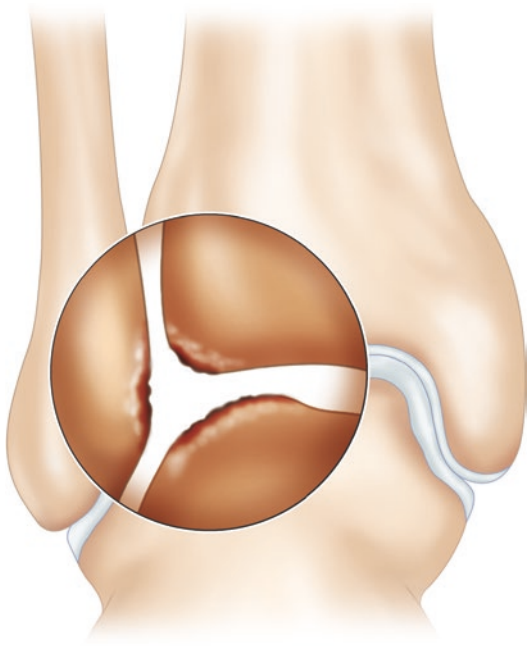

Fig. 14.4 Osteochondral lesions

The basic instrumentation includes a 4, 5 mm diameter and 30° arthroscope, a straight graduated probe, an electrocautering device, and a small joint shaver.

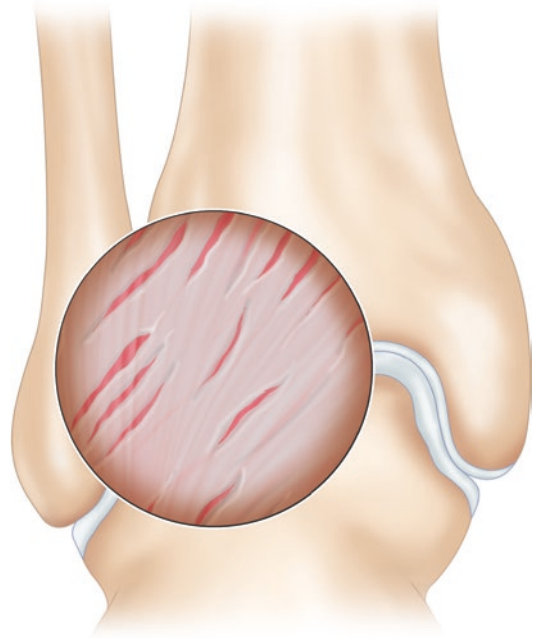

Fig. 14.5 Scarring

Once irrigated and inspected the joint from the anteromedial portal, the procedure continues with the execution of the anterolateral portal under direct control and transillumination.

We proceed with exploration of the joint, evaluating the cartilage lesions, the bones, the ligaments, and the capsule. During arthroscopy, it is then possible to remove loose bodies, remove blood clots which can lead to scarring (Fig. 14.5), eventually correcting the anterior osteophyte.

These lesions often with concomitance of other lesions regarding ligaments: syndesmosis, deltoid ligament, subtalar ligaments; bone and cartilage; regarding peroneal tendons (Fig. 14.6a, b).

A frequent damage in the medial compartment on the tibial and talar cartilage surface may occur in the majority of cases when the deltoid ligament has been injured [18].

The postsurgical recommendations are the following:

- Keeping the sterile dressing for 7 days and removing the stitches after a week.

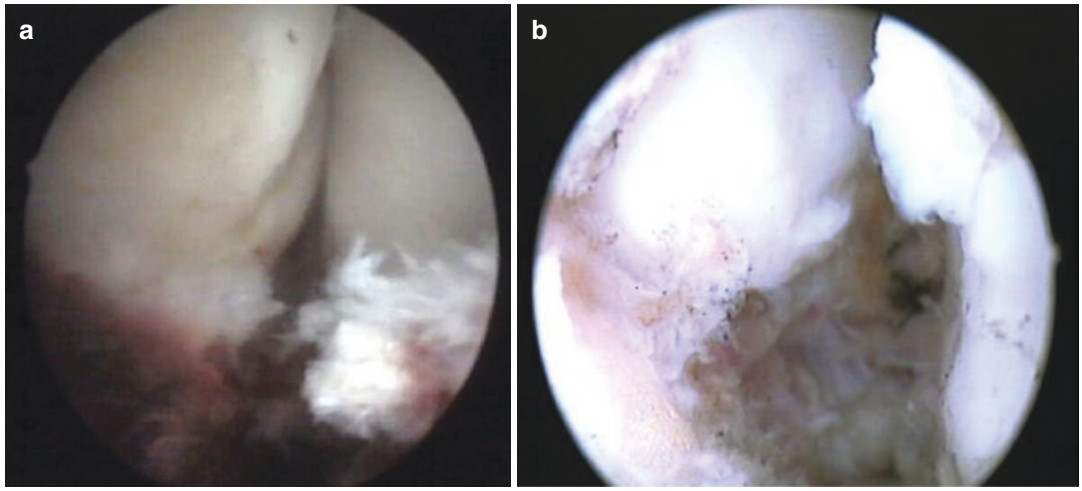

Fig. 14.6 (**a**) ATFL lesion. (**b**) Deltoid ligament lesion

Postoperatively, the leg is held in elevated position with an ice bag on the ankle for a few minutes every six hours each day. The patient is immediately able to start passive dorsiflexion and plantarflexion; the use of a brace prevents other movements: inversion ad eversion.

- After 4 weeks, the patient is able to start active physiotherapy, and proprioceptive and muscle-toning exercises.
- The partial weight bearing is allowed after the first week, then the patient can progressively abandon crutches.
- The daily activity is undertaken after 1 month, then the competitive sport activity after 3 months, depending on the severity of injury.
- Other surgical techniques are possible during arthroscopy, one of which is described by Hawkins: with this technique, the anterior talofibular ligament is retensioned and fixed to the talus with a cambra or with a small anchor, as proposed by Ferkel [17, 19, 20].

14.5 Conclusion

Our experience and past and present literature encourages the use of arthroscopy in acute trauma: it guarantees washing and removal of blood clots (cause of successive formation of adherence), it permits a rapid recovery or the range of motion, reducing the immobilization time.

The early diagnosis and treatment of cartilage defects allows a reduced joint degeneration and the subsequent ankle osteoarthritis.

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15 Instability of Ankle and Subtalar Joint

Alberto Ventura

Ankle instability treatments have recently been changed as a result of new arthroscopic techniques, which are as efficient as the traditional treatments.

Surgery treatment for ankle instability has increased in recent years, bringing new arthroscopic tenodesis techniques and new arthroscopic Brostrom techniques. The success of these new techniques is related to their good results, patient satisfaction, and the easiness of the surgical procedure.

In 1960, Broström [1] proposed his anatomic replacement technique. This approach was completely changed by Gould [2] in terms of results. The Broström-Gould technique is a nonanatomic treatment that became the gold standard in this pathology. At the end of the twentieth century, ankle instability had been treated with either the anatomic technique (Broström) or nonanatomic techniques (Broström-Gould or tenodesis).

At the beginning of the arthroscopic era, ankle instability had not been taken into consideration, but only outcomes had been considered. The first arthroscopic techniques proposed using cambers or metal anchors to treat only the anterior talofibular ligament.

In the past 15 years, interest has grown, followed by improvement of surgical treatments and the successful results.

In 2001, Oloff [3] published the successful results of lateral capsule shrinkage; other authors [4–6] then published their work and results in support of this technique.

From 2005, other work and successful results [7–10] supported the arthroscopic Broström technique instead. The surgeries utilized are as follows: repairing the articular capsule, repairing the Anterior Talo-Fibular Ligament (ATFL), and transposing the lateral retinaculum on the peroneal malleolus with anchors.

In the same period, other authors considered the anatomic replacement of the ATFL and calcaneo fibular ligament (CFL) with autografts and allografts [11–13]: these perfectly replaced the anatomic ligaments in terms of function. The results were positive; however, the technique and its learning curve were not easy.

Currently, arthroscopic techniques are limited by the vascular and nerve structures. Nerves that follow nonstandard paths could limit the site of treatment and cause uncomfortable neurological lesions for the patients.

The recent diffusion of ankle arthroscopy in acute lesions has helped us to better understand some problems linked to the failure of instability treatments. The tibiofibular joint has a key role in ankle instability treatment: the opening of the tibiomalleolar clamp changes the ankle biomechanics significantly and creates ligament laxity. A major

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problem is the fibula-tibia lesion without diastasis because it is not recognized during the usual clinical investigation and so is not treated. Additionally, in at least 30% of the cases, an ankle sprain causes a subtalar joint lesion with ligament laxity.

For the foregoing reasons, ankle instability treatment has to be revised and bring us from the past when it was clear that each ankle joint could not be treated separately because these are involved in a unique function. The conclusion is that there are many kinds of instability that must be treated with different surgical techniques.

An ankle instability classification, based on functional anatomic criteria (Fig. 15.1), has been developed by the Ankle Faculty of Arthroscopy Italian Society. A therapeutic algorithm has been implemented (Fig. 15.2).

Tenodesis was a valid method until the arthroscopy era because it permitted stabilizing the laxity of the three joints simultaneously. Now, authors are aware that tenodesis causes long-term joint degeneration because of the biomechanical modifications caused by the nonanatomic replacement. The Broström-Gould open approach is a good choice for all kinds of instability. The real advantage of this technique is the subtalar joint stability achieved by the inferior retinaculum, which must be considered as a subtalar ligament. The anatomic techniques permit avoiding degenerative results but require a more difficult surgery process and could not resolve the instability without an additional replacement.

In a recent review [14], the open and the arthroscopy techniques were compared. The

Fig. 15.1 Ankle Instability classification

INSTABILITY CLASSIFICATION

The ankle chronic post-traumatic lateral instability without medial lesion and without sindesmosis lesion.

1) Light instability
   a) External instability of the ankle by six months at least
   b) Lateral laxity with a positive anterior drawer test for the talus ligament
   c) Telos with difference to the non-pathological contralateral ankle of:
      i) > 5 mm anterior talus translation on tibia in Lateral view
      ii) < 10° external angolo di apertura in anterior posterior view
   d) Either positive RMN nor positive ECO for ATFL lesion

2) Moderate instability: all the previous criteria plus one of the following ones:
   a) talus tilt test positive
   b) Telos with difference to the non-pathological contralateral ankle of:
      i) > 10° external open angle in anterior posterior view
      ii) > 5 mm of anterior translations of the talus on the tibia in Lateral view
   c) Either positive RMN nor positive ECO for the ATFL and CFL ligaments.

3) Severe instability: all the previous parameters plus at least one of the following ones:
   a) calcaneus varus/escurvatus, cavus, 1st vertical radius
   b) subtalar instability
   c) peroneal tendons with lesions
   d) excessive extra rotation of the intermalleolar axis
   e) either positive RMN nor positive ECO for the peroneal tendinous.

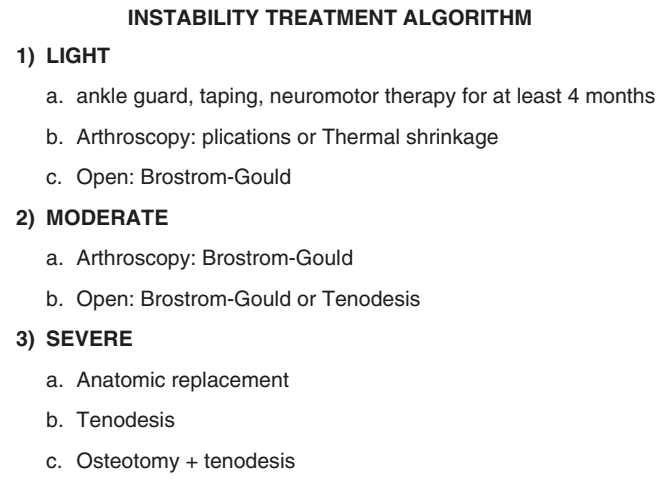

Fig. 15.2 Instability treatment algorithm

conclusion was that both are successful in the short term and mid-long term. The more reliable arthroscopy techniques are the thermal shrinkage, the Broström, and anatomic reconstruction.

Now arthroscopy is irreplaceable: it optimizes the precision of surgery and minimizes the surgical invasion. The real risk is to give more attention to the technique itself instead of overall ankle wellness.

In general, using different treatment types to stabilize the ankle joint completely could be the optimal approach.

In the near future, use of the open techniques will be reduced and the application of arthroscopic techniques will be increased. As for knee and shoulder joints, ankle instability will be treated exclusively with arthroscopy.

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16 All Inside Broström Arthroscopic Procedure

Francesco Allegra, Edoardo Barberini, and Stefano Carli

16.1 Background

Lateral ankle sprains account for 85% of all injuries to the ankle [1]. After injury, 80% of patients recover completely, but 20% develop recurrent lateral ankle instability [2]. A recent anatomic study [3] points out that the anterior talofibular ligament (ATFL) is divided into two separate fascicles, distinct structures both anatomically and functionally. The two distinct portions have different placement in the joint; the proximal one is intrarticular whereas the other is extrarticular, connected to the calcaneofibular ligament (CFL) by arciform fibers in a whole ligament complex. The clinical relevance is the loss of capability to heal the torn intrarticular fascicle, which leads to microinstability of the joint. Moreover, a lesion that involves the extrarticular ligament complex develops a frank ankle instability which in time becomes chronic. In the long term, chronic joint instability may predispose to the occurrence of degenerative changes [4]. In patients unresponsive to conservative management, surgery is indicated. Anatomic and nonanatomic repair and reconstruction procedures may be performed. In anatomic repair techniques, such as the Broström procedure [5, 6] and the Gould modification [7], local soft tissues are used to restore the anterior talofibular ligament. In nonanatomic procedures [8, 9], close tendons may be used to restore and augment the repaired anatomy, rendering these techniques more invasive and possibly resulting in both ankle and subtalar joint stiffness. Anatomic procedures for lateral ligament reconstruction respect the footprint [12] regardless of the techniques [7, 9–11] and grafts [2, 3, 11, 12] used, mostly reporting good results [7, 9, 13–16]. A recent review [17] on chronic ankle instability treatment shows the good outcome and efficacy of both open and arthroscopic surgical techniques, despite the higher complication rate of the latter. This higher complication rate represents the major issue for arthroscopy, despite which it remains strongly suggested for treatment of hidden ancillary disorders and although this does not seem to affect patient satisfaction.

16.2 Indication

Technological progress overcame the many difficulties in exploring such a narrow space until it was possible to raise arthroscopy to being a widely used procedure. Recently, satisfying outcomes have been reported, also in the long term, after anatomic repair combined with arthroscopy. This approach is minimally invasive, and allows us to successfully manage associated conditions

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[12, 14] such as loose bodies, malleolar ossicles, osteochondral defects, bony cystic lesions, soft-tissue impingement, scar tissue, and bony spurs. The Broström repair [5, 6] is the method of choice for primary operative treatment for acute and subchronic ankle instability, using the traditional open procedure to identify and restore the elongated anterior talofibular ligament. Gould modified [7] this by mobilizing and attaching the lateral portion of the extensor retinaculum to the distal fibula over ligament repair. Recently, some surgeons [18] proposed to perform this procedure by arthroscopic technique, adding an inferior anterolateral portal to the traditional ones for completing all the steps by scope. The arthroscopic Broström repair is a procedure that requires the proper indication as in cases of total detachment of the intraarticular portion of the anterior talofibular ligament (ATFL). Its residue on the peroneal side appears without any continuity from the talus footprint to the fibular one, which is totally bare. After the debridement of scar tissue to identify the whole ligament and its detached side, a suturing instrument can be introduced to hold it and to approach to the bone in the correct placement. Exploring the lateral compartment of the joint inferiorly and posteriorly, the calcaneofibular ligament (CFL) should be absent or insufficient as ligament texture or tension. In such cases is suggested to add the Gould procedure to the former one, shifting the retinacula of extensors on the repaired ATFL.

16.3 Surgical Technique

The patient is assessed in supine position with a tight tourniquet. The foot is placed on the end of the surgical bed. An ankle traction system is recommended to make the arthroscopic steps easier. Two anterior standard portals are initially recommended to proceed inside the joint and to permit an accurate exploration. To improve visualization, removal of soft tissue completely frees the articular surfaces from synovial coverage. The presence of some ancillary disorders caused by joint instability is better detected by dynamic evaluation of joint spaces and gutters, by moving to the ankle. Loose bodies and cartilage or bony fragments are removed with a grasper. Seldom, the presence of anterior tibial osteophytes, such as talar ones, requires a resection to better access the joint. Identification of the ligament lesion is led out in the lateral compartment, exploring both anterior and inferior gutters, after sinoviectomy if necessary. The absence of the AFTL confirms the pathology, exposing its bare footprint (Fig. 16.1). Furthermore, in about 30% of cases, the lesions of the CFL are seen on the tip of the fibular malleolus or on the calcaneal footprint side. The ATFL can appear detached from the anteroinferior surface of the fibular malleolus: mobilizing with a suture-retrieval device, it is reduced and fixed in the proper area by anchor suture. Further technical variations have been developed with the arthroscopic procedure, providing some adequate tissue has remained and possibly sutured again by side-to-side stitches. The bone refreshment of the fibular footprint is first performed with a shaver blade, removing the scar tissue and preparing the bony surface on which to fix the repaired soft tissues. After these steps, a spinal needle is inserted through the skin to the ligament footprint, accurately evaluating the correct direction with the aim of a new inferior portal opening (Fig. 16.2). This portal placement is essential because it permits the right direction of the

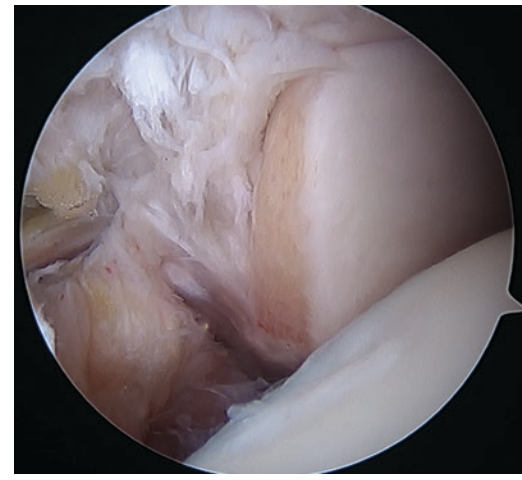

Fig. 16.1 Because the anterior talofibular ligament (ATFL) is completely detached, its footprint appears completely bare

anchor guide, 45° inclined on the bone surface, possibly in the footprint center and far from the malleolus tip. The anchor aimer is placed in this portal and gently pushed on the refreshed bone: one or two small holes are then drilled on the fibular malleolus surface to create the recipient socket for a provided number of anchors. After anchor placement by mallet inside their socket, the sutures are retrieved outside the joint (Fig. 16.3), toward the anterolateral portal or a new anterosuperior one, opened along Harty's notch by a spinal needle introduced from the proximal to distal side. A suture passer engages the tissue of ATFL and grasps the stitches, retrieving them outside. Depending how many anchors have been used, 2–4 sutures ensure the fixation of the ligament to its fibular footprint. A knot pusher completes the procedure by tying proper knots on the tissue to obtain a stiff repair (Fig. 16.4).

However, in cases in which a residual lateral ankle instability remains, an added factor of joint instability is revealed by CFL insufficiency. The Broström repair just described could be not sufficient to make a valid stabilization of the ankle. The Gould modification makes the lateral compartment stiffer by pulling the retinacula extensors closer to the lateral malleolus and by suturing it on the repaired ATFL ligament. Because its distal insertion, placed on the proximal-lateral side of the calcaneal lateral surface, is shifted in a more proximal and anterior direction, the Gould technique permits a new tension on lateral injured ligaments, increasing the lateral ankle stability. The first surgical step is to detect the retinaculum to ensure its engagement, because a suture punch

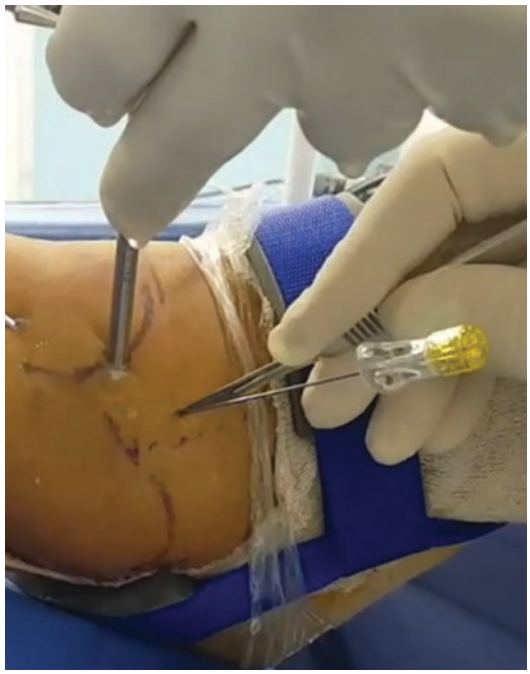

Fig. 16.2 The anterior inferior lateral portal is opened, along a needle placed under direct visualization, to choose the correct introduction of instruments aiming at the footprint

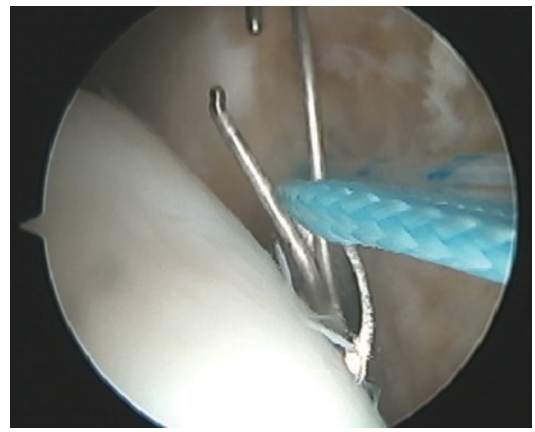

Fig. 16.3 The suture strand is secured by a suture retrieval device after penetrating the residual ATFL

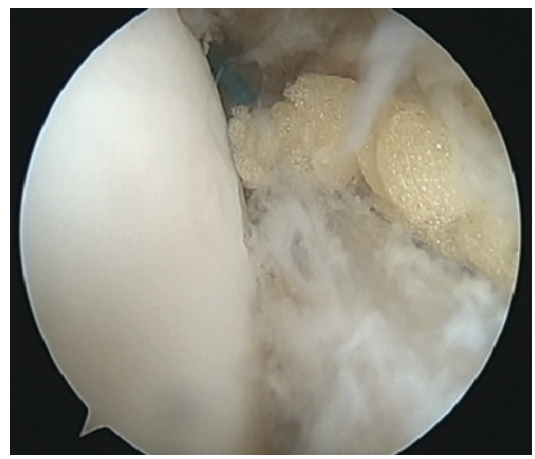

Fig. 16.4 The final aspect of the ATFL repair in its proper placement after knot tightening

is introduced from the inferior portal to pass through the retinaculum with the aim of reaching the lateral joint, and the risk could be high to involve some small branches of lateral sensorial skin nerves placed in the subcutaneous layer. However, some difficulties are described [15] in checking its exact placement under the skin, and the possibility of missing engaging the tissue is not uncommon. At the beginning of the learning curve of this surgery, it is suggested to be able to visualize the retinaculum with the scope placed under the subcutaneous layer of the skin and to penetrate the retinaculum under direct view. From the inferior portal is introduced a bird-beak punch or a suture passer, to pass through the retinaculum and to reach the joint. Then, the stitches of a new anchor suture are grasped directly by punch or indirectly by a suture shuttle passed by a suitable device. The sutures are pulled on and the knots are tightened outside the joint under the skin. A tape bandage completes the procedure. The patient is admonished to move his joint; partial weight-bearing assisted with crutches is suggested for 4 weeks until rehabilitation begins, for a variable period of a further 4–8 weeks.

16.4 Conclusions

The arthroscopic Broström repair is a safe and biomechanically valid method to obtain a complete joint stabilization, within the correct indications. It generates good to excellent results in most patients. Prompt soft-tissue healing can be expected, and the repair allows a functional rehabilitation with full weight-bearing. A meticulous knowledge of the retinacular anatomic position, far from the peroneal tendon sheaths, is strongly recommended: the surgeon should be able to engage it with the sutures and to shift it to the fibular malleolus. The possibility of treating unstable ankles with arthroscopy reduces complication rates, with clear cost benefits compared with open surgery, making the arthroscopic Broström a safe and reliable technique.

Traps	Tricks
Low-quality visualization of the joint	Clean up compartments of the joint accurately Use small shaver blades or radiofrequency wands to move into gutters
Difficulties in choosing the placement for the inferior anterolateral portal	Use traction at major load, then release as done Insert spinal needle at all times you need to visualize perfect access and direction
Possible unsafe placement of sutures through retinacula	Check correct placement of the retinaculum exploring the subcutaneous layer by scope Move foot fingers to ensure sutures did not involve tendons Explore by scope subcutaneous layer to identify proximal retinacular edge

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Capsular Plications Repair

17

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17.1 Introduction

Ankle chronic instability is a common and extremely disabling condition in athletes. This instability leads to significant limitations in performance and training programs, and when the instability becomes serious, it can also affect nonathletic people in their daily activities [1].

The anterior talofibular ligament (ATFL) is involved in most cases. The peroneal calcaneal ligament (PC) and the posterior talofibular ligament (PTFL) are involved in 50–75% and less than 10% of cases, respectively [2].

Several predisposing factors can cause different ankle sprains, such as varus midfoot, first plantar-flexed metatarsal, and claw foot [3–5]. Usually these patients present a medical history of ankle sprains characterized by instep inversions. They may complain about persistent pain in the anterolateral portion of the ankle associated with swelling, and they report episodes of joint failure, relapsing hydrarthrosis, analgesic limitation to the plantar flexion of the foot, and functional limitation in ankle dorsiflexion.

This persistent ankle symptomatology persists in approximately 20–25% of the subjects who incur red ankle inversion sprains, even when properly treated, reaching 35–38% if not properly treated [6].

Chronic instability must be distinguished between functional and mechanical. Freeman [7] described the first as a subjective sensation of instability during physical activity. Functional instability causes damage to capsular proprioceptive receptors and muscular hypotrophy, whereas chronic mechanical instability occurs as a result of ligamentous laxity and the pericapsular soft tissues [8, 9]. According to different studies [10–13], conservative treatment and physical therapy can obtain excellent results in patients with functional instability or generalized laxity of the ankle, whereas patients presenting mechanical instability require stabilization surgery, often simultaneously with the treatment of associated diseases that usually concern the cartilage.

Given that only recapture of the athlete's proprioceptive capabilities will allow return to the sport, the recovery of motor skills and coordination is crucial [14]. Devices such as the Delos Postural System or Pro Kin [15] provide visual feedback, with the patient's eyes either open or

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closed, through the special software that allows drawing on the screen to follow the tracks of the predetermined paths. In fact, by moving the platform with the foot, it is possible to verify in real time the accuracy and the committed errors through open or closed cone graphics that represent the patient's rehabilitation progress [16].

Different surgeries, which can be distinguished as anatomic and nonanatomic, have been reported. The anatomic techniques used for acute injuries concern the direct repair of the ligament injury and capsular residues using either ligament tissues or tendon grafts. These procedures present several advantages such as the ease of execution, the possibility of reconstituting the anatomic conformation of the joint, and preservation of subtalar mobility [17, 18].

The nonanatomic procedures includes tenodesis to replace injured ligaments, resulting in a limitation of the ankle and subtalar functional articulation [19–21].

Injuries of the lateral compartment have been classified in several ways, but we believe that the most immediate procedure to encode a therapeutic path is the following [22, 23]:

- Grade I: sprain or partial rupture of the anterior talofibular ligament (ATFL).
- Grade II: complete rupture of the ATFL, with peroneal-calcaneal sprain (PC).
- Grade III: complete rupture of the ATFL and the PC.

The injuries of the medial compartment are classified as follows [23, 24]:

- Stage I: distraction of the superficial deltoid ligament (SD).
- Stage II: rupture of the SD and distraction of the deep deltoid (DD).
- Stage III: rupture of SD and DD.
- Stage IV: rupture of the SD and DD, with rupture of the posterior tibial tendon.

In this chapter, we describe a new method of arthroscopic surgery in (a) subjects not involved in competitive sports who are suffering from minor ankle instability, and (b) active people, or people whose working activity requires prolonged standing, who present instability that is serious but which can benefit from minimally invasive surgery [25].

17.2 Surgical Technique

Our technique of ankle arthroscopy is conducted with locoregional anesthesia, sometimes using a pneumatic tourniquet applied on the thigh. The patient is in supine position on the surgical table. We use an infusion pump, but this surgery is possible even without it, and a 30° arthroscope of 4.5 mm.

The arthroscopy considers the use of the standard anterolateral and anteromedial portals without the use of an ankle distractor to perform inversion and eversion movements in addition to flexion-extension maneuvers.

During the initial diagnostic phase, with the intent to obtain adequate visualization of the articular surface of the talus-tibial and lateral and medial recesses, we do a joint debridement and an accurate synovectomy, which often causes anterior impingement, therefore pain, even using a motorized tool and VAPR. This procedure lengthens the duration of surgery but is essential.

After evaluating the anterolateral portion of the capsule, the radiofrequency terminal is introduced where previously the two ligaments were located. The repeated movements of the radiofrequency terminal in the posteroanterior direction allow us to directly visualize the macroscopic tissue changes. This shrinkage is useful, but not indispensable.

This procedure continues with the second part of the surgery: the plications. This technique involves the use of two or three percutaneous absorbable monofilament (PDS) #1 sutures in the horizontal posteroanterior direction.

Two technical variants are possible. The first technique is performed with an anteromedial arthroscopic control. A #18 needle is introduced at the level of the fibula (Fig. 17.1), very close to the periosteum, where the suture thread is passed, grasped with an arthroscopic forceps from the lateral portal, and then extracted from the anterolateral portal.

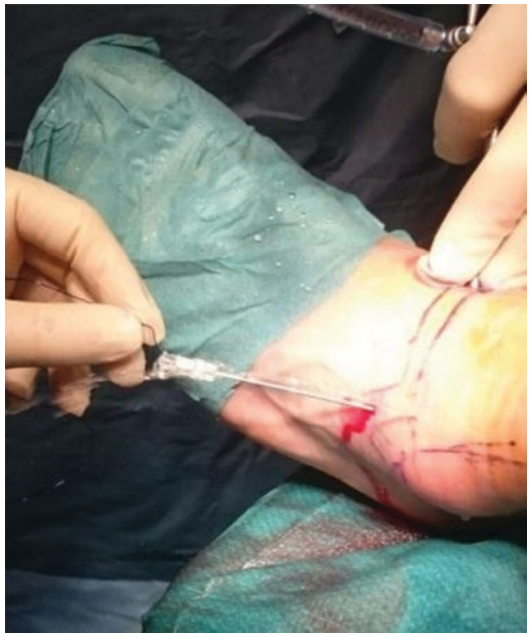

Fig. 17.1 Introduction of needle (number 18) at the level of the fibula

From the same portal, with a subcutaneous Klemmer, the perimalleolar percutaneous thread is retrieved (Fig. 17.2a, b) and, via a pusher (Fig. 17.3), the knot is executed that plics the capsule–ligament component, previously blood-stained, with the motorized tool by directly controlling tensions with ankle mobilizations. The knot may be repeated with a second suture, which is essential, and a third, rare but often useful with respect to the initial laxity, may be done (Fig. 17.4).

The PDS thread is cut under the skin. The anterolateral portal, which widens during the suture, it should be sutured both in depth and at the skin level.

The second technique involves the use of a #18 percutaneous spinal needle at the retinaculum level where the PDS is introduced for the suture. Through a small perimalleolar incision approximately 1 cm from the anterolateral portal, a pointed gripper is introduced (e.g., such as those used for shoulder surgery or one release

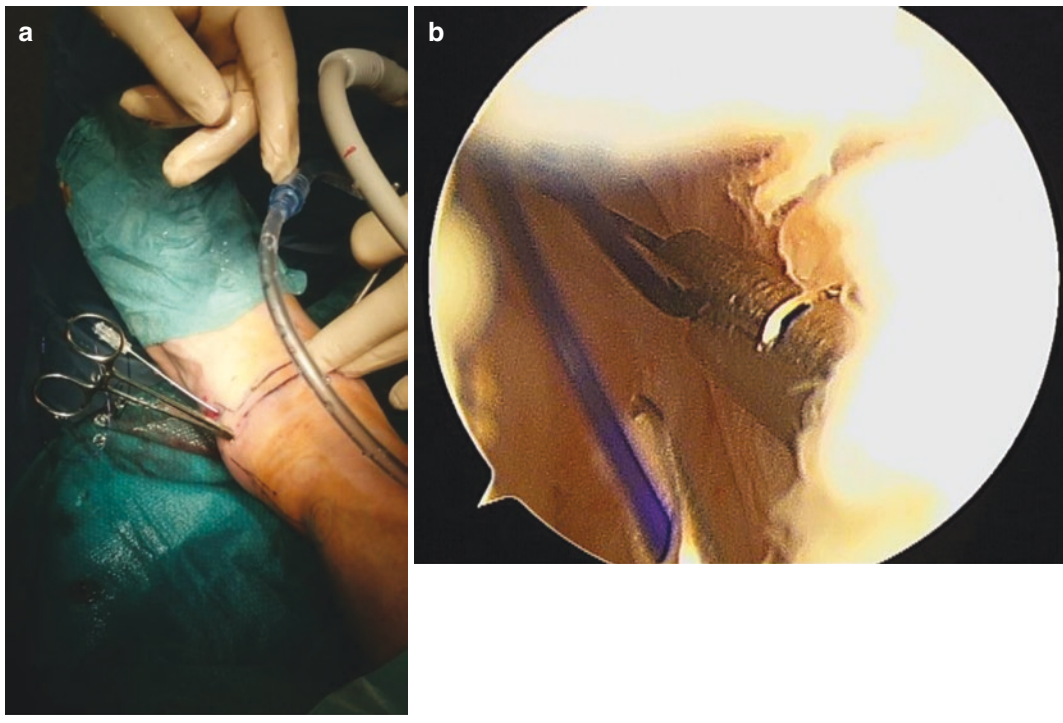

Fig. 17.2 (**a**, **b**) The perimalleolar percutaneous thread is retrieved with a subcutaneous Klemmer

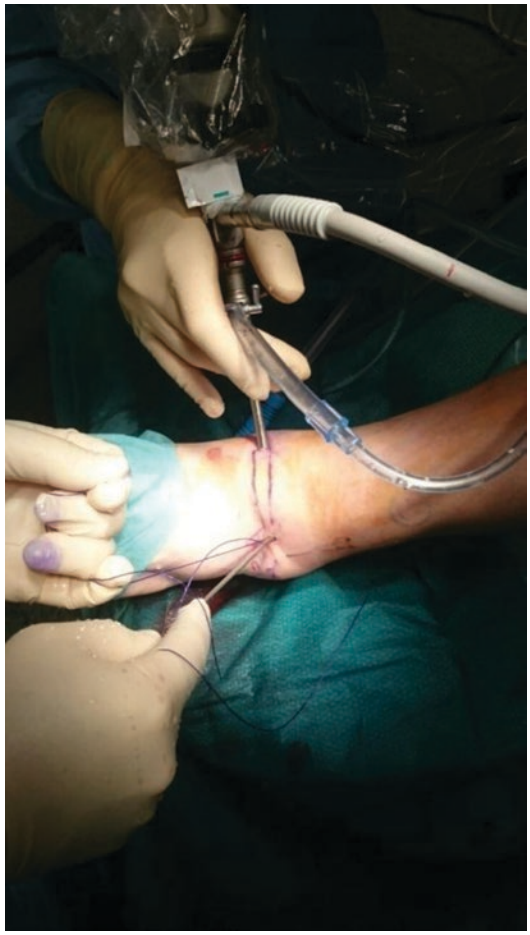

Fig. 17.3 Via a pusher, the knot that plics the capsule–ligament component (previously bloodstained) is executed with a motorized tool

shuttle that causes little damage to the articular capsule) to retrieve the thread that comes from the anterior area (Fig. 17.5). Once the two threads from the portal are recovered through a Klemmer with subcutaneous methodology, a knot is executed that is pushed and tightened via the arthroscopic portal.

During the postoperative phase, the ankle is wrapped with a bandage for a few days, which is then replaced by a bivalve brace for 3 weeks. This brace is needed to avoid foot inversion and eversion and to limit the flexion-extension. A partial load is allowed between the second and the third week.

The rehabilitation program then provides for the recovery of the full range of motion, of proprioception, and the muscle.

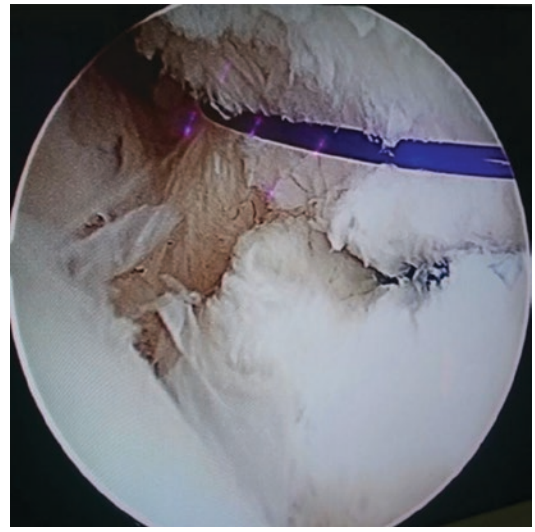

Fig. 17.4 Second plication

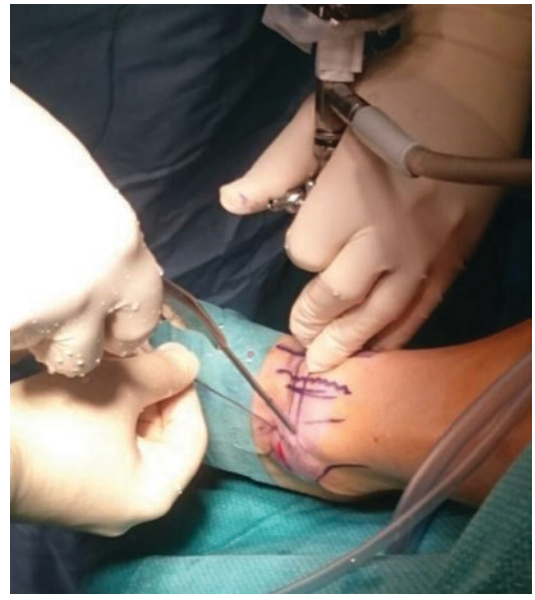

Fig. 17.5 A pointed gripper is introduced to retrieve the thread

17.3 Discussion

Of all acute lateral ligament injuries, 20% usually become chronic and require surgical treatment [7, 9].

A proper diagnosis is crucial to exclude associated injuries, such as cartilaginous or osteochondral lesions, fractures, or impingement.

Considering that patients are more likely to arrive at the clinic for associated injuries than for ankle instability, we believe that it is improper to treat only the osteochondral lesions without assess the capsule–ligament instability that may be the only and real cause of the ongoing symptoms.

The different proposed procedures for the treatment of such conditions can be divided into two main categories:

1. Direct repair of soft tissues.
2. Tendon transpositions, in which we can include the tenodesis.

The modified Broström technique constitutes the most widespread direct repair methodology and is characterized by the highest percentage of success in both the short and long term [26–28]. However, the previous literature also shows some weak results, particularly in patients characterized by congenital ligamentous laxity, inveterate ligamentous injuries, and previous intervention failures [27, 28].

Techniques with tendon transpositions allow a stable anatomic reconstruction, but the tendon removal requires an extensive exposure by increasing the risk of surgical wound complications, lesions of the nervous structures, development of fibrosis, and ankle stiffness [4, 29–32]. In addition, patients must undergo long rehabilitation programs that are not always properly observed and practiced.

Ankle stiffness occurs also in cases of tenodesis, which we believe to be a valid methodology only in severe instability and for workers forced to stand for long periods [33].

Despite the significant results of the direct repair technique, it is not advisable to perform it when the capsular tissue may be degenerated, as is frequently observed in cases of inveterate instability.

In the latter cases and in minor instabilities, we prefer to use the "thermal capsular shrinkage." This tool is used to obtain a synovectomy rather than a tissue retraction, often the main cause of the pain, associated with a reduction of capsular redundancy through lateral percutaneous plications under arthroscopic control.

The duration of the articular arthroscopic cleaning along with open surgery cause soft-tissue imbibition, making difficult the implementation of surgical procedures such as the Broström-Gould, because of an anatomy often not easily identifiable.

In 1996, radiofrequency (RF) was introduced in ankle arthroscopy, demonstrating a thermal shrinkage effect on the capsular ligamentous tissue that from being lax and redundant, through a thermal effect, it is able to shrink and scab, becoming a more consistent retracted fibrous tissue [34–37].

The use of RF for a capsular thermal shrinkage in symptomatic instability is a widespread practice that can obtain excellent results in selected cases [34, 35]. In fact, the RF removes most of the pain, but it does not guarantee stability. This lack has led us to add a technique that gives functional stability in ankles with chronic laxity that often have loose bodies or impingement.

We have identified the anterolateral portion of the ankle as a "safe zone," in the proximity of the peroneal malleolus, to avoid noble structures that can be damaged with the passage of the instruments and to not compress them with the plications [25].

Reduction of the capsular redundancy through these methods may be indicated in minor instability, in constitutional laxity after repeated distortion, or as a complementary element in those patients who should be treated for chondral pathologies [38, 39].

17.4 Conclusions

This technique appears to be useful for amateur athletes, for laborers with precarious balance (for example, bricklayers who require greater functional stability), for insufficient or nonfunctional cases of conservative treatments or in the presence of associated pathologies, to make the repairs more durable over time given that this does not happen with the simple shrinkage.

This is not a substitute technique for the traditional Broström-Gould, which remains the gold standard in severe instability, but it is an alternative that can be used in many pathologies that are

not disabling. In fact, the advantage of the plication technique is that it is all arthroscopically executable, with a more rapid rehabilitation and consequent functional recovery.

It is also useful when there are associated pathologies related to the trauma, but with symptoms that can appear late in relationship to alterations of the osteocartilage sector or synovial hypertrophy, rather than to ligamentous laxity.

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Radiofrequency Repair

18

Alberto Ventura

Arthroscopic procedure for the treatment of chronic lateral ankle instability has been proposed in order to minimize invasiveness, reduce operating time and allow a faster rehabilitation period, and an earlier return to work [1–6]. Arthroscopic thermal-assisted capsular shrinkage is an established option for the treatment of functional joint instability. Thermal energy, provided by radiofrequency and laser, produces an effective reduction in capsular volume and joint translation by shortening collagenous structures within the connective tissue [7].

The role of arthroscopy as a definitive therapeutic approach in the treatment of chronic ankle instability is recently progressing [1–6]. The Anterior Talo-Fibular Ligament (ATFL) is continuous with the joint capsule and therefore is readily accessible during ankle arthroscopy, and some surgeons adopted arthroscopic thermal-assisted capsular shrinkage for the treatment of functional instability. This condition, as opposed to mechanical instability, refers to a situation of subjective giving-way, persistent pain, and inability without anatomic ligamentous incompetence. In fact, in most studies, the procedure has been reserved to the cases when the ATFL was attenuated, not avulsed [1, 2]. Advantages of arthroscopic procedures compared to open techniques are the reduced invasiveness and surgical morbidity, less operating time, and quick return to work and leisure activities.

18.1 Surgical Technique

Arthroscopy is performed under local anesthesia with patient in supine position on the operating table and with slight elevation of the ipsilateral buttock. A tourniquet is positioned around the upper thigh. The skin is marked preoperatively to identify the course of the tibialis anterior tendon and the extensor digitorum longus tendon. The joint is filled with saline solution inserted through a spinal needle, to ease portals location. Standard anteromedial portal is established medial to the tibialis anterior tendon; anterolateral portal is placed lateral to the extensor digitorum longus tendon, with care taken to avoid the superficial branch of the peroneal nerve. No joint distraction is necessary.

Preliminary arthroscopic inspection is performed with a 4.0 mm 30° angle scope in order to confirm the diagnosis and to detect the presence of any concomitant synovitis, cartilage, or ligament injury. Attention is given to the assessment of the ATFL.

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Care is taken while performing synovectomy in the anterior chamber, because in this area, the neurovascular bundle lies immediately superficial to the capsule. The shaver resector is then addressed toward the ATFL lesion borders in order to obtain a debridement of the ligament remnants and the adjacent area.

The radiofrequency system is used to perform all thermal capsular shrinkage procedures.

The shrinkage procedure is performed with the ankle placed in an everted position starting from the ATFL remnants and progressing on the adjacent capsule. The probe is swept serially until all the area is treated basing on visual observation of the macroscopic tissue contraction. The thermal probe is then directed from the posterolateral to the anterolateral aspect toward the capsule with constant sweeping motion in order to reduce the time of exposure and the stress applied to the treated tissue. At the end of the procedure, joint flushing is performed and portals were closed and a sterile dressing is applied.

18.2 Rehabilitation Protocol

Immediately after the operation, an ankle brace is applied. For the first 3 weeks, patients are instructed to walk with non-weight-bearing with the use of two crutches. After the removal of the brace, patients are encouraged to the regaining of weight-bearing as tolerated and instructed to a physical rehabilitation protocol including proprioception and complete active and passive ankle range of motion exercises. Return to sport is permitted 12 weeks postoperatively.

Recently, several studies documented successful outcomes after arthroscopic thermal-assisted capsular shrinkage for the treatment of functional instability . Oloff et al. [6] first reported on the use of thermal stabilization procedure in the ankle joint on ten patients. At nine-month follow-up, they observed an improvement in the AOFAS score from 58.3 (sd: 8.96) preoperatively to 88.1 (sd: 11.09) postoperatively and a consistent reduction in ankle varus stress test and anterior drawer measurement. In the study by Khan and Fanton [4] on 23 patients who underwent shrinkage procedure, AOFAS score improved from 57.5 at baseline to 86.5 at follow-up. All patients stated they would undergo surgery again. Similarly, Maiotti et al. [5] looked at 22 patients 2.5–5 years out after thermal ankle shrinkage reporting 86% good to excellent results. Radiological anterior drawer sign reduced of 55% and talar tilt test of 80%.

Ventura et al. [8] adopted the shrinkage procedure for the treatment of functional ankle instability without ligamentous incompetence, and the procedure has been reserved to the cases when the ATFL was attenuated, not avulsed.

Synovitis is often characterized by swelling of the ankle joint, with or without associated pain and a feeling of constant or periodic discomfort. In the ankle affected by chronic instability, synovitis is a common finding as it represents the reaction to an insult to the lateral ligament complex.

Among lateral ankle ligaments, the ATFL is the only one, which is continuous with the joint capsule and therefore is easily accessible during arthroscopy. The resection of the borders of the avulsed ligament and the debridement of the adjacent area allow the stimulation of fibrous tissue and enhancement of the healing process of the ATFL complex. In this way, they act in the same fashion as in the debridement of cartilage lesions [9].

The shrinkage procedure provided by radiofrequency promotes structural change in the collagen triple helix and capsule healing through repopulation of the fibroblasts in the treated area [7]. In order to obtain denaturation of collagen with subsequent shrinkage of the fibers, optimal temperature range required should be 65–70 °C [10]. The shortening of collagenous fibers within the connective tissue leads to an effective reduction in capsular volume and allows to enhance joint stability [11, 12].

We prefer use brace immobilization in a neutral position with no weight-bearing for 3 weeks. Other studies reported absence of weight-bearing from 2 to 3 weeks [1, 3–5]. An exception is represented by the study by de Vries et al. [2], in which a compression bandage was applied only for three to five days and early weight-bearing was allowed 5–7 days postoperatively. Immobilization with

cast or brace and no weight-bearing are important in order to prevent lengthening of the treated tissue in the postoperative and allow the healing and reconstitution process [1, 11]. Patients should be instructed to avoid untimely return to sport activity that could affect the outcomes and lead to ankle instability.

We believe arthroscopic thermal-assisted capsular shrinkage itself to be insufficient for the treatment of chronic ankle instability with ATFL lesion.

Previous reports concerning arthroscopic treatment of ankle instability routinely made use of joint distraction [13]. In contrast, performing ankle arthroscopy without joint distraction allows the surgeon to fully dorsiflex the ankle joint by leaning against the patient's foot [14]. Dorsiflexion widens anterior working area and allows to address more easily anterolateral ankle problems and to remove loose bodies and osteophytes, which are usually located in the anterior compartment of the ankle joint [14]. Thus, in our experience, anterolateral ankle pathology can be managed without requiring joint distraction. Similarly, although the use of a 2.7 mm diameter arthroscope has been previously reported [13], we found that the ankle joint can be easily addressed using a regular diameter 4.0 mm scope.

Complication rate during ankle arthroscopy has been assessed in 9%, with neurologic injury being the most common occurrence [15]. In our case series, no major complications occurred in all patients considered. Complications, which persisted at follow-up, were damage to a superficial branch of the peroneal nerve in three subjects and deep peroneal injury in two cases. However, these complications did not affect significantly the overall functional outcome.

Injuries to the superficial peroneal nerve can be produced by recurrent ankle sprains. In order to discriminate between traumatic and iatrogenic neurologic lesions, an accurate neurovascular examination should be performed before surgery. Deep peroneal nerve injury can be related to capsular penetration following anterior orientation of a motorized shaver during synovectomy [16]. Alternatively, it can result from malpositioning of the trochar prior to joint penetration. To prevent the incidence of neurologic injuries, it is important to carefully consider neural structures close to the ankle joint capsule.

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Ligaments Anatomic Repair

19

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19.1 Introduction

There are many surgical techniques reported for the treatment of chronic lateral ankle instability (LAI). One of the procedures of the anatomical reconstruction, Broström procedure [1] with or without Gould, gives good clinical results in the long term and would be a golden standard for LAI. However, some patients who had attenuated lateral ligaments or failed previous reconstruction of lateral ankle ligaments had poor results by Broström procedure [2–4]. This population needs a anatomical reconstruction using by some graft. We show here an all-arthroscopic technique for anatomical reconstruction of the anterior talofibular ligament (ATFL) and the calcaneofibular ligament (CFL) by gracilis graft.

19.1.1 Indications

Surgery is indicated for patients with recurrent instability of the ankle secondary to injury to the lateral ankle ligaments after failure of conservative treatment. In addition, it is a good indication in cases of failed previous repair, where the patient's remaining tissue is insufficient or with generalized ligamentous laxity or excessive body mass index or high-demand heavy athlete.

19.2 Surgical Technique

19.2.1 Equipment

The technique is performed either with a standard 4.0 mm diameter arthroscope. Irrigation is provided by gravity pressure with a hanging saline bag or 50–60 mmHg pressure using an irrigation system. Arthroscopic dissection is performed using a 4.5 mm bone/soft tissue shaver blade.

19.2.1.1 Patient Positioning

The patient is placed in the lateral decubitus position with the pelvis slightly rotated 30° posterior. For position 1, the hip is externally rotated and the knee is flexed for harvesting the gracilis tendon for the autograft. Position 2 is used for anterior arthroscopy with the knee extended and the hip externally rotated. Position 3 is utilized for lateral endoscopy with neutral rotation of the hip and the knee extended (Fig. 19.1).

19.2.1.2 Portal Design

The patient is placed in position 2. Three portals can be created to perform the procedure. The anteromedial portal is the first portal (portal 1).

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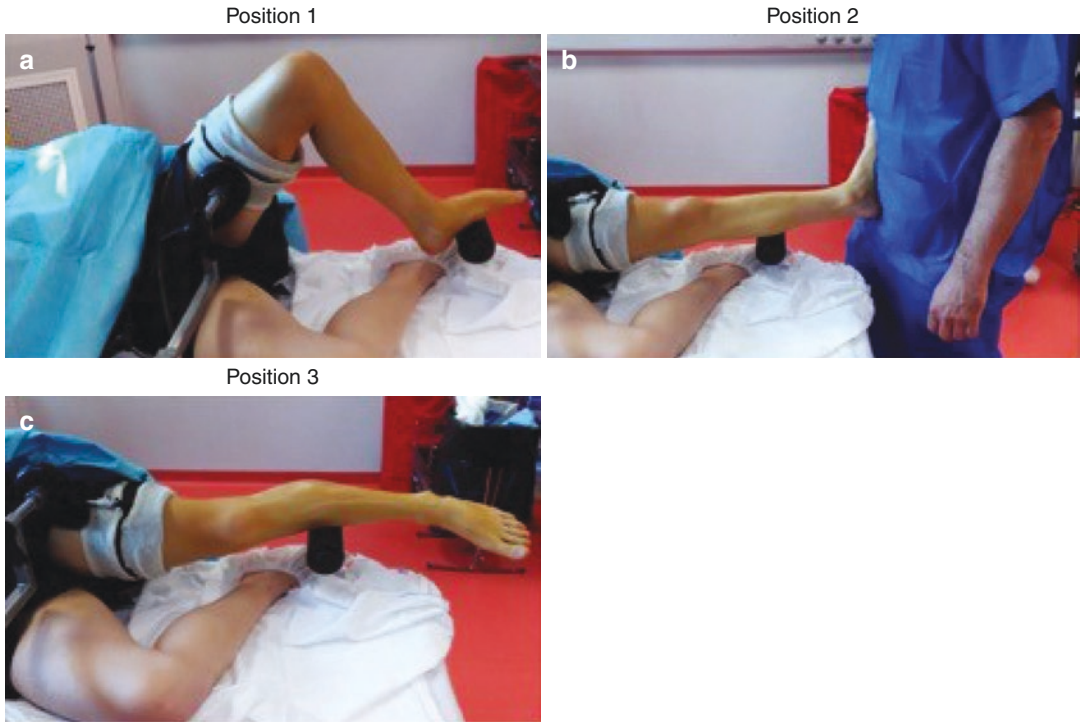

Fig. 19.1 Each position for operation. (**a**) Position 1 for harvesting the graft. (**b**) Position 2 for anterior arthroscopy. (**c**) Position 3 for lateral hindfoot endoscopy

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 while viewing the lateral gutter. The delay in developing an arthroscopic lateral ligament reconstruction was in part due to the difficulty in visualizing the CFL because of its extra-articular position. The CFL merges with the lateral talocalcaneal ligament and the peroneal tendon sheath as these attach to the calcaneus. Therefore, portal 3, which is localized to visualize CFL, is made at the cross section of two lines. The first line follows the superior border of the peroneus brevis tendon. The second line follows the direction of the lateral malleolar tunnel. This axis of the malleolar tunnel is 10°, rotating anteriorly to the axis of the fibula (Fig. 19.2).

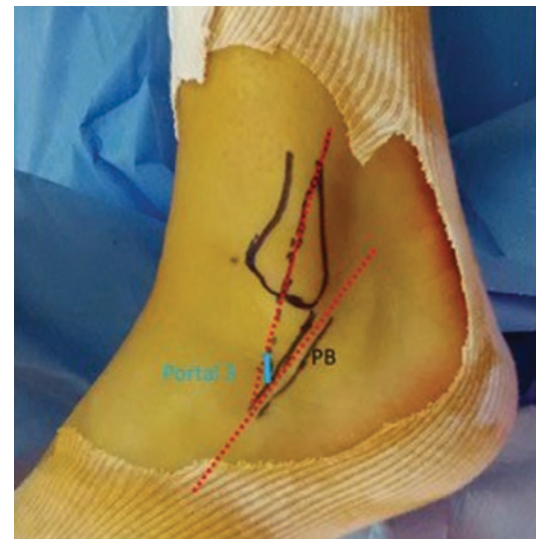

Fig. 19.2 Preparation of the graft when the talar side is fixed

19.3 Step-by-Step Description of the Technique(s)

19.3.1 Graft Harvesting

After harvesting, the graft is folded to create a 1-cm-long doubled segment with two single strand limbs, one for the ATFL reconstruction and one for CFL. A whip-stitch is placed on the 1-cm doubled end (which will be placed into the fibular tunnel) attached to a fibular internal fixation device. Whip-stitches are also placed on the CFL end (approximately 5.5 cm in length) and the ATFL end (approximately 4 cm in length). A similar preparation is performed if using an allograft gracilis tendon.

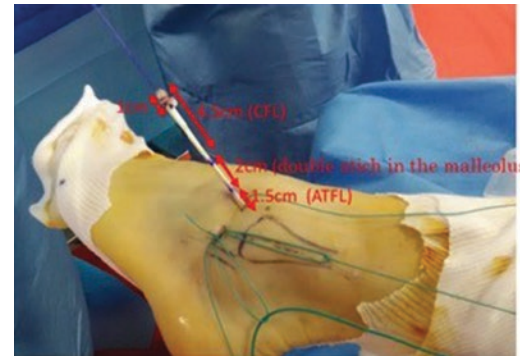

Fig. 19.3 Suture relay technique. After the graft is passing from portal 2 to portal 3 by passing suture loop. Head of red arrow: suture around the graft to control the graft length after introducing the graft to malleolus tunnel. Red arrow: suture loop of the endobutton. Blue arrow: the other suture of the endobutton to induce the graft into fibula tunnel

19.3.2 Step 1

The arthroscope is placed in the anteromedial portal (portal 1). It is important to make the portal with the ankle positioned in dorsiflexion and closely the tibialis anterior tendon in order to achieve a good view of the lateral joint space. Adequate position of this portal allows viewing the anterolateral gutter, including the fibular origin of the ATFL, the ATFL itself, and the insertion of the ATFL on the talus. Transillumination is used to create the accessory anterolateral portal (the position of the anterolateral portal is determined using the transillumination technique; portal 2) and a shaver is introduced through this portal to debride the lateral gutter exposing the fibular origin of the ATFL, the ATFL itself, and the insertion of the ATFL on the talus. Once the ATFL footprint has been debrided, the position for the fibular tunnel guide pin insertion between the CFL and the ATFL is identified.

19.3.3 Step 2

The arthroscope is now placed in portal 2. Portal 3 is created at the sinus tarsi in the marked position as described previously (Fig. 19.3). A shaver is then introduced through portal 3 to complete the preparation at the malleolar ligament insertion. A Beath pin is then inserted and drilled across the fibula, exiting the fibula posteriorly and proximally, with care taken to protect the peroneal tendons. The pin is overdrilled using a 4.5-mm cannulated drill, and then a 6-mm cannulated drill is used to make the tunnel to a depth of 1 cm. With the arthroscope in portal 3, the dissection is continued along the lateral articular surface of the talus and then the anterior side of the posterior subtalar joint. This dissection is guided by the remaining CFL until the insertion on the calcaneus, between the lateral cortex of the calcaneus and the peroneal tendons. This step must be performed with great care in order to properly identify the fibers of the CFL insertion. A Beath pin is then placed in the footprint and drilled through the calcaneus. The direction is inferior, posterior, and medial. A cannulated 6-mm drill is then introduced through the two cortexes.

19.3.4 Step 3

The arthroscope is now inserted into portal 3. Using a shaver placed in portal 2, the talar

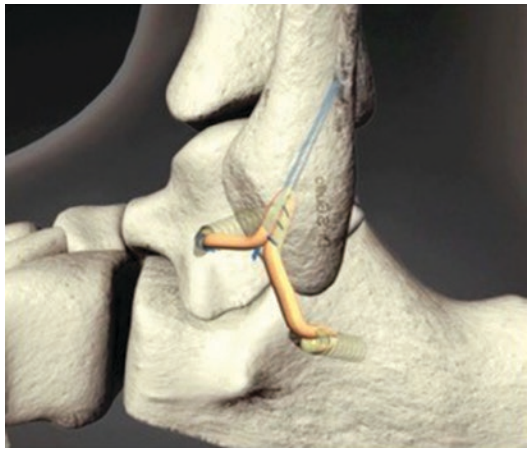

Fig. 19.4 Final aspect after tensioning of the graft in neutral position

insertion of the ATFL is completely exposed. A pin is then placed under direct visualization into the center of the talar insertion of the ATFL, and then the pin is overdrilled to a depth of 20 mm using a 6-mm cannulated drill. The direction of the tunnel is slightly upward, centered toward the middle of the talus.

19.3.5 Step 4

Step 4 corresponds to the placement of the ATFL transplant. Arthroscope is placed in portal 3. Graft is induced into talar tunnel by interference screw from portal 2 under perfect vision by arthroscopy. Graft is measured (Fig. 19.4) and then endobutton is induced through portal 3 to fibula tunnel and then hooked on the posterior and lateral cortex of the malleolus. By pulling the other sutures of endobutton, graft is induced fibula tunnel. It is important to check the marking of graft length by arthroscopy.

19.3.6 Step 5

The arthroscope is now placed in portal 2. By pulling the traction suture on the opposite side, the CFL limb of the graft will be introduced into the calcaneal tunnel. The final fixation is achieved with interference screw introduced into the tunnel. Because of the poor quality of the calcaneal bone, the screw is oversized and a long screw is used. For a tunnel of 6 mm, it is recommended to use a 7 × 25-mm screw. It is important to use the arthroscope to control the complete introduction of the screw head to avoid impingement with the peroneal tendons. Finally, graft tension is decided in neutral position of ankle by controlling the traction suture from endobutton.

19.4 Post-operative Care

The patients are immobilized with a soft ankle orthosis for 15 days. Active and passive dorsi- and plantar flexion range of motion and weight bearing are started at 15 days. Inversion and eversion exercises are started at 6 weeks after surgery and full athletic activity at 12 weeks after surgery.

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Syndesmotic Joint Instability Arthroscopic Repair

20

Francesco Allegra and Francesco Lijoi

Ten percent of all ankle fractures sustain an associated syndesmotic injury. Syndesmosis injuries have been reported in conjunction with ankle sprains to ankle fractures, with an incidence between 1% and 18%. Isolated ligamentous syndesmosis sprains are rare and consequently are often undiagnosed, progressing to chronic ankle symptoms such as pain and disability, until arthritic joint changes indicate the original pathology. Syndesmotic injuries, even isolated, are not always easy to diagnose because they range from a simple sprain to frank diastasis. Among the most important predictors of functional results, on treatment of syndesmotic injuries, is the accurate restoration of the syndesmotic space. The suspension technique can achieve flexible fixation of the syndesmosis and permit full range of motion of the tibiofibular joint, thanks to starting rehabilitation exercise at an early stage after surgery.

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20.1 Anatomy and Biomechanical Background

The distal tibiofibular syndesmosis is a joint with a low degree of mobility between tibia and fibula. The fibula lies on the "incisura fibularis" of the tibia. In 75% of the cases a true articulation is present, with articular cartilage on the two contact facets, forming a synovial joint with a fat-containing synovial fold interposed between the synovial lining and the fibula: this synovial fold contains loose connective tissue with an abundance of blood vessels and occasionally some small nerves [1]. The syndesmotic ligament complex consists of the following. (a) The anterior inferior tibiofibular (AITF) ligament has a triangular aspect with multiple tight fibers interspersed with some fat. The fibers start at the broad-based anterior tibial tubercle (Chaput) and converge toward the fibular tubercle (Wagstaffe–Le Fort) [2]. (b) The posterior inferior tibiofibular (PITF) ligament is a strong ligament that extends from the posterior tibial malleolus to the posterior tubercle of the fibula and runs from proximal-medial to distal-lateral, forming a 20–40° angle with the horizontal plane [2]. It is a thick and strong ligament: very high rotational stress force more often results in a fracture of the posterior tibial malleolus than in a rupture of the ligament. (c) The interosseous ligament (IL) is a thick and round ligament that runs horizontally between the proximal margin of the fibular malleolar fossa and the dorsodistal rim of the tibia. It can be

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considered a distal continuation of the interosseous membrane at the level of the tibiofibular syndesmosis [3]. The interosseous membrane is not a true ligament, but it is essential in maintaining connection between tibia and fibula and acts as a reinforcement structure.

Another indirect stabilizer is the deltoid ligament, which acts on the medial side to hold the syndesmosis stabilizing the talus to the tibial malleolus. The AITFL limits the fibular external rotation, the PITFL limits the posterior translation, whereas the IOL prevents the lateral translation of the fibula [4, 5]. On the other side of the joint, the deltoid has a critical function in limiting talar abduction, pronation, and external rotation [5]. The resistance to diastasis comes from the PITFL and IT for 40–45%, the AITFL for 35%, and the interosseous membrane for 20–25% of cases [6].

The most widely accepted mechanism resulting in injury of the syndesmotic ligaments is external rotation and hyperdorsiflexion. Distal tibiofibular syndesmosis is essential for the stability of the ankle mortise. In normal situations, the ankle mortise widens only 1 mm during gait [7]. During the stance phase of the gait cycle, weight-bearing determines a lateral thrust of the talus against the fibular malleolus. When syndesmotic and deltoid ligaments are disrupted, the talus is functionally disconnected from the leg, and the patient complains of instability during weight-bearing. The incidence of ankle syndesmosis injuries is higher in athletes [8, 9] because of their movements: planting the foot and then performing cutting motions, as well as the possibility of direct associated blows to the lateral ankle [10], can progress to a sprain. The reported percentage of syndesmotic injuries is variable because of difficulties of a correct diagnosis, but it varies from 1% to 18% of all ankle sprains [7, 11], excluding the cases of ankle fractures.

20.2 Classification

A classification of syndesmosis injuries has been proposed by Porter [10] as follows:

- *Grade 1*. Injury to the anterior deltoid ligament, to the AITF ligament, and sometimes to the interosseous ligament, but without tearing of the interosseous membrane or the deep deltoid ligament. This kind of lesion is by definition stable because there is no widening of the syndesmosis.
- *Grade 2*. The anterior and deep deltoid ligament is involved so far as the syndesmosis in the AITF and interosseous ligament. Distal tibiofibular syndesmosis is unstable but the ankle is normally aligned on nonstress radiographs. This kind of occult instability is often difficult to recognize.
- *Grade 3*. The injury involves the entire deltoid ligament and syndesmosis is widely disrupted: the fibula is often fractured above the syndesmosis, sometimes at its proximal part (Maisonneuve lesion). A more recent classification has been proposed distinguishing acute isolated syndesmotic injury as stable or unstable [12].

20.3 Diagnosis

Diagnosis is achieved at the clinical examination for patients sustaining an instable syndesmotic injury who complain of persistent ankle pain, sensations of giving way, proximal palpation pain superiorly to the mortise, and difficulty when walking on uneven ground [13, 14]. As concerns the diagnosis of syndesmotic lesions, many clinical tests have being described: the Cotton test and the fibula translation test are the most reliable for diagnosis [12, 15], by comparing to the opposite side. Many imaging signs are described in the literature for the diagnosis of ankle syndesmosis disruption: the tibiofibular clear space and the tibiofibular overlap sign are the most often described, paying attention to false images caused by the joint rotation [16]. The medial clear space, between the lateral border of the medial malleolus and the medial border of the talus, is a valid radiographic sign. In the mortise view this measure should be equal to or less than the superior clear space between the talar dome and the tibial plafond [17].

Weight-bearing radiographs are very useful in disclosing occult lesions. Magnetic resonance imaging (MRI) can be very useful in visualizing disruption of syndesmotic ligaments [18], and

computed tomography (CT) can identify displacement of the distal tibiofibular syndesmosis using the tibiofibular line [19].

20.4 Treatment

Treatment of syndesmotic lesions depends on the degree of the lesion [20]. Stable lesions (grade 1) must not be treated surgically [10, 12]. The RICE protocol is recommended in the first 7–10 days, with complete weight-bearing with crutches in the first 1–2 weeks. The rehabilitation program permits an average time to return to the sport in 4–8 weeks. Unstable lesions (grade 2–3) are treated surgically, but the choice of the type of surgery depends on the type of instability, whether acute, subacute, or chronic. In acute cases, the treatment is preferably performed with trans-syndesmotic screw fixation. In the subacute setting (6 weeks to 6 months), the focus is to restore the normal anatomy by repairing the elongated ligaments and by placing a positioning screw [14]. If the remnants of the AITF are inadequate, a free tendon graft (plantaris tendon, peroneus longus tendon, or fascia lata) can be used to replace it. For chronic lesions [15, 19], those more than 6 months old, the treatment should be fusion of the distal tibiofibular syndesmosis. In subacute lesions, this type of reconstruction must be associated with an arthroscopic debridement of the syndesmosis and always with a syndesmotic screw fixation. In the past decade, suture-button devices have been used instead of screws, alone in acute cases or associated with other techniques (graft, anatomic reconstruction of AITF ligament) in subacute or chronic settings. In a recent systematic review [21] of suture-button versus syndesmotic screw techniques, the suture-button fixation group showed similar American Orthopaedic Foot & Ankle Society (AOFAS) outcome scores (91.06 points) compared to the conventional screw fixation (87.78 points) group, and the rate of implant removal and poor reduction was lower in the suture-button fixation group.

Arthroscopic debridement of syndesmosis and arthroscopically assisted insertion of the suture-button device is today's technique. The patient is assessed in a supine position, with the leg in a noninvasive traction device. In addition to the two usual portals, an ancillary anterosuperior portal is used for a more accurate removal of the scar tissue inside the syndesmotic space (Fig. 20.1). After the complete cleanup of the scar tissue and an evaluation of the lesion including the medial compartment (Fig. 20.2), the pathological mobility of the joint is assessed (see Videos 20.1 and 20.2). The K-wire of the suture-button device is then passed along both

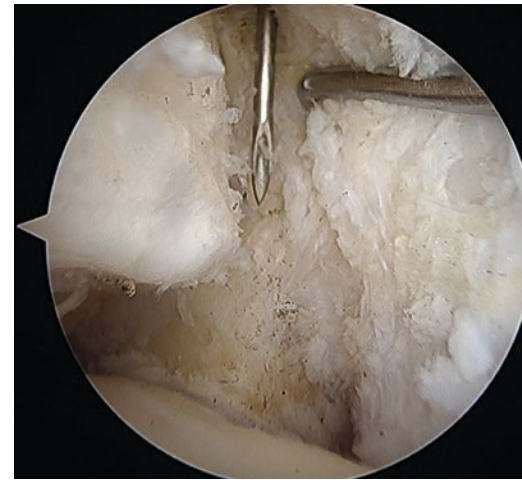

Fig. 20.1 The anterosuperior ancillary portal is opened along a needle placed on the skin, marked before surgery, to address surgery by shaver inside the syndesmotic space

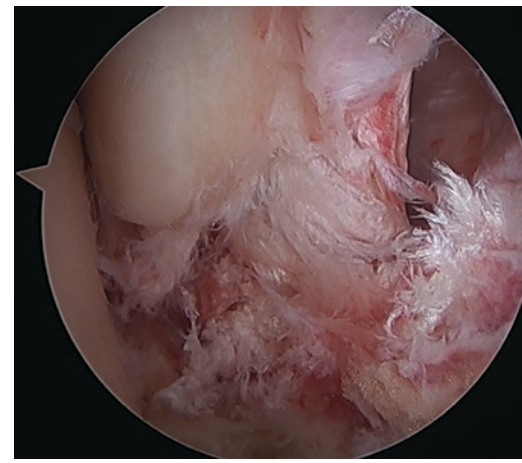

Fig. 20.2 Exploring the ankle joint compartments: a lesion on the tibial side indicates total disruption of the deltoid ligament

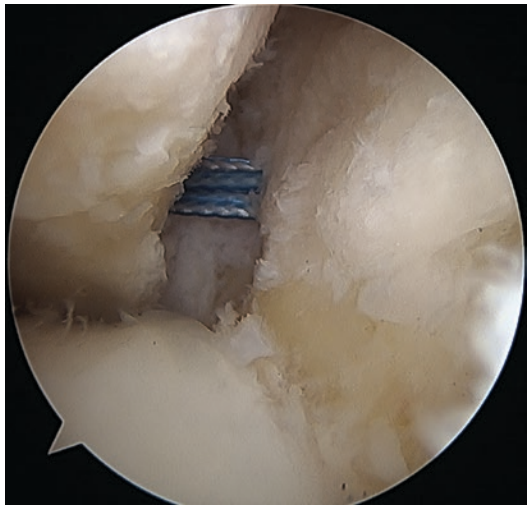

Fig. 20.3 The suture button lies in place, ready to be tightened

the fibular and tibial shaft, choosing the exact level at which to insert by fluoroscopy. The wire is pulled ahead until its tip emerges from the cortical bone inside the syndesmosis, under direct scope visualization. The wire is then completely drilled out the contralateral side of the leg, until the button engages the cortical bone. When both buttons are regularly placed on the two sides of the syndesmosis, on the lateral cortical side of the fibula and on the medial side of the tibia, it is possible to reduce the syndesmotic lesion under direct scope visualization (Fig. 20.3) by simply tightening the sutures.

Traps	Tricks
Uncertain decision about treatment	Make an accurate classification of the lesion
Low visualization with scope	Clean up the scar tissue in the syndesmosis with shaver and radiofrequency wand
Difficult to move instruments and visualize syndesmosis together	Provide opening an anterosuperior accessory portal for shaver using anterior for scope
Doubts about the placement of the K-wire guide for suture button	Use both scope and fluoroscopy at the same time

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Part VI

Upper and Lower Ankle Arthritis

Ankle Arthritis: Etiology and Classifications

21

Andrea Pantalone, Matteo Guelfi, Vincenzo Salini, and Marco G. B. Guelfi

21.1 Introduction

Ankle arthritis is less common than osteoarthritis (OA) of the other major joints of the inferior limb; the patients with this pathology have a lower quality of life and functional limitations [1, 2].

OA of the tibiotalar joint is mostly post-traumatic: this explains why ankle OA affected a younger and higher demanding population if compared with hip and knee.

The diagnosis starts with clinical assessment (including alignment and stability) and weight-bearing radiographs of the foot and ankle; in fact, several ankle OA classification systems are based on radiographic evaluation.

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21.2 Etiology

Osteoarthritis (OA) of the tibiotalar joint is present in 4.4% of the patients affected by OA of the lower limbs; primitive ankle OA hits only 9% of the cases.

Fifty-percent of elderly patients has some form of arthritis involving the foot or the ankle [3, 4], because the number of such cases has risen in step with the aging of the population.

Ankle arthritis is peculiar, although less frequent than the other forms affecting other joints [5], due to a fundamental fact: the ankle bears a weight significantly higher than the knee or the hip, for instance [6]. The anatomic ground for the high load tolerated by the ankle lies in two basic aspects:

(a) The ankle surface area exposed to load is approximately one-third than the corresponding areas in knee or hip [7].
(b) The thin (1–2 mm) cartilage of the ankle is far more resilient [8] than those of the other joints; such a feature explains the lower prevalence of OA in ankle. Therefore, the cartilage layer is exposed to shear rupture in case of traumatic impact. Besides, in the superficial layer of the cartilage, the chondrocytes are presented in groups [9] and are metabolically more active than those of the knee and present greater aggrecan turnover and greater sensitivity to anabolic stimuli [10].

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The secondary causes, due to a variety of underlying diseases or disorders (rheumatoid arthritis, aseptic necrosis, hemophilia, hemochromatosis, acromegaly, Paget disease, Ehlers-Danlos syndrome, Gaucher disease, Stickler syndrome, ochronosis, or neuropathic arthropathy) [11], are present in 13% of the patients.

Specifically, there is a high prevalence of foot and ankle involvement in rheumatoid arthritis (RA) with over 90% of patients reporting pain during the course of the disease [12] (Fig. 21.1). Over 60% of patients report walking disability and foot involvement impacts negatively on health-related quality of life. Atraumatic causes include also infection and crystalline arthropathies.

21.2.1 Posttraumatic Changes

Posttraumatic changes are the main reasons of ankle osteoarthritis development (about 65–80%), and this may explain the different reported epidemiology with a younger population affected [13–15]. The ankle is the most commonly injured joint in sport activities, with more than 300,000 injuries per year reported in the USA, and an estimated 52.3 ankle injuries per 1000 athletic exposures in high school-aged athletes [16].

The causes of posttraumatic ankle arthritis are the following:

- Ankle fracture
- Pilon fracture
- Tibia or fibula fracture
- Talus fracture
- Ankle dislocations
- Recurrent sprains
- Persistent ankle instability
- Osteochondritis dissecans of the talus or distal tibia.

In the athletes, the cause is repetitive minor trauma due to sport activity or due to ligamentous instability. A high percentage of residual complaints are reported after an acute lateral ankle ligament rupture. At long-term follow-up, 30–40% of patients report restrictions. Development of residual complaints after supination trauma is related to the initial pathology. In particular, recurrent ankle sprains in sports (e.g., soccer) are the main causes of ligamentous posttraumatic ankle OA with concomitant varus hindfoot deformity [17].

In adult active population, ankle fractures surgically treated are the most frequent cause. Several factors have been implicated in the development of OA, including fracture severity, osteochondral impact at the injury instant,

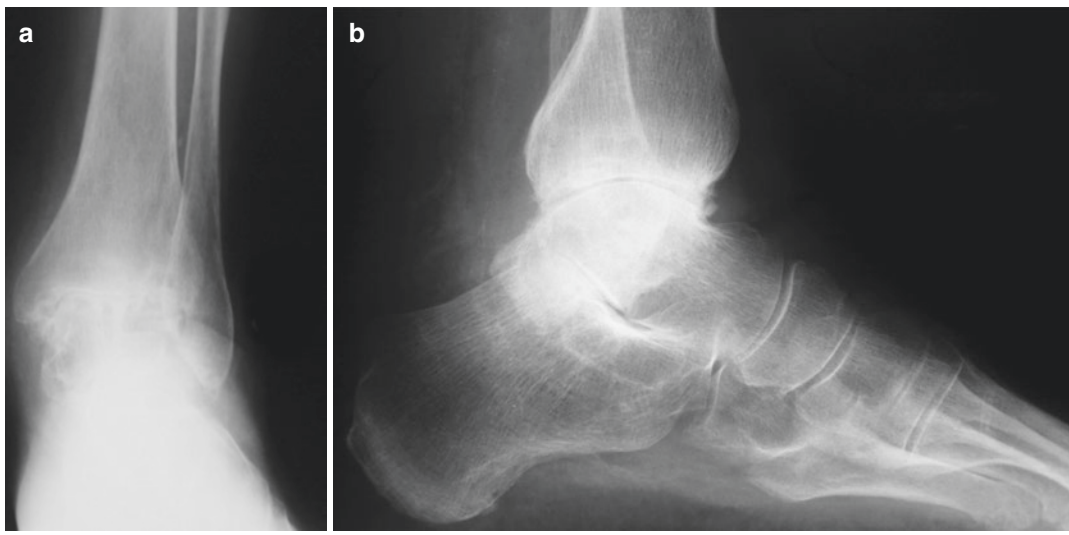

Fig. 21.1 Ankle arthritis in a 57-year-old RA patients: (**a**) anteroposterior and (**b**) lateral views

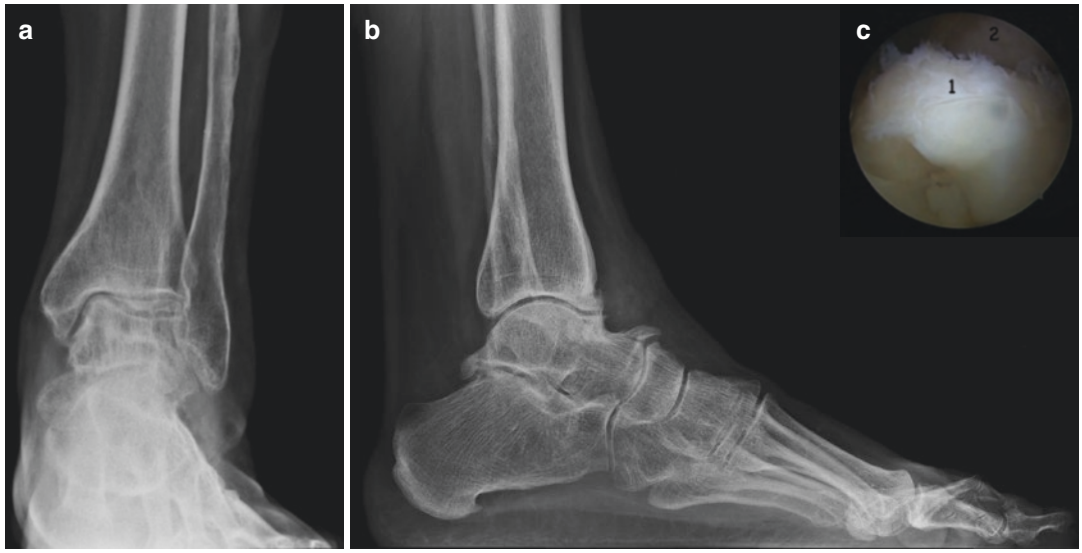

Fig. 21.2 Weight-bearing (**a**) anteroposterior and (**b**) lateral views of the ankle of a 60-year-old female showing a posttraumatic ankle OA. Lateral view also shows subtalar and talonavicular arthritis. (**c**) Arthroscopic view of medial side from anterolateral portal: a big talar osteophyte (1) hits tibia anterior edge (2) limiting dorsiflexion

and a nonanatomic reduction [18]; some studies regarding ankle fractures have shown poor clinical results including chronic pain, arthrofibrosis, recurrent swelling, and perceived instability despite anatomical restoration of the ankle joint and mortise following fractures [19, 20].

Joint trauma is the main etiological factor, especially malleolar fractures and distal fractures of the tibia, but also chronic malalignment, chondral joint damage, chronic instability [21], and microinstability [22]. In particular, Functional Ankle Instability (FAI), introduced by Freeman in 1965 and defined as the subjective feeling of giving way, which may occur despite an absence of deviation beyond the normal physiological range of movement of the talus [23–25], is a risk factor for osteoarthritis. In fact, Golditz et al. [26], using T2-mapping, described early cartilage damage in young athletes with FAI and demonstrated that Function Instability causes unbalanced loading in the ankle joint, resulting in early cartilage alterations.

21.3 Classifications

The specific system for staging OA is controversial. The current ankle OA classification systems are based on radiographic evaluation of degenerative changes of the joint. Most of this classification systems divided ankle OA into 4 stages. These generally describe stage 1 as early, stages 2 and 3 as intermediate, and stage 4 as late.

In 1995, Takakura [27] used weight-bearing radiographs to classify the arthritis as follows (Fig. 21.2): Stage I: No narrowing of the joint space, but early sclerosis and formation of osteophytes; Stage II: Narrowing of the medial joint space; Stage III: Obliteration of this space with subchondral bone contact; Stage IV: Obliteration of the whole joint space with complete bone contact.

Tanaka and colleagues [28] updated the Takakura's classification as reported in Table 21.1.

In 1997, however, Van Dijk and colleagues [29–30] proposed their classification, similar to Takakura's one. They showed that the osteoarthritic classification proved to be more discriminative than the existing impingement classification as a predicting value for the outcome of arthroscopic surgery for anterior ankle impingement: patients with grade II–III OA were considered to be unsuitable for arthroscopic debridement. This classification is reported in Table 21.2.

Based upon a historical consensus that the diagnosis of OA can be determined by radio-

Table 21.1 Ankle arthritis classification system according to Tanaka and colleagues [28]

Stage	Ankle degenerative changes on radiographic evaluation
Stage 1	Early sclerosis and formation of osteophytes without narrowing of the joint space
Stage 2	Narrowing of the medial joint space
Stage 3A	Obliteration of the medial joint space with subchondral bone contact limited to the medial malleolus
Stage 3B	Subchondral bone contact extending to the roof of the dome of the talus
Stage 4	Obliteration of the entire joint space, resulting in bone contact throughout the ankle

Table 21.2 Ankle arthritis classification system according to Van Dijk and colleagues [29, 30]

Stage	Ankle degenerative changes on radiographic evaluation
Grade 0	Normal joint or subchondral sclerosis
Grade I	Osteophytes without joint space narrowing
Grade II	Joint space narrowing with or without osteophytes
Grade III	(Sub)total disappearance or deformation of the joint space

graphic features, the Kellgren and Lawrence (K&L) scale was chosen by the World Health Organization as the accepted reference standard [31], but some authors stated that there are no correlations between clinical outcome and radiological OA signs [32]. Recently, Holzer et al. [33] demonstrated a relationship between clinical outcomes and severity of posttraumatic ankle OA as assessed with the K&L scale: with increasing K&L grades, all clinical scores decreased, indicating more pain and greater functional disability. The authors formulated a modified [33, 34] K&L scale as follows:

Grade 1: Osteophytes of doubtful meaning on the medial or lateral malleolus, rare tibial sclerosis, joint space width unimpaired; Grade 2: Definite osteophytes on the medial malleolus, joint space width unimpaired; Grade 3: Definite osteophytes on the medial and/or lateral malleolus, moderate joint space width narrowing (<50%) with talar tilt <2° (Subgrade 3A) or talar tilt >2° (Subgrade 3B); Grade 4: Definite osteophytes on medial and lateral malleoli as well as tibiotalar joint margins, severe (>50%) to complete joint space narrowing, constant tibio-talar sclerosis.

Recently, Claessen and Ankle Platform Study Collaborative: Science of Variation Group documented fair interobserver agreement for the van Dijk osteoarthritis scale, and poor interobserver agreement for the Takakura and the Kellgren osteoarthritis classification systems: this study revealed that those systems cannot be used for clinical decision-making [32]. The authors stated that classifications composed of a fewer number of categories might result in higher reliability and are therefore more valuable for clinical practice [35].

21.4 Conclusion

The knowledge of the underlying etiology is fundamental for choosing the best treatment. Nowadays, there is no definitive consensus about the most valid and reliable scale for assessment of ankle OA.

Arthroscopic approach, if possible, is a viable tool for treatment of all stages of ankle arthritis: from arthroscopic debridement to ankle fusion. These procedures appear to be safe and effective and allow for management of intra-articular pathology.

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Ankle Joint Debridement

22

Paolo Ceccarini, Andrea Pantalone, Pierluigi Antinolfi, Giuseppe Rinonapoli, Auro Caraffa, and Marco Guelfi

22.1 Introduction

Ankle degenerative pathology is the main cause of a rigid ankle, characterized by pain and loose of range of motion (ROM). As discussed in previous chapter, ankle ostheoarthritis (OA) is post-traumatic in 70–80% of instances and has progressive features such as joint space narrowing, osteophytes, cartilage lesions, and loose bodies [1, 2]. Also anterior ankle bony impingement is an initial pattern of ankle arthritis.

After a post-traumatic ankle pathology, for initial-mild stage of osteoarthritis, can be performed innumerable surgical options. These include arthroscopic debridement, resurfacing, and osteotomies. The rationale for ankle arthroscopy is to reduce surgical insult to the soft tissue envelope with the aim of decreasing complication's rate, hospital length of stay, and a faster recovery time, especially in young adult population.

In the athletes, the cause of the ankle pathology is repetitive minor trauma due to sport activity as in footballer ankle or due to a ligamentous instability. A high percentage of residual complaints is reported after an acute lateral ankle ligament rupture. At long-term follow-up, 30–40% of patients report restrictions. Development of residual complaints after supination trauma is related to the initial pathology.

In adult active population, ankle fractures surgically treated are the most frequent cause and can evolve in a progressive ankle posttraumatic arthritis even if the surgery is well performed [3, 4]. Some studies regarding ankle fractures have shown poor clinical results, including chronic pain, arthrofibrosis, recurrent swelling, and perceived instability despite anatomical restoration of the ankle joint and mortise following fractures [5].

The incidence of intra-articular injuries following ankle fractures and their optimal treatment remain unclear despite multiple clinical investigations [6].

Complaints may be caused by bony spurs, irritation from internal fixation hardware, and soft tissue impingement [7, 8]. Complaints may also be generalized and caused by synovitis or post-traumatic arthritis. However, the etiology of residual pain after ankle fractures and the optimal treatment remain unresolved [5].

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The degenerative pathology is the main cause in the origin of the ankle joint stiffness. The anterior impingement could be associated with minor or major traumatic events.

The arthrofibrosis is the most dangerous complication. The edema contributes to aggravate the symptoms and stiffness. Usually, the pain is widespread.

Arthroscopy has been taking place in recent years, especially in young patients after posttraumatic disorders. The goal of arthroscopic debridement is the removal of the fibrous adhesions, synovectomy, removal of osteophytes and loose bodies if present, assessment and treatment of osteochondral lesion, and eventually capsulectomy.

Contraindications are bony or fibrous ankyloses, bone deformities, major axial deviations, neurovascular deficit, and active infections.

22.2 Clinical Examination

Patients who report pain at maximum degrees of motion or certain activities (i.e., stair climbing, patients with anterior impingement, etc.) are ideal candidates for arthroscopic osteophyte resection and debridement [9]. Arthroscopic debridement is selected for patients with large osteophytes, which limited motion and with residual symptoms.

On physical examination, we can observe limited dorsiflexion and particularly some pain of the most anterior aspect of the ankle joint. The ankle will not necessarily appear unstable, although the presence of diffuse pain in an active individual should always make the clinician consider an ankle instability in differential diagnosis.

Overall, the investigation may be not impressive except for the presence of pain with palpation of the anterior osteophytes. Surgical intervention is recommended after failure of conservative treatment for six months, treatment options will include the use of nonsteroidal antiinflammatory drugs, activity modifications, physiotherapy and the use of protective devices, and even the possibility of intra-articular viscosupplementation.

Anterior ankle impingement may be due to soft tissue or osseous. This is frequent after trauma to the ankle joint with the development of an increased and unusual inflammatory response. The synovial tissue will have an excessive response to an intra-articular hematoma [10–12].

In patients with bony impingement, the location of tibial spurs is reported to be at the joint level and always inside the joint capsule [13, 14].

Several studies suggested that treatment and prognosis are dependent on the stage of ankle osteoarthritis. Posttraumatic ankle arthritis can be a very disabling condition, and therefore, adequate treatment is helpful [10].

Van Dijk et al. reported good or excellent results for arthroscopic treatment in 76% of patients if complaints could be attributed clinically to anterior bony or soft tissue impingement [15, 16].

Thomas et al. retrospectively reviewed 50 patients who had ankle arthroscopy to evaluate residual pain after an ankle fracture. A synovitis was found in 46 ankles, and arthrofibrosis was observed in 20 cases [7]. Chondral lesions of the talus or tibia were present in 90% of patients. Utsugi et al. performed arthroscopy at the time of hardware removal in 33 consecutive patients who had undergone open reduction and internal fixation for ankle fractures. Articular cartilage damage was noted in 33% and arthrofibrosis in 73% of patients. Arthroscopic debridement of fibrous tissue led to improved joint function in 89% of patients with functional deterioration after an ankle fracture [8]. Arthroscopic debridement of fibrous tissue in these patients was associated with a better clinical outcome, especially if related to the low degree of arthritic degeneration (grade 1–2). These results suggest that ankle arthroscopy may be of value in identifying and managing chronic pain caused by various intra-articular lesions after ankle fracture [6].

Arthroscopic debridement is also associated with good results especially in cases of “frozen ankle” in which an adhesive capsulitis involves the entire joint capsule. In these patients, a posterior endoscopic approach combined with an anterior one, made it possible to obtain encouraging results [13, 15].

Furthermore, ankle stiffness can be caused by extra-articular pathology. Tendon adhesions or muscle contracture around the ankle are the most common extra-articular cause of posttraumatic stiff ankle [13].

We recommend to perform standard X-ray, weight-bearing radiographs of the ankle in antero-posterior and lateral, anterior medial impingement (AMI view), and a hind-foot alignment view (Saltzmann view) [17, 18]. In case of clinical doubt of an osteochondral lesion and negative X-rays for additional diagnostic assessment, a CT scan and MRI have demonstrated to have a comparable accuracy [12] (Fig. 22.1).

As Van Dijk reported, we believe that MRI allows visualization of the overlying abnormalities in the cartilage, but it is not as useful in showing the exact cortical outlines. The true size of the lesion can be obscured by concomitant bone marrow edema. For a more exact preoperative planning, we advise to perform a CT scan [12] (Fig. 22.2).

22.3 Surgical Treatment: Ankle Debridement

A debridement for ankle arthritis is useful in the earlier stages of the arthritic process. It is normally performed as a day-case procedure and generally patients can bear weight straight away on the operated ankle. The success rate for the procedure is about 75–80% in the early osteoarthritic stages [6, 12]. This corresponds to the percentage chance of improvement but not always with complete symptom resolution. Sometimes it can take a number of months before this benefit is achieved. It needs to be borne in mind that there is approximately a 5% chance of significant symptomatic worsening following debriding an arthritic ankle [19, 20].

Before proceeding with ankle arthroscopy for arthritis, it is important to realistically way up the pros and cons with your patients. There is a chance that they would need more extensive surgery shortly after this initial procedure if they fall into the 5% bracket of symptoms getting significantly worse. Debridement is a procedure to remove loose bodies, inflamed synovial tissue, and bone osteophytes from the joint. There is an indication for removal of osteophytes in case the patient presents with localized tenderness on palpation without deep ankle pain. Removal of these osteophytes, loose bodies, and inflamed synovium results in 50% of good or excellent results [14].

The patient is placed supine in anterior arthroscopy or prone in posterior arthroscopy, with the tourniquet placed at the tight. Usually, the traction is not necessary; otherwise, in some cases, a noninvasive ankle distractor is used. Routine joint distraction is not necessary to perform ankle arthroscopy. However, there are situations in which distraction helps to get access to a posterior osteochondral defect. A soft tissue distractor has been developed for better access to posterior lesions (Guhl distractor). This sterile strap is attached to a sterile belt, which is placed around the waist of the surgeon. When the surgeon leans backward, distraction of the ankle joint is obtained [12].

A 4.0 mm 30° arthroscope is introduced while the ankle is in full dorsiflexion, according to Van Dijk technique [12]. This protects the talar cartilage as it is covered by the tibial cartilage. For irrigation, normal saline is used; flow is obtained by gravity, although use of a pump system can also be applied.

The anteromedial portal is located medial to the tibialis anterior tendon at the level of the ankle joint. Care should be taken to avoid injury to the long saphenous vein and nerve usually located medial to the portal.

The anterolateral portal lies on the anterior joint line just lateral to the peroneus tertius tendon or alternatively lateral to the extensor digitorum longus tendons. The intermediate cutaneous branch of the superficial peroneal nerve lies in close proximity to this portal. Posteromedial and posterolateral coaxial portals lie parallel to the bimalleolar axis.

As suggested by Van Dijk in anteromedial impingements, the arthroscope remains introduced through the anterolateral portal, while the anteromedial portal is the working portal. In anterolateral impingement, the arthroscope is introduced through the anteromedial portal, in order to work in the anterolateral portal.

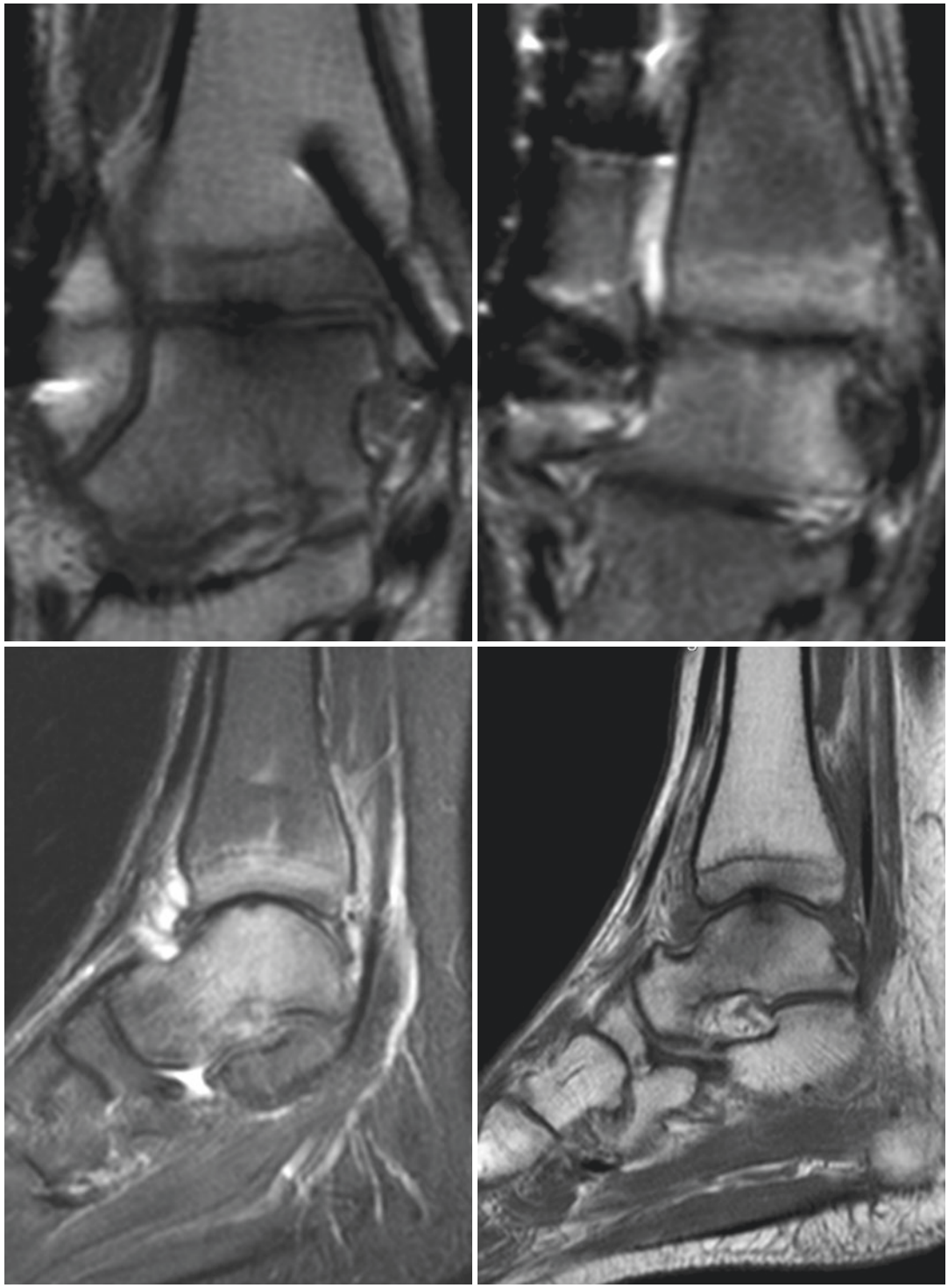

Fig. 22.1 A clinical case of posttraumatic ankle in a 20-year-old girl, with arthrofibrosis, OLT, and ankle joint narrowing

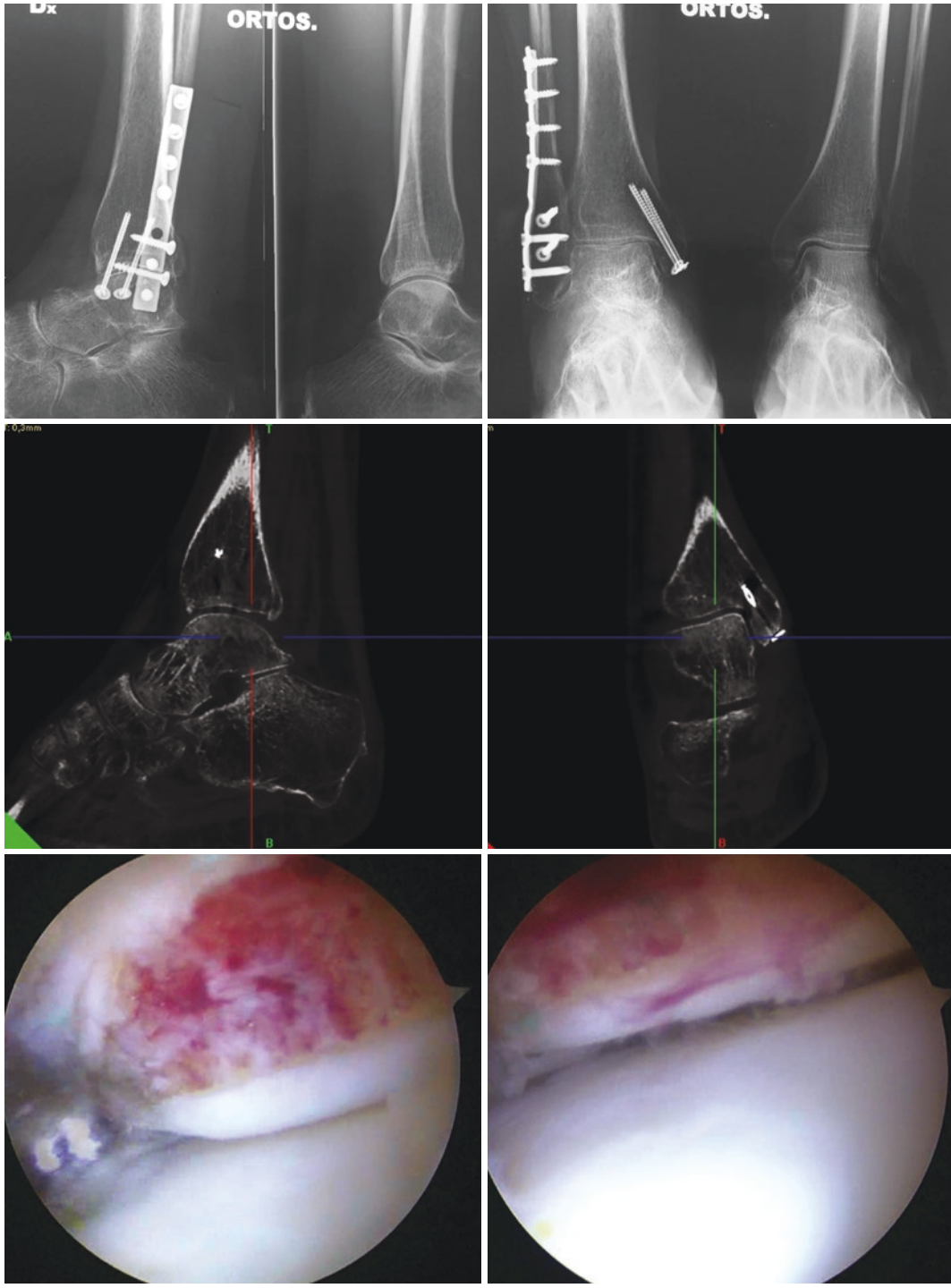

Fig. 22.2 Weight-bearing radiographs and CT scan showed a case of mild osteoarthritic ankle after trimalleolar fracture treated with arthroscopic debridement

One of the difficulties in rigid and arthritic ankles is finding the anatomical structures, especially at the beginning of the procedure; special attention must be done in the early stages, in order to release the joint space from the hypertrofic synovial and fibrous tissue.

The treatment of the osteochondral lesions of the talus (OLT) in the arthritic ankle is debated.

In our opinion, according to some other authors, asymptomatic OLT should not be treated. Many incidentally discovered OLT do not become symptomatic and are often unrelated to the trauma after which the imaging study that led to the detection of the OLT was performed. When, however, the OLT is the most likely source of pain and nonoperative treatment has failed, we recommend arthroscopic surgery for evaluation and treatment of the OLT [6].

Tibial and talar osteophytes can easily be detected during an arthroscopic procedure with the ankle in forced dorsiflexion. The capsule does not need to be detached to locate these osteophytes [12]. It is important to remember that in the most cases, osteophytes are the secondary manifestation of osteoarthritic changes [6, 7]. In these cases, the results could be worse compared to treatment of primary osteophytes without arthritic ankle joint (Fig. 22.3).

Special attention should be paid to the status of the joint as a whole and especially to the presence of loose bodies and the condition of the gutters.

It is not infrequent, especially for the less experienced arthroscopist, to have some component of "tunnel vision" and to address only the area of pathology without having a sense for the condition of the remainder of the joint. Unexpected pathology amenable to treatment may be found during the initial inspection of the joint, and providing adequate treatment to it will only improve the benefit from surgery to the patient. (Table 22.1).

Recently, Osti et al. concluded their study affirming that ankle arthroscopy and concomitant arthroscopic management of secondary injuries, when performed in selected patients with mild-to-moderate degenerative changes to the ankle, provide high rates of satisfaction and good functional results with positive impact on the quality of life [21].

In patients with anterior bony impingement, at a mean follow-up of 9 years, the AOFAS scores were still significantly improved, but age at surgery, radiographic changes, and concomitant cartilage lesions were negative prognostic factors [21].

Phisitikul et al. reported that arthroscopic debridement showed benefits not only in the treatment of arthritic disorders primarily involving synovium of the ankle joint but also in the treatment of rheumatoid arthritis, localized pigmented villonodular synovitis and hemophilic arthropathy [22].

22.4 Postoperative Rehabilitation

There is no consensus on postoperative rehabilitation and time to return to activity after ankle arthroscopy [23]. Osti et al. reported that all patients returned to full weight-bearing 6–8 weeks after surgery, especially in case of OLT, in order to allow osteoblasts to form new bone and chondroblasts to produce a matrix containing type II collagen and proteoglycans, all forming fibrocartilaginous tissue [21]. After this time, a hyaline-like cartilage with a high component of type II collagen can be detected [24], and the osteochondral defects are completely filled with mostly hyaline-like tissue [25].

The use of viscosupplementation (VS) in ankle osteoarthritis (OA) has been advocated for years. Carpenter et al. in 2008 compared ankle arthroscopy versus ankle arthroscopy combined with weekly intra-articular viscosupplementation with statistically significant improvement in regard to pain relief in the treatment of osteoarthritis of the ankle [26].

Migliore et al. in a review of 2011 reported that VS could be useful in ankle OA; nevertheless, future research should focus on obtaining sufficiently long-term follow-up (up to 1 year) on all patients using a systematic and prospective approach [27].

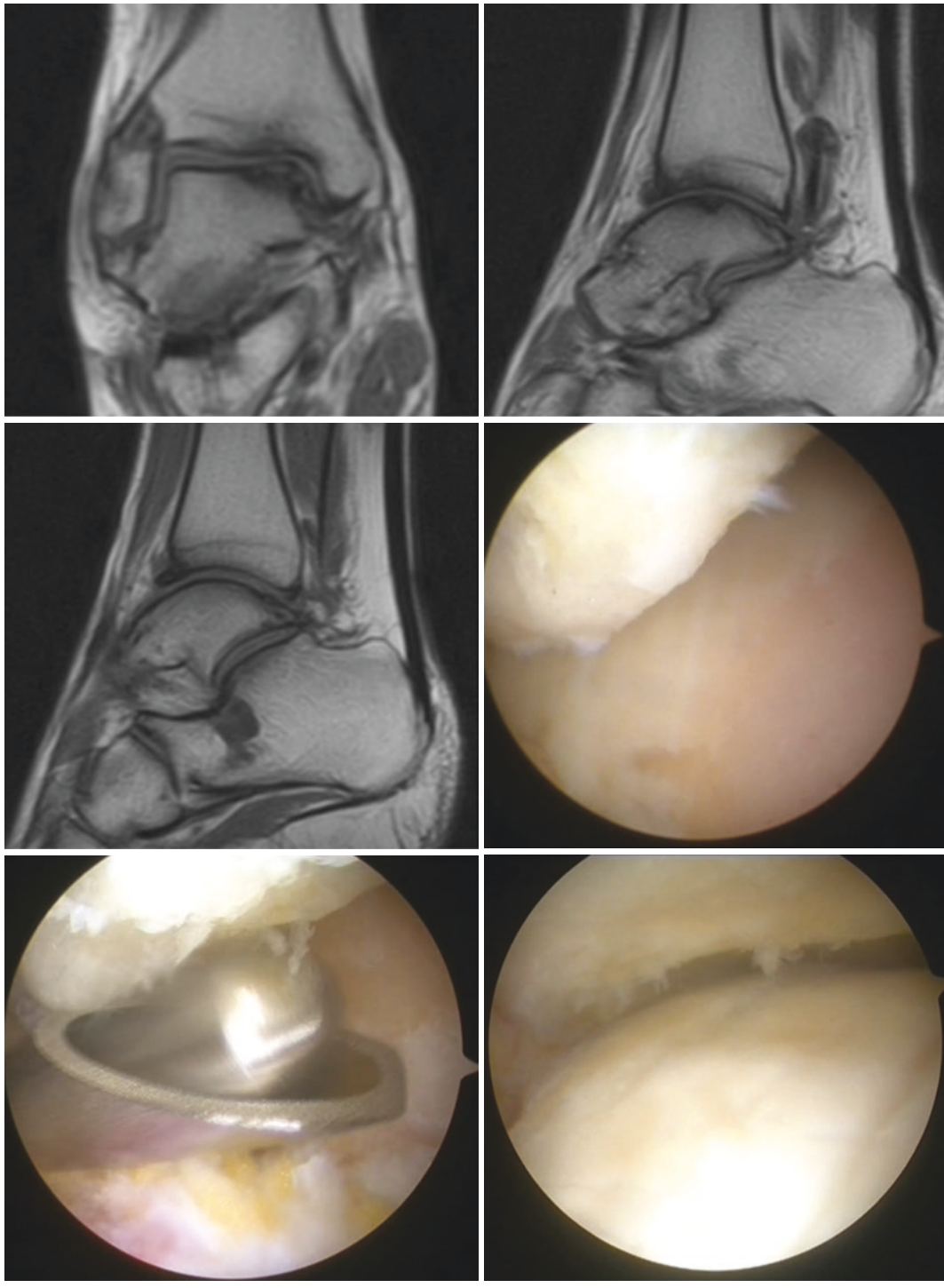

Fig. 22.3 A clinical case of a symptomatic osteophyte with an asymptomatic OLT. In this case, only partial removal of osteophyte and soft tissue debridement was performed

22.5 Complications

Complications such as neurologic, tendon, and ligament injuries, wound complications, infection, and instrument breakage can occur in foot and ankle arthroscopy. The most common complication is neurologic injury, with the SPN being the most commonly injured nerve [28]. The dorsiflexion method for anterior ankle arthroscopy has been evaluated and suggested to decrease the overall complication rate to 3.5%, with the incidence of neurologic injury at 1.9% [28]. In a recent systematic review, the overall complication rate in ankle arthroscopy for anterolateral impingement using the standard 2-portal technique was 4% [12, 29].

Infection is a potential complication in any operative procedure, but the incidence is less than 0.2%.

Most common complication after ankle arthroscopic debridement is the inefficacy of the procedure, including persistent pain and re-operation. Prognostic factors contributing to a poor outcome include chondral lesions, advanced age, and history of a previous trauma. Careful surgical indications based on accurate history, physical examination, and appropriate imaging studies will help to minimize the risk of surgical failure [30, 31] (Table 22.2).

Table 22.1 Authors' preferences in performing arthroscopic ankle debridement

✓ The authors recommend to perform the procedure with standard arthroscopic instrumentation (4-mm diameter). Traction should be avoided
✓ Treatment: Symptomatic osteochondral lesions of the talar dome: Asymptomatic lesions of the talar dome do not need surgery
✓ In case of mild ankle osteoarthritis, it is important to remove inflamed intra-articular soft tissue. Do not remove entirely osteophytes otherwise could create an arthritic ankle instability
✓ Arthroscopic debridement in an arthritic ankle is performed to reduce pain; improvement of ankle R.O.M. is not sure. Patients should be appropriately counseled of this risk before surgery

22.6 Conclusion

In conclusion, the arthroscopic approach represents an additional treatment option for those cases of osteoarthritis of the ankle in which conservative treatment is ineffective and when is inappropriate to proceed with a joint replacement or other major surgeries on the ankle joint. Furthermore, ankle arthroscopy may be useful in identifying and managing chronic pain caused by various intra-articular lesions after ankle trauma.

Table 22.2 Published results of arthroscopic debridement in posttraumatic ankles

	No. patients	F-U (months)	Results	Complications
Lui et al. [19]	5	32.6	AOFAS pre-op: 63.8 AOFAS post-op: 88.6	Not reported
Utsugi [8]	33	43	Improved articular function In 89% of patients In 32/33 patients, R.O.M. did not change	Not reported
Kim et al. [13]	19: Hardware removal—conservative treatment	12	AOFAS: from 74 to 76	2: Superficial wound infection
	22: arthroscopy and hardware removal		AOFAS: from 75 to 85	1: Superficial wound infection 3: Mild pain at arthroscopic portal at 6 months
Dawe [30]	49	48	75% good results 50% were able return to normal Activity after surgery	Re-operation 10% after 1 year 34% after 4 years

However, arthroscopic debridement of the ankle can offer relief to approximately two-thirds of patients; it is mandatory to inform patients that the degree of improvement could be limited and not-definitive.

Moreover, in many cases, intra-articular pathology was frequently more severe than estimated preoperatively, and patients should be properly instructed of the risk of re-operation after an ankle arthroscopy for ankle arthritis.

Arthroscopic management for chronic pathology after ankle fractures has showed significant benefits, and although evidence on its use in the acute setting is limited, we feel there is an important role for its use as an adjunct to the management of fractures in the injured footballer, in both aiding diagnosis and optimizing management.

While some controversy exists regarding arthroscopic debridement of early ankle osteoarthritis and impingement, there is evidence to support benefit of the treatment. By allowing direct, minimally invasive visualization and manipulation of intra-articular structures, ankle arthroscopy offers an important surgical option for the properly selected patient.

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Ankle Fusion by Screws

23

Matteo Guelfi, Jordi Vega, Francesc Malagelada, Miquel Dalmau-Pastor, and Marco G. B. Guelfi

23.1 Introduction

The ankle is one of the few major joints, in which arthrodesis is a valid treatment for end-stage arthritis. The fusion of the tibiotalar joint can result in a pain-free ankle that withstands the normal daily activities, even in a young, high-demand, active individual [1]. In addition, the alternative of total-ankle replacement is not a good option for all patients and entails higher complication and revision rates [2].

A variety of surgical approaches and methods of fixation have been described for ankle arthrodesis. Open or miniopen techniques have traditionally been associated with a number of complications like nonunion, delayed union, malunion, infection, wound necrosis, and neurovascular injury [1, 3].

Arthroscopic ankle arthrodesis (AAA) has been shown to be an effective option with high fusion rates and low complication rates [4]. Arthroscopic techniques compared to open demonstrated quicker time to union with equivalent or higher union rates and faster recovery [5]. In addi-

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tion, AAA techniques require shorter hospital stay and reduced costs, both relevant nowadays [6, 7].

The aim of this chapter is to describe the surgical technique, indications, and limitations of ankle fusion using screws and anterior ankle arthroscopy.

23.2 Indications and Limits

General indications for tibiotalar fusion include end-stage arthritis from any cause that cannot be controlled with conservative treatment, which includes physiotherapy, orthosis, NSAID treatment, and viscosupplementation. As previously reported in this book, ankle arthritis is most commonly posttraumatic in nature followed by primary arthritis due to rheumatoid arthritis, inflammatory arthropathy, infection, Charcot neuroarthropathies, and avascular necrosis.

Because of the minimal aggression to soft tissues, AAA is particularly suitable for patients with associated diseases that compromise wound healing like diabetes, chronic vascular disease, coagulopathies, and prolonged steroid therapy [8, 9]. Posttraumatic cases often present with previous scars and metalwork; in those, wound healing would be compromised when using open techniques. To avoid previous metalwork and scars, it may be necessary to modify the original technique when placing the screws [10].

The presence of active infection and active Charcot arthropathy is an absolute contraindication for both open and arthroscopic techniques. Avascular necrosis of the talus is a relative contraindication to traditional open arthrodesis, although it can be considered for the arthroscopic technique [10].

Correction of significant deformity is a relative contraindication to AAA. Historically, the AAA has been performed as an in situ fusion; fixed significant varus or valgus malalignments have represented the primary contraindication for AAA [1]. However, more recently, some authors have argued that with greater experience, 15° of deformity can be accepted [11].

Surgeon experience and steep learning curve are other limitations of AAA. Arthroscopy in posttraumatic cases with joint narrowing and big anterior osteophytes is challenging and requires an experienced ankle arthroscopist [11].

23.3 Surgical Technique

Authors' preferences are reported in Table 23.1.

23.3.1 Patient Positioning

The patient is positioned supine under spinal or general anesthesia. The use of a flow pump is not necessary and adequate flow is obtained by gravity. A thigh tourniquet is recommended and a bolster under the ipsilateral buttock helps control limb rotation. The affected extremity can rest on the table or on a thigh support below the knee. With the latter, both the hip and the knee are flexed approximately 45°, placing the leg parallel to the floor and the ankle free for range of motion.

The arthroscopy stack and C-arm are on the ipsilateral side while keeping the medial aspect free for screw insertion gestures.

A soft tissue distractor can be helpful to access the joint in tight ankles and to reach the most posterior cartilage. In any case, it is not routinely necessary and will depend on the surgeon's preference.

23.3.2 Surgical Instrumentations

A standard "knee arthroscopy set" comprised of a 4–4.5 mm 30° scope, and 3.5 or 4.5 mm motorized shaver and burr are used. Curettes and small osteotomes are helpful in removing articular cartilage and exposing the subchondral bone.

Table 23.1 Authors' preferences in performing arthroscopic ankle arthrodesis

– Spinal anesthesia and thigh tourniquet
– Sandbag under the ipsilateral buttock and affected extremity on a thigh holder located under the knee
– Assistant hand-made distraction when necessary
– 4.5 mm 30° scope and 3.5 or 4.5 mm full-radius shaver and burr ("knee arthroscopy set")
– Preparation of both medial and lateral gutter with curettes. Medial gutter requires switching of portals (anterolateral as viewing and anteromedial as working portal)
– If it is difficult reaching posteriorly: resections of more bone anteriorly and use of angled curettes
– Fixation with two crossed 6.5-mm cannulated screws

Finally, two 6.5 mm compression screws and instruments for their insertion are necessary for internal fixation.

23.3.3 Surgical Approach and Procedure

Routine anteromedial and anterolateral ankle portals are performed. To achieve initial visualization, it is seldom necessary to perform an anterior joint synovectomy with a shaver. Care must be taken to avoid damaging noble soft tissues by pointing toward the articular surfaces with the shaver blade. Once clear visualization is achieved, tibial osteophytes are removed with a burr. This step is important to allow neutral flexion at the time of fixation (Figs. 23.1, 23.2, and 23.3).

The articular cartilage of the tibial plafond and talar dome is completely removed, progressing from anterior to posterior with curettes, osteotomes, and burrs. Introducing a curette into each gutter is helpful in preparing the lateral and medial articular surfaces of the talus and malleoli. Although some authors suggest not to prepare the lateral gutter [11], it is the authors' preference to prepare both gutters in the same way.

If the posterior part of the joint is difficult to reach, one can use angled curettes, resect more bone anteriorly, or perform a posterolateral portal. In addition, sectioning the ATFL through a small anterolateral incision allows better joint distraction [12].

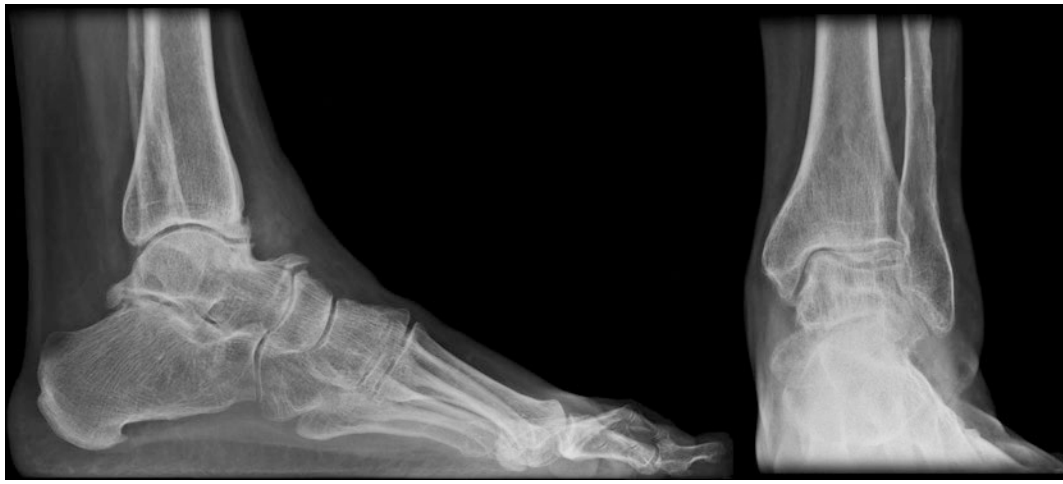

Fig. 23.1 Lateral and Anteroposterior view of patient with ankle arthritis. Joint narrowing and osteophytes on the tibia and talus are clearly seen on lateral view

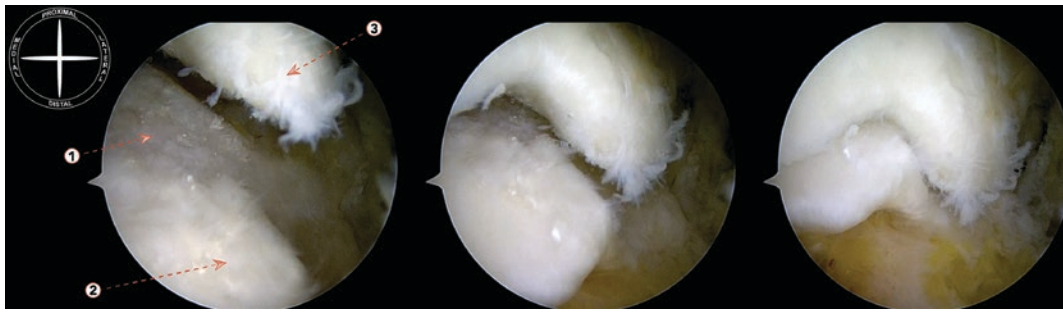

Fig. 23.2 Arthroscopic views showing talar cartilage (1), osteophytes on talar neck (2), and on distal tibia (3). Ankle joint is in plantarflexion (left), in neutral position (middle), and in dorsiflexion (right). In the latter, osteophytes perfectly engage with each other

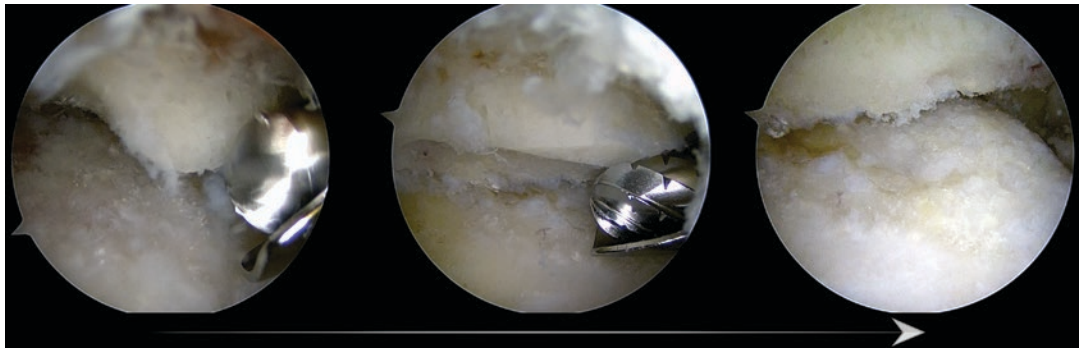

Fig. 23.3 Removing tibial osteophytes with a burr allows better visualization

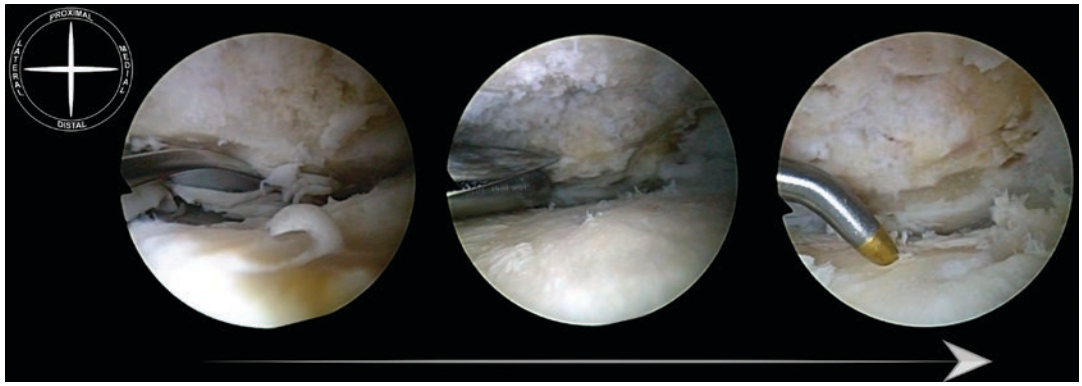

Fig. 23.4 The articular surfaces of both tibia and talar dome are prepared. Before articular cartilage is removed with angled curettes (left), a burr is used to expose underlying healthy cancellous bone (middle). Finally, microfractures (right) at both tibia and talar sides are performed

After excision of the articular cartilage, a burr is used to remove the thin layer of subchondral until bleeding cancellous bone is exposed.

At this point, axial corrections can be achieved by removing a thicker layer of bone on the medial or lateral aspect depending on the deformity. Surgeons must be aware that maintaining congruent bone contours of the talus and tibia is essential to maximize the contact of both surfaces and to achieve bone fusion. Some surgeons perform small holes in the talar dome and tibial plafond to increase bone bleeding in the area (Figs. 23.4 and 23.5, Video 23.1).

23.3.4 Fixation

Once the arthroscopic preparation of the joint surfaces is achieved, the fixation can take place under fluoroscopic control. The ankle must be fixed in neutral flexion, 0–5° of valgus and slight external rotation (5–10°).

In patients with an equinus foot deformity in whom neutral ankle position is not obtained, a percutaneous Achilles tendon lengthening may be necessary.

The arthrodesis is fixed with two 6.5 mm cannulated screws placed in a crossed or parallel fashion from the medial side. The authors' preference is to use two crossed, 6.5 mm cannulated screws.

The screws form an angle of about 50° on the anteroposterior view and 10° on the lateral, with the medial screw going slightly anterior and the lateral slightly posterior. Care must be taken to avoid penetration of the subtalar joint.

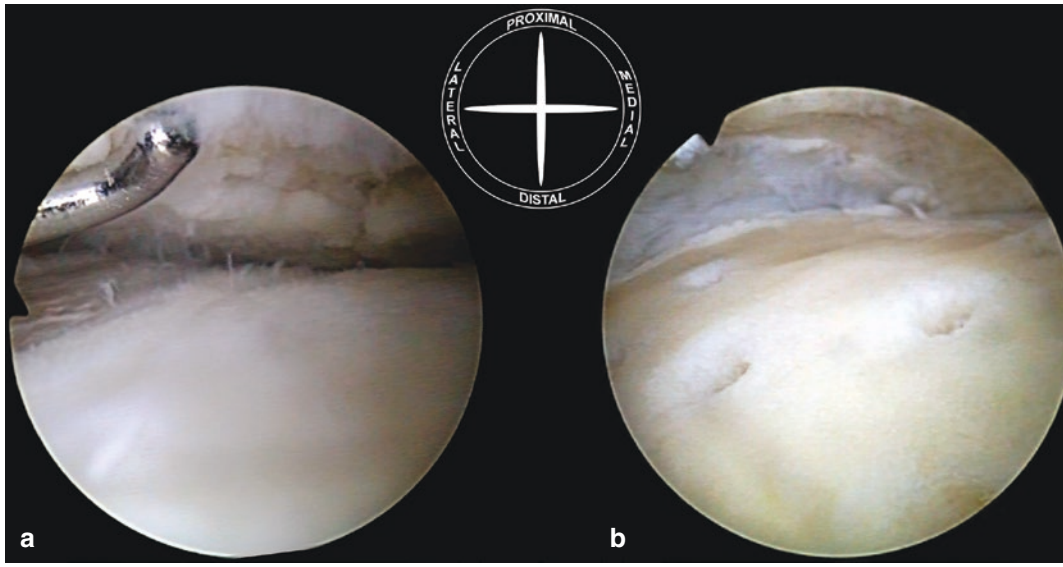

Fig. 23.5 Arthroscopic view from anteromedial portal before (**a**) and after (**b**) articular surfaces preparation

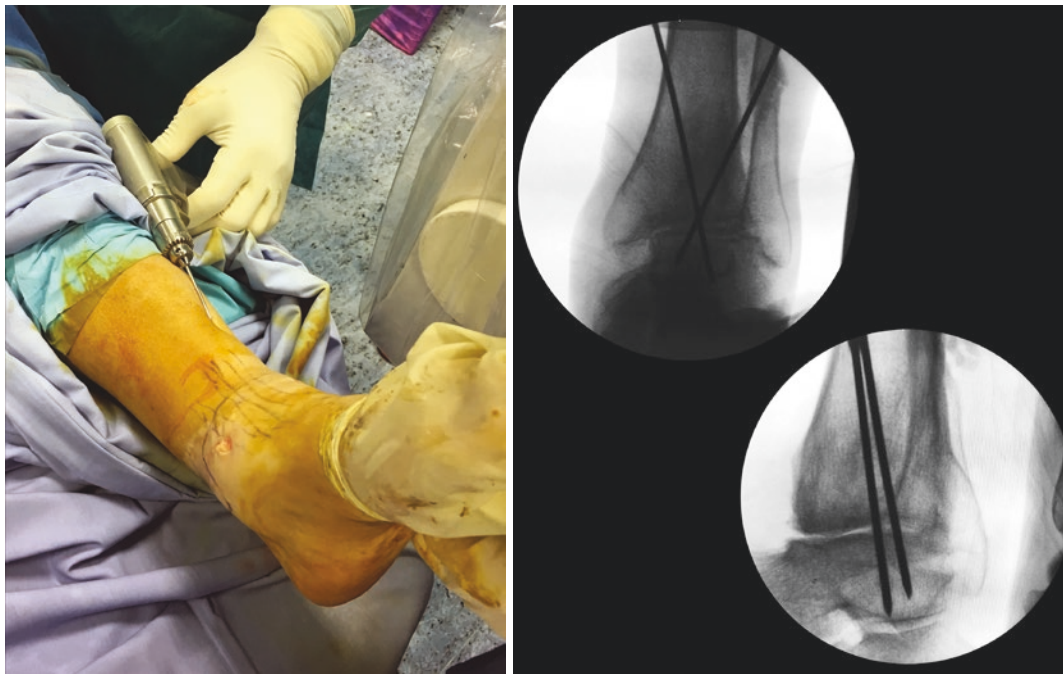

Fig. 23.6 Two crossed guide wires are placed with the ankle held in proper position (left). Good position of guide wires is confirmed using anteroposterior and lateral fluoroscopic views (right)

Fluoroscopic anteroposterior, lateral, and oblique views are obtained to check for screw placement and ankle position.

Wounds are closed with nonabsorbable sutures. A sterile compressive dressing and a walking boot are applied (Figs. 23.6 and 23.7).

23.3.5 Postoperative

Patients are discharged from hospital the day after surgery. Sutures are removed 1 week postoperatively. The walking boot is worn for approximately 3 months or until radiographic fusion is noted. Weight-bearing as tolerated is allowed at 4 weeks after surgery. Radiographs are performed at 1, 3, 6, and 12 months of follow-up.

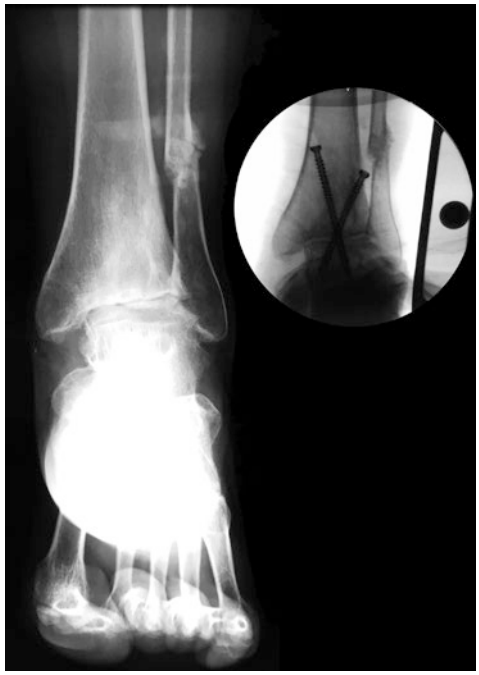

Fig. 23.7 Preoperative anteroposterior radiograph (left) and final fluoroscopic anteroposterior control after arthroscopic ankle arthrodesis (right)

23.4 Results

The published results of AAA are reported in Table 23.2 [11, 13–19]. These studies revealed fusion rates between 90 and 100%. Three articles directly compared open versus arthroscopic techniques reporting equivalent fusion rates and clinical results with shorter hospital stays and operative times and faster fusion in the arthroscopic group [5, 15–17].

Complications reported for both open and arthroscopic techniques include infection, neurovascular injury, malunion, and nonunion. The latter is the main complication and has been reported in six of eight studies assessed in Table 23.2. These cases were significantly associated to smoking and successfully achieved union after an open technique [11, 14].

Finally, ankle fusion causes increased motion in the neighboring joints, resulting in long-term subtalar degenerative changes in 10–60% of patients [13, 14]. This has been documented as a cause of patient dissatisfaction after ankle fusion.

Table 23.2 Published results of arthroscopic ankle arthrodesis

Article	No.	Follow-up (months)	Union rate (%)	Good results (%)	Complications (no. cases)
Winson IG. J Bone Joint Surg 2005 [11]	105	65	92	79	11 major, 27 minor
Gougoulias NE. Foot Ankle Int 2007 [13]	78	21	98	80	2 major, 2 minor
Dannawi Z. Foot Ankle Surg 2011 [14]	55	63	91	82	5 major, 8 minor
Myerson MS. Clin orthop Relat Res 1991 [15]	17	23	94	–	None
Nielsen K. Foot Ankle Surg 2008 [16]	58	–	90		3 major, 15 minor
O'Brien T. Foot Ankle Int 1999 [17]	19	–	84	–	3 major
Bai Z. Foot Ankle Int 2013 [18]	10	21	100	80	1 minor
Yoshimura I. Arthroscopy 2012 [19]	50	42	92	–	4 major

23.5 Conclusions

The AAA is a suitable option for most patients who undergo an ankle fusion. It has showed favorable long-term results in cases of end-stage ankle arthritis. The arthroscopic technique shows equivalent clinical results and faster recovery when compared to open. Minimal soft tissue aggression makes AAA ideal for those patients with compromised wound-healing potential (e.g., diabetics). The surgeon must be aware that performing an arthroscopic ankle fusion may be challenging and broad experience in ankle arthroscopy is required.

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24 Arthroscopic Assisted Ankle Fusion by Retrograde Intramedullary Nail

Francesco Allegra, Giovanni Corsini, Giovanni Pizza, Roberto Zannoni, and Andrea Billi

24.1 Indications

Symptomatic degenerative joint disease of the tibiotalar joint (so-called upper ankle), together with the involvement of the subtalar joint (so-called lower ankle), is the correct indication for intramedullary nailing arthrodesis by open technique [1]. Arthroscopy was suggested only to check the internal joint associated lesions and to treat with an articular debridement [2]. In the younger, active patient, the most common indication for arthrodesis of the ankle is early post-traumatic arthritis, the risk of which is increased where congruity and the dynamic function are altered. Surgical techniques less invasive and less aggressive to the joint has been suggested to spare tissue and to reduce the joint surgical morbidity [3, 4]. Arthrodesis may be achieved by open or arthroscopic means, with the latter being unable to correct significant axial deformity of leg or ankle. Although these restictions in indications, the use of scope as needful tool has become popular by years, confirming its important role in this surgical procedure [5–8]. Partial loss of the talus is a rare but devastating condition for the ankle joint: it may be caused by trauma, avascular necrosis, and previous surgical interventions. With severe further loss of bone stock at the distal tibia, stability can be achieved only by using an intramedullary nail for ankle fusion [9]. Although the survivorship of the second generation of total ankle replacements has improved, it is known that it still does not match that of total hip and knee replacements. A higher level of reoperation after total ankle replacement has been reported: arthrodesis remains the primary revision procedure in patients with aseptic failure of replacement. Failed surgery addressed to bony infection treatment such as talectomy or to tumors is one of the main indications for this technique (Fig. 24.1). For these patients, it is better to stage arthrodesis after removal of all components and after debridement of the joint: external fixation before further bone surgery is required until the infection has been eliminated and skin recovery is complete. Tibiocalcaneal arthrodesis may be the only mean for obtaining a painless and stable limb in the presence of talar bone loss: this procedure requires an intact bony stock from the head and neck of the talus as much as possible [10]. The advantage of intramedullary fixed-angle devices is that they provide good primary stability even in the presence of low-quality bone, and they cause limited soft-tissue damage compared to the use of fixation screws, external fixators, or compression plates. Malalignment hazards while doing

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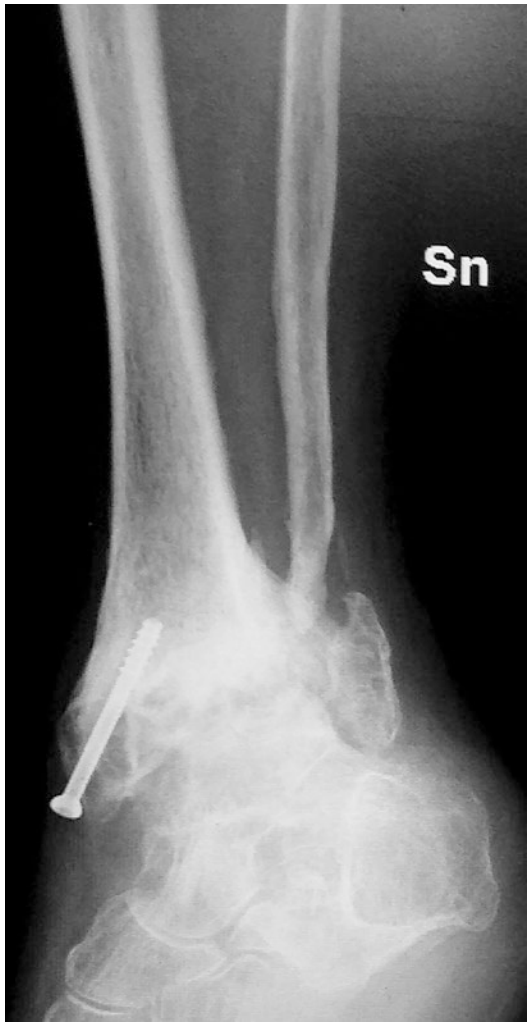

Fig. 24.1 Plain X-ray scan shows complete overthrow of the ankle joint after a deep infection secondary to fracture with large exposition, recovered at time of this surgery

ankle fusion are minimized by respecting the shape of the ankle mortise because no osteotomy of the lateral malleolus is performed.

The use of arthroscopic procedure in ankle arthritis treatment has been proposed by years, belonging to the story of ankle arthroscopy. The large advantage was pointed out in achieving the best visualization of different joint compartments while performing the arthrodesis, obtained thanks to the improvements, as both techniques and hardware were very poor at that time [11]. The arthroscopic approach in ankle arthrodesis has shown better pain control during the postoperative period, less soft-tissue and skin morbidity, and a faster return to normal life after rehabilitation, with reduced costs compared with open arthrodesis [12]. The use of arthroscopy has gained a proper place in procedures for the treatment of ankle arthrodesis [13]. Nevertheless some concerns have been expressed regarding arthroscopic ankle fusion, including the ability of correcting significant angular deformities or bone loss with the arthroscopic technique (Fig. 24.2).

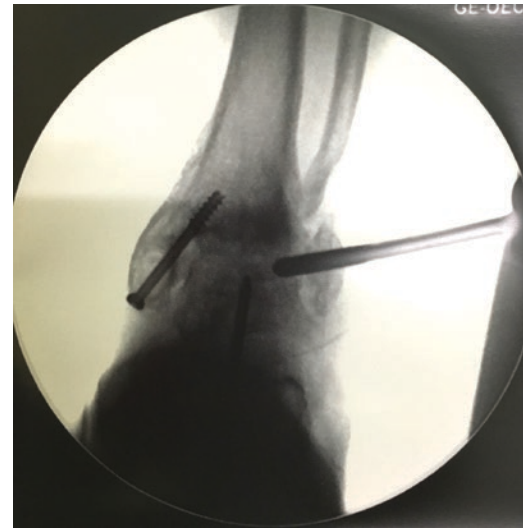

Fig. 24.2 Intraoperative fluoroscopy shows correct assessment of the ankle joint under direct scope visualization placed on external side

24.2 Surgical Procedure

The patient is assessed in supine fashion. With a tight tourniquet, the foot placed beyond the end of the surgical bed. Fluoroscopy before surgery is performed to evaluate the reciprocity of different bones and their shape. An ankle traction system is recommended to make the arthroscopic step easier. Two anterior standard portals are enough to proceed inside the joint and to make an accurate articular exploration. The dynamic evaluation of gutters and compartments is suggested, moving the ankle to detect the presence of some different-sized

bony fragments that can appear to be unstable and not safe to guarantee a strong nail compression. All hardware present inside or around the joint must be removed at this step. Arthroscopy starts with a intrarticular soft-tissue removal to completely free the bony surfaces from their scar or synovial coverage, using a proper-sized shaver blade. The bone could be soft and easily compressed and removed even by scope suction: in such cases it should be left in place, avoiding pauperization of the bone stock. Loose bodies and dead bony fragments are taken out using a grasper. Then, a complete cleanup of the joint by removing all the remaining cartilage layers or fragments just beyond the cartilage calcified plate is appropriate to refresh the bone. A single- or double-sharp ring curette proceeds from the medial and lateral portal to detach both from the tibial plafond and talar dome, with particular attention to the medial gutter where contact between the two bones must be achieved. The use of a round burr or an acromionizer blade is seldom suggested, taking care to respect the soft sick deep bone and not to make grooves or holes deeply inside it. In some cases, resection of anterior tibiotalar osteophytes is required to access the joint better. Sometimes is necessary to model the shapes of the two tibial and talar extremities aiming to obtain a more congruent one. It is preferred not to extend the fusion to the talofibular aspect of the joint and to perform a simple shaving of the lateral gutter to allow apposition of the tibiotalar surfaces and obtain a fusion in a better anatomic position. Once this accurate preparation is completed and adequate bleeding achieved, the traction is removed. The perfection of the joint congruency is evaluated, and the K-wire of the cannulated nail is drilled into the calcaneus through the talus under fluoroscopy, until the wire emerges inside the articular space. If its placement appears by arthroscopic visualization to be incorrect or shifted too far laterally or medially, the K-wire must be removed and drilled again until its perfect central placement is achieved (Fig. 24.3).

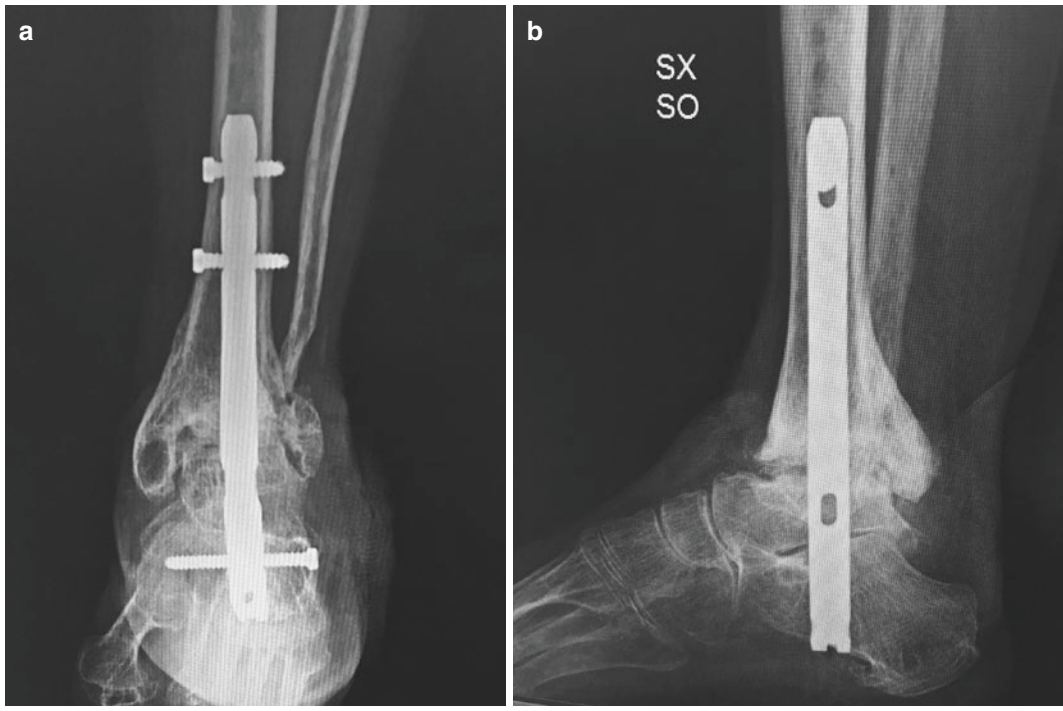

Fig. 24.3 (**a**, **b**) Final X-ray "scans" in coronal and sagittal planes "show" the good position of nail, screws, and the compression between tibia, talus, and calcaneus

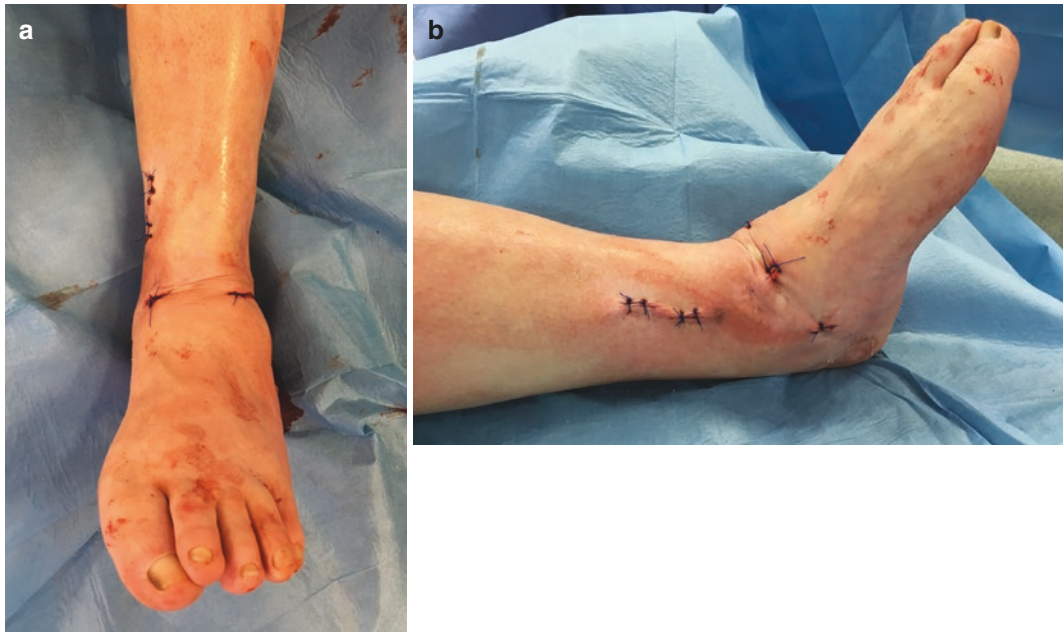

Fig. 24.4 (**a**, **b**) Immediate postoperative aspect of joint shows good correction of the deformity and the presence of small skin approaches and portals, sparing skin traumatized by previous surgeries and infection

Now the external procedure to fix the joint with the nail is started, opening a 2-cm incision on the heel. The tibiocalcaneal arthrodesis is performed using a retrograde intramedullary nail. Different devices are available, with different sizes, similar features, bent or straight, and with lateral inclination in the valgus to enhance the fit through the calcaneus. The driving end diameter is generally common to all sizes. The nail design provides for distal fixation with a talar and/or lateral and posterior bicortical calcaneal screw to create a fixed-angle construct. The nails may be used with static or dynamic compression using a dedicated instrumentation for strongly pressing together tibia, talus, and calcaneus, thus providing sufficient stability for tibiocalcaneal arthrodesis. The multiplanar screw fixation guarantees a single balanced compression and permits optimizing the stability and alignment of the arthrodesis. Once contact between viable bone in the tibia and the calcaneus had been obtained, intramedullary nail arthrodesis is performed. Because internal compression through the talar locking screw, which is part of the nail system, could not be used, patients are managed either with external compression on the calcaneus only, or with impaction of the fusion site using controlled tapping of the intramedullary nail following distal interlocking. A splint is generally applied until the soft-tissue healing is complete. Then, patients are supplied with a walking cast for some weeks until the plain X-ray scan shows bony fusion between the ankle articular surfaces. Some patients require fitting with orthopedic footwear incorporating a heel-to-toe rocker sole (Fig. 24.4).

24.3 Discussion

It has been suggested by Coester et al. [14] and Fuchs et al. [15] that arthrodesis of the ankle leads to osteoarthritis of the joints of the ipsilateral hindfoot and midfoot. These degenerative changes are said to be caused by altered biomechanics following increased movement to compensate for the loss of ankle motion. Coester et al. [14] and Fuchs et al. [15] assumed this follows the surgery, but equally it could have been present preoperatively. This possibility therefore needs to be considered when evaluating the

results of arthrodesis of the ankle. It cannot be stated that ipsilateral arthritis in the foot is an absolute long-term effect of arthrodesis, as it may be predetermined and multifactorial. In his study, Sheridan et al. [16] demonstrated that hind- and midfoot arthritis is very common with coexistent ankle arthritis and that the subtalar joint is usually most severely affected. Gait analysis suggests increased ipsilateral movement in the foot after arthrodesis of the ankle [17]. A radiographic evaluation of tarsal movement following ankle arthrodesis by Abdo and Wasilewski [18] did not show a significant difference between ipsilateral and contralateral joints. Nonunion, an important complication following foot and ankle arthrodesis, causes substantial morbidity and disability [19]. In patients undergoing hindfoot and ankle arthrodesis, autogenous bone graft or a suitable alternative is often used to promote osseous fusion across the joint in case of poor bone stock preoperatively. Klos et al. [10] consider intramedullary nailing for tibiocalcaneal arthrodesis to be a minimally invasive internal-fixation technique that promises a good outcome in a comparatively short time. They concluded that if the loss of the talus is not caused by infection, this technique is superior to external fixation and plate techniques. In a recent review [20], Cottino et al. examined the results of arthroscopic ankle arthrodesis by screw fixation and confirmed the excellent results compared to open technique, pointing out the popularity of this technique has been increased by the evident advantages in the past two decades. The coupling of the arthroscopic procedure with tibiocalcaneal arthrodesis by retrograde intramedullary nail permits bypassing the limits of an insufficiently stable hardware construct with all the advantages of a low morbidity technique.

24.4 Conclusions

Internal fixation is a safe and biomechanically stable method to obtain a solid ankle fusion; it generates good to excellent results in most patients. Prompt bone healing can be expected and allows a functional rehabilitation with full weight-bearing. A meticulous resection of all cartilage and sclerotic bone as provided by the arthroscopic procedure represents an atraumatic surgical technique, being essential for preventing major complications. The possibility to treat ankles with marked deformity successfully, along with a slightly shorter time to union, reduced complication rates, and clear cost benefits compared with open surgery, makes arthroscopic ankle fusion by retrograde nail a safe and reliable technique.

Traps	Tricks
Low visualization of the joint	– Clean up accurately both sides of the joint – Use curettes and round bur blades
Difficulties entering the joint with cannula because the articular space is narrow	– Use traction at maximum load, then release – Use small burr blades at beginning
Difficulties in respecting the talar and tibial shape	– During the procedure check frequently for congruency and coplaning
Placement of the intramedullary nail guide appears not to be centered	– Check tip of the K-wire nail guide with both scope and fluoroscopy – Leave in place when correct position is achieved

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25 Subtalar Joint Arthritis: Treatment and Arthroscopic Lateral Procedure

Yoshiharu Shimozono, John G. Kennedy, and James W. Stone

25.1 Introduction

Conservative treatment for subtalar joint arthritis includes oral nonsteroidal anti-inflammatory drugs, analgesics, orthotics, and physical therapy. Oral nonsteroidal anti-inflammatory drugs are often attempted first. Sinus tarsi or subtalar joint injections consisting of local anesthetic and a corticosteroid are helpful in temporarily relieving symptoms and inflammation. The injection is also a good diagnostic maneuver as complete pain relief after local anesthetic injection confirms that the subtalar pathology is responsible for the patient's symptoms. Biologics, including platelet-rich plasma and concentrated bone marrow aspirate, are also useful options as immune or inflammatory modulatory agents primarily. Orthotics are commonly used to address hindfoot malalignment. In some patients with chronic subtalar instability, physical therapy focusing on proprioception training and peroneal strengthening may be of benefit [1, 2].

If conservative treatment fails, then operative treatment, including arthroscopic debridement or subtalar joint arthrodesis, should be considered. While arthroscopic debridement has no clear indication criteria and lacks the evidence of effectiveness for long-term benefit for subtalar joint arthritis, it may be useful in some cases. Subtalar arthrodesis is an accepted surgical procedure for treating posttraumatic osteoarthritis and rheumatoid disease. In general, open subtalar arthrodesis is preferred for pathologies involving significant hindfoot malalignment, bone loss of the talus or calcaneus, and prior subtalar joint non−/malunion. Excellent outcomes of arthroscopic subtalar arthrodesis have been documented in the orthopedic literature in patients with degenerative joints but minimal deformity. The arthroscopic approach has several potential advantages compared with open arthrodesis, including smaller incisions, fewer morbidities, and the theoretical preservation of blood supply, resulting in higher fusion rates and faster fusion times. The arthroscopic procedure may be performed using either a lateral or a posterior approach. This chapter will focus on the lateral approach to arthroscopic subtalar arthrodesis.

25.2 Preoperative Preparation/ Positioning

Subtalar joint arthroscopy is performed using small joint instrumentation including a 2.7 mm arthroscope with both 30° and 70° viewing, along with small joint shavers and burrs 2–3.5 mm diameter. If a very tight joint is encountered, then it is useful to have a 1.9 mm arthroscope available. As arthrodesis of the joint requires removal

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of any remaining articular cartilage, large joint motorized instruments including shavers and abraders of 3.5–4 mm diameter can often be used and are more efficient at removal of cartilage and debris from the joint. Cartilage resection and subchondral preparation are performed with curettes, chisels, and burrs. The use of distraction facilitates joint visualization. A spreader or distractor using Kirschner wires (K-wires) can be used. The fusion is fixed with cannulated screws (6 mm or more in diameter) under fluoroscopy.

A tourniquet is placed around the upper thigh. For the lateral approach, the patient is in the lateral or "semilateral" decubitus position with a bump positioned beneath the hip. The operated ankle is placed on a firm support under the lower leg. In this position, the foot can be forced into varus position to open the subtalar joint, thereby facilitating arthroscopic instrument access and the arthroscopic procedure.

25.3 Operative Technique

Establishing the portal sites precisely is crucial to allow proper access to the joint and to perform the procedure smoothly. As the subtalar joint has a unique geometry with complex curved articular surfaces, which are highly congruent, arthroscopic visualization can be challenging. Fluoroscopy can be used to confirm portal location if necessary. Several portals for the lateral approach have been described, including anterolateral, posterolateral, middle, accessory anterolateral, and accessory posterolateral portals [3]. With lateral subtalar arthroscopy, it has been demonstrated in cadavers that 90% of the posterior facet of the subtalar joint can be resected using only the anterolateral and middle portals, leading some surgeons to avoid creating the posterolateral portal, which has the potential for sural nerve and small saphenous vein injury [4]. For this reason, one senior author (JGK) prefers the 2-portal lateral approach (anterolateral and middle portals). The other senior author (JWS) routinely uses the posterolateral portal (the same portal utilized routine ankle joint arthroscopy, positioned immediately adjacent to the lateral margin of the Achilles tendon 1 cm distal to the level of the ankle joint) as it allows for a dedicated inflow portal and for direct posterior instrumentation of the posterior one-third of the joint.

25.3.1 Portal Placement

The anterolateral portal is placed 2 cm anterior and 1 cm distal to the tip of the lateral malleolus (distal portion of the sinus tarsi). This portal is used primarily as a viewing portal. The middle portal is placed just distal and inferior to the tip of the lateral malleolus, and used primarily as a working portal. If required, the posterolateral portal is placed approximately 2 cm posterior and 1 cm proximal to the tip of the lateral malleolus, close to the Achilles tendon to avoid injury to the sural nerve (Fig. 25.1). If ankle arthroscopy is performed prior to subtalar joint arthroscopy, then the posterolateral portal used for that procedure may be redirected into the posterior subtalar joint.

The anterolateral portal is identified first with palpation and a spinal needle is directed into the anterior subtalar joint. The entry point is just posterior to the talocalcaneal interosseous ligament. The anterior subtalar joint is insufflated with 10 cc of saline and a small skin incision is made with a #15 blade scalpel, followed by blunt dissection down to the interosseous talocalcaneal ligament. The nick and spread technique for portal creation should be performed in order to minimize the chance of nerve injury, incising only the skin, then spreading the subcutaneous tissues with a mosquito clamp, and entering the joint with a blunt trochar. The arthroscope is then introduced. The middle portal is created through a small superficial incision under direct arthroscopic visualization from the anterolateral portal.

25.3.2 Joint Preparation

As a first step, a primary synovectomy and debridement usually via the middle portal are necessary for better visualization (Fig. 25.2). A

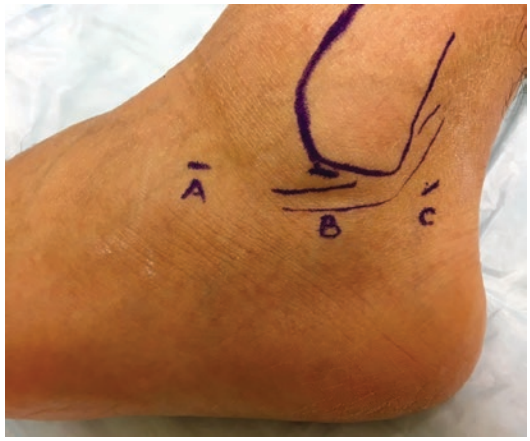

Fig. 25.1 Portal location for lateral approach. (A) anterolateral portal, (B) middle portal, (C) posterolateral portal

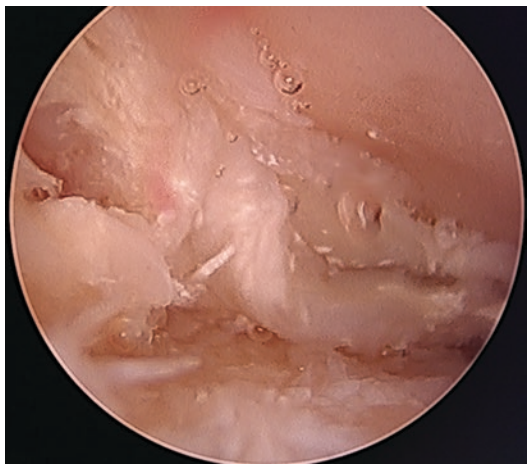

Fig. 25.2 Soft tissue debridement is performed for better visualization

distractor using K-wires placed above and below the joint may improve access to the subtalar joint. Arthroscopic resection of the interosseous talocalcaneal ligament also may provide additional distraction, which opens up the sinus tarsi, allowing further exploration of the posterior and middle parts of the subtalar surface. Resection of the cartilage is started at the middle of the subtalar surface using a curette or shaver (Fig. 25.3). The remaining cartilage of the posterior subtalar surface is then gradually removed. As the talocalcaneal geometry is unique, a variety of instruments, including straight and angled curettes, shavers

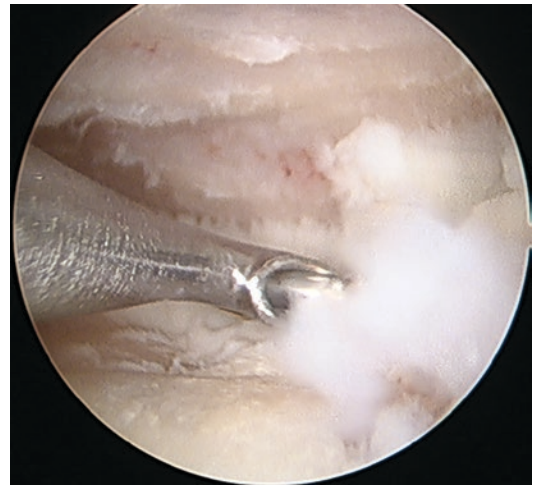

Fig. 25.3 After working space is achieved, a curette or burr is used to remove cartilage

and burrs, are required. Switching the arthroscope and instruments among the portals allows access to the entire joint for debridement.

A cartilage resection of the articular surfaces must be completely carried out down to the subchondral bone. The medial and lateral edges of the posterior subtalar joint are the critical sites to achieve optimum surface contact for the appropriate alignment of the fusion. Once the cartilage resection is completed, approximately 1–2 mm of the subchondral bone should be removed using a round burr to expose the cancellous bone in order to promote the bone fusion (Fig. 25.4). Care should be taken not to alter the contours of the talus and calcaneus, because this will lead to poor coaptation of the joint surfaces. The neurovascular bundle is located in the posteromedial corner, and it should be protected during the procedure. The tourniquet is released, and adequate bone bleeding should be verified.

25.3.3 Screw Placement

After the distractor is removed, the hindfoot is placed in approximately 5 degrees of valgus. This position may be altered according to the anatomy and biomechanical arrangement of the midfoot and forefoot. Varus alignment should be avoided. The

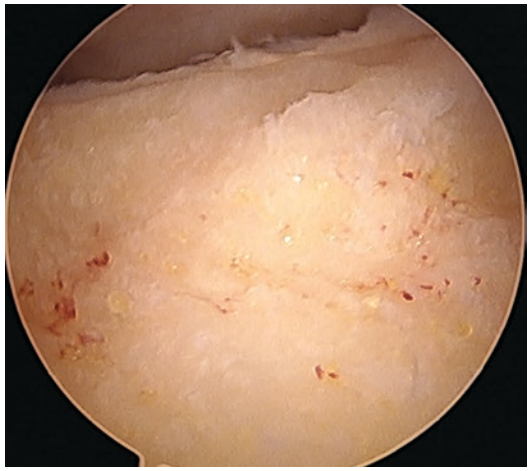

Fig. 25.4 Cancellous bone is exposed prior to screw fixation

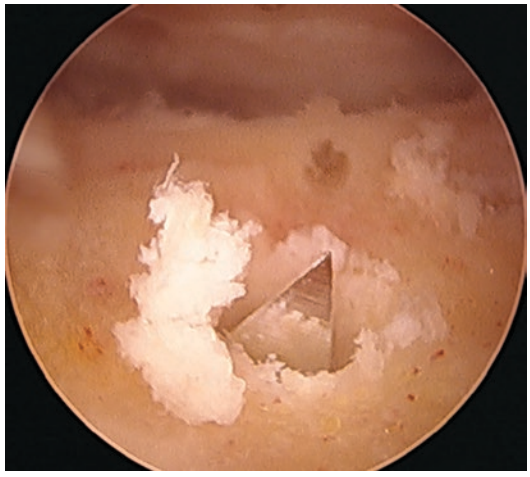

Fig. 25.5 K-wire is placed in the posterior facet of the subtalar joint

fixation is achieved with two cannulated screws in the posterior facet of the subtalar joint. Two diverging K-wires are inserted through the calcaneus, proximal to the weight-bearing surface (Fig. 25.5). One is directed into the talar neck across the subtalar joint, and the second K-wire is placed superior to the first one in the sagittal plane, and is directed into the talar body. The wires are then measured, and the screw holes are drilled. Two 6.5-mm partially threaded cannulated cancellous screws are then placed (Fig. 25.6). Concentrated bone marrow aspirate (CBMA) containing growth factors and stem cells may then be added to augment the biologic milieu for osteogenesis.

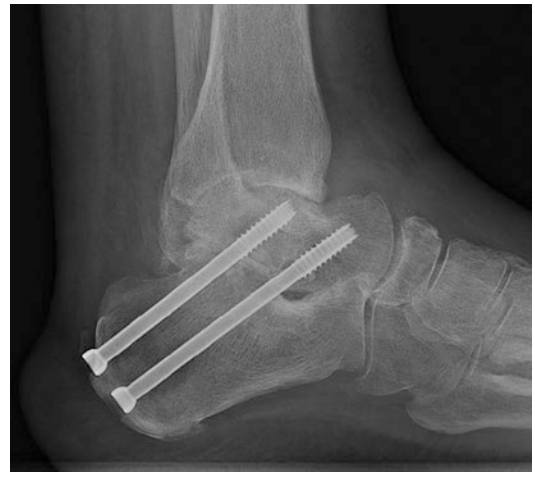

Fig. 25.6 Postoperative radiograph demonstrating subtalar arthrodesis

25.4 Postoperative Management

The ankle joint is placed into a postoperative splint for 2 weeks. At 2 weeks, postoperative the splint is removed and replaced with a Cam walker boot with continued non-weight-bearing until approximately 4 weeks postoperative. After this period, the patient is allowed to initiate partial weight-bearing, progressing to full weight-bearing by 6–8 weeks after surgery.

25.5 Complications

Complications, which are specific this procedure, are injuries of the sural nerve and superficial peroneal nerve. They can occur when making the posterolateral and the anterolateral portals, respectively. Other possible complications are infection, deep vein thrombosis, malalignment, and nonunion.

25.6 Outcomes

Theoretically, the arthroscopic procedure preserves the blood supply of the calcaneus and talus, compared with conventional open procedures. Fusion rates of arthroscopic subtalar arthrodesis reported in the literature range from 89% to 100%. Glanzmann reported that in a prospective case series of 41 arthroscopic subtalar arthrodeses with lateral portals, AOFAS score improved from 53 to 84 at a mean 55-month follow-up with a 100% fusion rate at a mean of 11 weeks [5]. In a comparison study of open and arthroscopic subtalar arthrodesis, Scranton reported a 100% fusion rate in 5 arthroscopic procedures and 1 nonunion in 17 open procedures, but this was small sample size with limited statistical power [6]. However, several studies have shown a trend toward higher fusion rates with the use of arthroscopic procedures [5, 7]. Easley reported a fusion rate of 84% in 148 patients who underwent open subtalar arthrodesis, compared with a 100% fusion rate in the two arthroscopic studies by Tasto and Glanzmann [5, 8]. A systematic review reported by Stegeman demonstrated that AOFAS scores were higher after an arthroscopic procedure compared with open procedures, although this may be confounded by lower preoperative AOFAS scores in the open procedure group. The time to fusion was also reported to be faster in the arthroscopic compared to the open group [9].

25.7 Summary and Conclusions

Arthroscopic subtalar arthrodesis has several advantages over the open procedure, including lower morbidity, preservation of blood supply, and a higher fusion rate. However, it must be emphasized that the application of arthroscopic technique is limited in the patients who have severe varus or valgus deformity. Arthroscopic subtalar arthrodesis provides good clinical outcomes and high fusion rates, and this procedure can be achieved through a 2-portal (anterolateral and middle) approach or a 3-portal technique using an additional posterolateral portal. Further well-designed research will be needed to compare open and arthroscopic procedures to determine whether these apparent advantages are substantiated, but for now, arthroscopic arthrodesis appears to have few disadvantages and several potential advantages over open subtalar arthrodesis.

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Arthroscopic Subtalar Arthrodesis by Posterior Approach

26

Francesco Lijoi

26.1 Indications

The indication for arthroscopic subtalar arthrodesis is an intractable subtalar pain or instability secondary to post-traumatic arthritis, talocalcaneal coalitions, primary osteoarthritis (rare), rheumatoid or other rheumatic arthritis, gross instability, neuropathic pathologies, and chronic posterior tibial tendon ruptures [1]. In our opinion, in these two last indications, several times arthrodesis of other joints of the midfoot (usually in the medial column) are mandatory: in these cases, arthroscopic arthrodesis of the posterior facets of the talo-calcaneal joint is not advisable and an open procedure is to be followed. If ankle arthrodesis must be associated with a subtalar arthrodesis, a double arthroscopic arthrodesis can be done with posterior portals in prone position [2, 3].

26.2 Contraindication

Presence of great deformities, infections, presence of hardware after calcaneal or talar fractures, osteophytes precluding a work chamber, eccentric talus, severe malunion, failure of previous arthrodesis, bone defect requiring large grafting, associated midfoot deformity are contraindications to perform a subtalar arthrodesis arthroscopically [3–5]. Concerning the degrees of malalignment, more than 15° of valgus or 5° of varus are a contraindication to perform an arthroscopic subtalar arthrodesis because of the difficulty to obtain an appropriate correction [4]. In other words, the surgeon is able to perform a correction of about 10° in valgus or in varus with this technique.

26.3 Operative Technique

The patient is in general or peripheral anesthesia, a tourniquet is inflated around the thigh. He or she is in a prone position with the foot to operate beyond the end of the operating table and the other leg on a lower level not to interfere with the surgical maneuvers.

The arthroscope and the instruments used in this kind of procedure are the same as that used in knee arthroscopic procedures; no specific tools are needed.

At the beginning of the intervention, the technique is that described by Van Dijk et al. [6]. Two posterior portals are outlined with a pen on the skin on the medial and lateral borders of the Achilles tendon, slightly above a line drawn tangent to the tip of the lateral malleolus and parallel to the lateral border of the foot with the ankle at

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90°. Two small incisions are made at this level. With a mosquito clamp, the subcutaneous tissues under the lateral incision are blunted and dissected following a direction toward the second metatarsus, until the instrument touches the posterior border of the subtalar joint below the prominence of the posterior talar process. After the clamp has been withdrawn, a 4.5 mm arthroscope with a blunt trocar is inserted in the lateral portal in the same direction (see picture in picture view). On the medial side, the subcutaneous dissection is carried out on an horizontal plane, on the front of Achilles tendon, until the tip of the instrument touches the shaft of the arthroscope. The mosquito clamp is thus being slided on the shaft of the arthroscope toward the posterior talar process.

The clamp is then exchanged for a 5.5 full radius shaver; the fibrous and scar tissues in front of the posterior aspect of the subtalar joint are removed on the lateral side of the posterior talar process.

On the medial side, the FHL must be visualized and debrided from any scar adhesions.

The posterior border of the joint can be arthroscopically focused, and the medial gutter must be freed from the synovial tissue to allow the insertion of a needle from the anterolateral portal at the level of the sinus tarsi [7]. Through this portal a short, large diameter, blunt trocar must be inserted to distract the joint: the direction of the trocar must be nearly parallel to the subtalar joint and the instrument sometimes must be forced into the joint especially if a talocalcaneal coalition is present or in post-traumatic arthritis (like in the clinical case shown in the video).

This distraction allows the penetration of a curette and of the shaver from the PM portal to debride the cartilage.

The arthroscope, the instruments, and the blunt trocar must be interchanged in the three portals to reach every area of the posterior talocalcaneal joint: the instruments usually approach the lateral and the central areas from the PM portal, the medial area from the PL portal. Due to the curved shape of the joint in the sagittal plane, many times, they must be placed in the AL portal to work in the more anterior areas of the joint.

After the entire joint is debrided from the cartilage, the hard, frequently sclerotic subcondral bone must be abraded with a 5.5 round burr to allow a good vascularization of the bony surfaces.

The arthrodesis can be stabilized with two lag screws: they must be inserted in a posteroanterior and distal-proximal direction from the great tuberosity of the calcaneum (just proximal to it) to the body of the talus. The prone position allows an accurate evaluation of the alignment of the hindfoot before the fixation.

Many times, insertion of two guide-wire under fluoroscopic control allows a more accurate placement of two 6.5 cannulated screws with a full compression of the arthrodesis. The wires can be placed in the calcaneus with the aid of a guide for LCA reconstruction (see picture in picture views) until their tips are barely visible at the calcaneal surface of the joint and the correct position checked arthroscopically and with fluoroscopy.

If the position of the wires is adequate, a manual compression over the joint in a correct talocalcaneal alignment is applied and the wires are being forwarded to the subcondral bone of the talus. The length is checked, and the screws are introduced to fix the arthrodesis. The compression and the bleeding can be seen arthroscopically and/or with the fluoroscopy.

The portals and the sites of entry of the screws are closed using nonabsorbable sutures. A walker boot or a cast is applied for 4 weeks with non weight-bearing; then, a partial weight-bearing is allowed for 2 weeks more.

At 6 weeks, the boot (or the cast) is removed and standard radiographs are made. If signs of consolidation of the arthrodesis are present, the patient is allowed full weight-bearing without any support. In case of non-complete consolidation, two further weeks with complete weight-bearing are prescribed with the boot.

26.4 Advantages of Arthroscopic Technique over Open Surgery

Arthroscopic technique offers important advantages related to possible vascular damages to soft tissues: no early complications due to extended skin wounds are present in the arthroscopic surgery. The joint visualization and the subchondral

bone exposure until bleeding are much better in arthroscopic than in open technique during the entire surgical procedure. A possible faster consolidation time in arthroscopic arthrodesis than in open technique has been reported [8].

With posterior arthroscopic approach, it is possible to perform more balanced resections of the lateral or of the medial area of the joint to obtain a better correction of the alignment of the hindfoot.

26.5 Potential Dangers of the Technique

Potential dangers of an arthroscopic subtalar arthrodesis performed with posterior portals may be similar to those described for posterior ankle arthroscopy. They include possible damages to the sural nerve and the peroneal tendons (posterolateral portal) and damages to the posterior tibial neurovascular bundle and calcaneal nerve (posteromedial portal) [9]. An accurate location of the posterior portals, the correct technique in introducing the arthroscope and the instruments, and the use of the tendon of the flexor hallucis longus as a landmark to protect the medial neurovascular bundle permit to perform a safe and reproducible surgery [10].

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Part VII

Ankle Tendinopathies

Flexor Hallucis Longus Tendinopathy

27

Marcello Lughi

Flexor hallucis longus (FHL) tendinopathies are typical of ballet dancers and other athletes that repetitively use their ankles and feet in hyperflexion and hyperextension in a forced manner [1–4].

Flexor hallucis longus is topographically divided into three zones.

- Zone 1: corresponds to the tendon portion, which crosses behind the posterior ankle district.
- Zone 2: corresponds to the tendon portion, which passes under the sustentaculum tali until Henry's knot.
- Zone 3: extends from Henry's knot to the base of the distal phalanx of the big toe. The portion of the tendon more subject to pathology is that behind the posterior ankle district (zone 1) with frequent extension of the condition also to the tendon portion, which passes under the sustentaculum tali until Henry's knot (zone 2) [5–8]. (Fig. 27.1).

Flexor hallucis longus tendinopathy is recognized by medial retromalleolar pain, but often, pain is associated with posterior ankle impingement symptoms.

Medial retromalleolar pain can get worse by contemporary pressure and hallucis passive mobilization.

In the degenerative tendinopathy, especially with nodular or fibrotic tendon hypertrophy, we can find snap hallux or pseudohallux rigid with limitation in dorsiflexion.

Analyzing the anatomy of the posterior ankle portion, the FHL runs closely with capsular, ligament, and bony structures that could be the site of impingement (os trigonum mobilization, posterior talus tubercles, etc.). Posterior ankle impingement symptoms are plantar flexion test and posterolateral pain (Fig. 27.2).

Diagnostic imaging may use surveys with different sensitivity and specificity between them.

Ankle radiographs (in latero-lateral and oblique projections) are positive in the case of bony posterior ankle anomalies.

Ultrasound is positive in the case of peritendinous pathology.

Magnetic resonance imaging is the best procedure for the quantization of peri- and intratendinous lesions.

Computer axial tomography is good for the quantization of bone lesions.

With all of this clinical and instrumental information, we can obtain an adequate diagnostic point of view. Consequently, we can treat the diagnosed disease as well as possible (Fig. 27.3).

If a period of rest, steroid and non-steroid drugs, stretching, and/or changes in the execution

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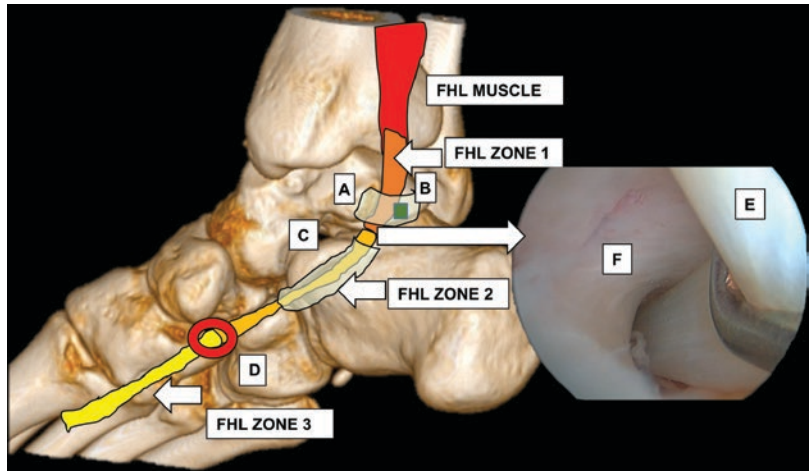

Fig. 27.1 Flexor hallucis longus topographic anatomy. FHL (E) three zones. Zone 1: posterior to the ankle to the osteofibrous talus tunnel (formed groove (F) between posterolateral (B) and posteromedial (A) tubercles and fibrous tissue tense between them (green square) Zone 2: from the end of talus tunnel FHL run under sustentaculum tali (C) to Henry's knot (D). Zone 3: from Henry's knot (D) to the big toe

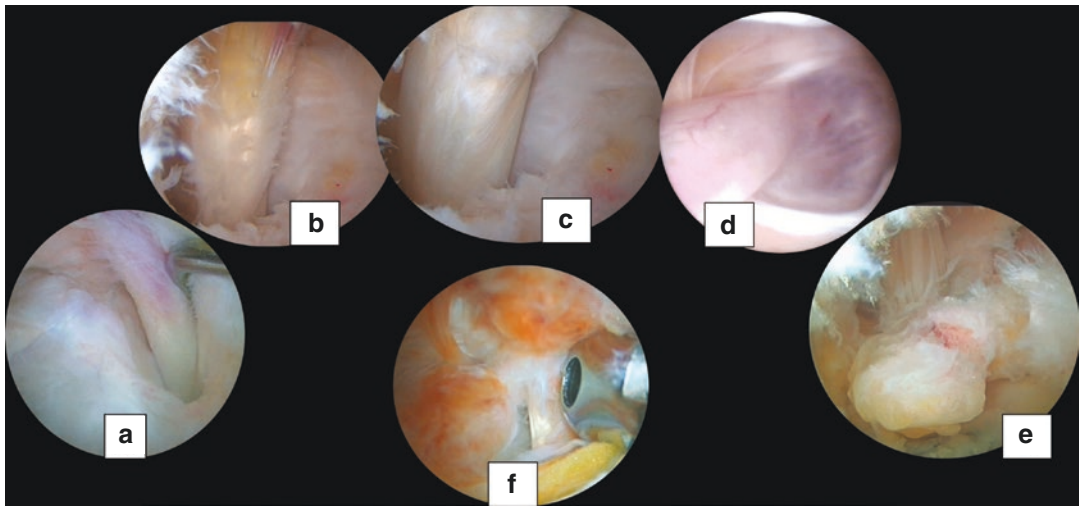

Fig. 27.2 Flexor hallucis longus lesions. (**a**) Normal. (**b**) Tenosynovitis. (**c**) Nodular degeneration. (**d**) Cystic lesion. (**e**) FHL degeneration plus Os trigonum mobilization. (**f**) Villonodular synovitis

of the sporting gesture do not lead to relief of pain, we must opt for a surgical treatment.

Over the years, there has been a gradual improvement in results after application of minimally invasive endoscopic techniques that have shown lower incidence of complications compared to open surgery. The data collected in the literature speaks of an incidence of complications of 3.8–8.5% of endoscopic surgery versus 10–24% of open surgery.

Today we can treat with endoscopic techniques, with good results and with few complications: FHL pathologies in Zone 1.

In the last few years, we can apply tendoscopic techniques also in Zone 2.

However, these are still a few cases and applications with some limits of reliable reproducibility.

The majority of FHL pathologies are treated with posterior endoscopic technique that allows good visualization of the tendon until it changes orientation, but also a good quantization of conflict from diseases that are often associated with tendon pathologies.

It is a proven technique, safe and reproducible [9–14].

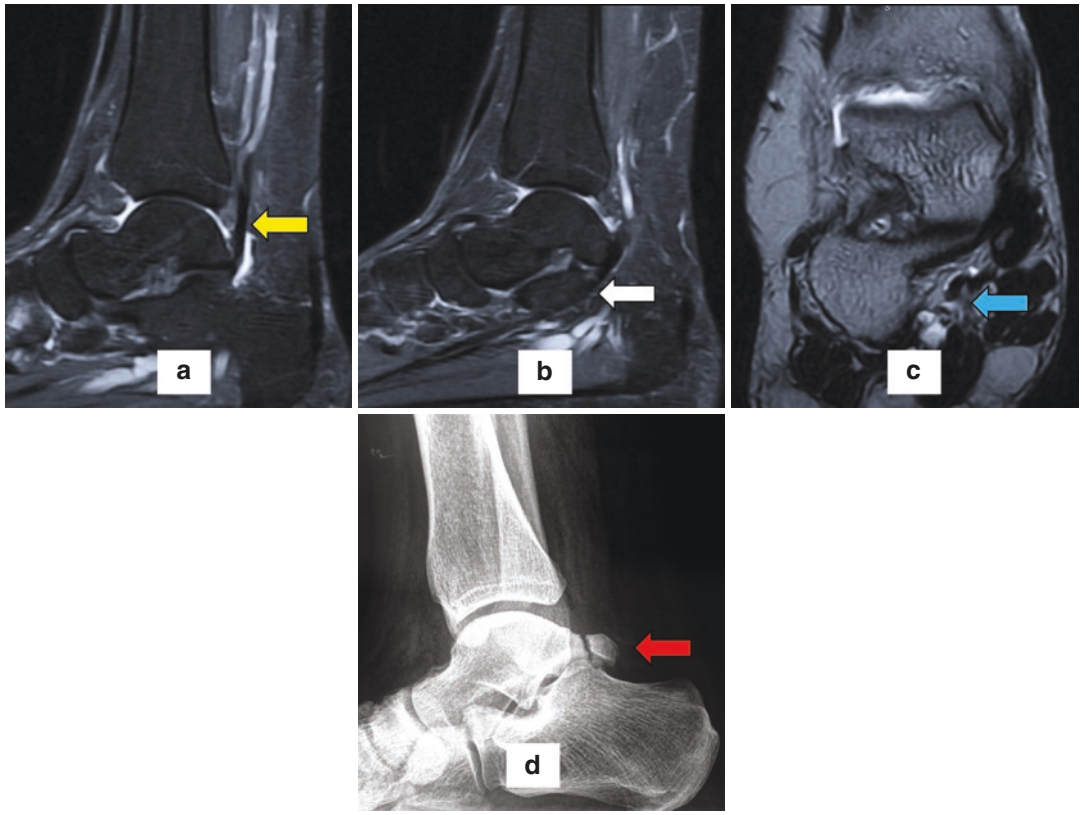

Fig. 27.3 Flexor hallucis longus instrumental diagnosis. (**a**) Yellow arrow: NMR sagittal view, FHL zone 1. (**b**) White arrow: NMR sagittal view, FHL zone 2. (**c**) Blue arrow: NMR coronal view, Henry's knot. (**d**) Red arrow: X-ray sagittal view, os trigonum

27.1 Surgical Technique Step-by-Step

The patient is placed in a prone position, and a tourniquet is applied around the thigh. The operated foot exceeds the edge of the operating bed. In this way, passive ankle and hallux flexo-extension is possible. In the ideal position, the foot's major axis is perpendicular to the floor.

The nonoperated foot is lower than the operated foot. A 4.5 mm, 30° arthroscope is routinely used.

The infusion of saline solution harnesses the force of gravity.

27.1.1 Landmarks and Portals

We have to mark the lateral malleolus tip, the medial and the lateral border of the Achilles tendon. The ankle is brought into a neutral position, and a perpendicular line is drawn from lateral malleolus tip to the lateral border of the Achilles tendon. Posterolateral (PL) portal is situated just above this line, about 1 cm. Posteromedial portal (PM) is at the same level of the PL portal. With a bistoury, we lance only the skin at the PL portal The subcutaneous layer is split with mosquito clamp that is directed anteriorly pointing in the direction of the interdigital web space between the first and second toes.

When the tip of the mosquito touches the bone, it is exchanged with a 4.5 mm arthroscope in the same direction. Then we can lance the skin at the PM portal and, in the same way, we split the subcutaneous layer, but here, the mosquito is directed horizontally toward the arthroscope shaft at a 90° angle. After that subcutaneous layer and deep tissue are split, with the same direction of the mosquito clamp's tip, it is possible to introduce 4.5 mm arthroscope. This manner of instruments introduction through the PM portal represents an original operative dynamic motion by Van Dijk [15]. The first step of endoscopic procedure is the crural fascia resection. Once the fascia is opened, we encounter the subtalar joint and the posterior portion of the ankle and the prominence posterior talar process. With a shaver, we must isolate the FHL tendon. FHL tendon isolation is very important, because it represents an anatomical limit not to be exceeded. Medially and anteriorly to the FHL tendon, there are nervous and vascular structures to be respected. With the FHL visible, we can now treat primary and secondary FHL pathologies (Fig. 27.4).

We can push ahead in the osteofibrous tunnel but, only until a certain point and the tendon release is not always complete. Furthermore, there is the risk of damage (neurapraxia) to the first medial and lateral branches of the posterior tibial nerve. The best treatment of the FHL pathologies in Zone 2 (under sustentaculum tali until Henry's knot) is tendoscopic [16–20].

The proposed technique provides for the use of three portals: the PL and PM portals for visualization of the FHL tendon in Zone 1 and the third plantar portal. Landmarks for PM portal: medial border of the Achilles tendon and a line between the sustentaculum tali, the lower edge of the first cuneiform and first

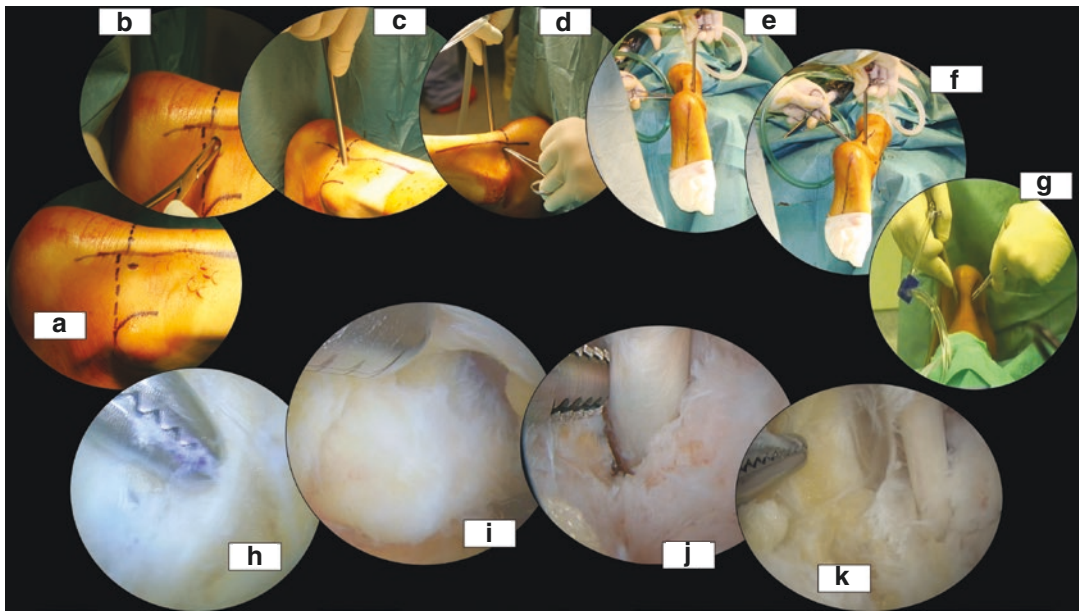

Fig. 27.4 Posterior ankle endoscopic technique and arthroscopic views. (**a**) Posterolateral (PL) portal 1 cm above, draw a line from tip of the lateral malleolus to lateral border of the Achilles tendon (foot at 90°). (**b**) Preparation of the PL portal with mosquito clamp. (**c**) Introduction of the arthroscope through the PL portal vertically, oriented to posterior aspect of the ankle. (**d–g**) Posteromedial (PM) portal, the same level of the PL portal. Introduction of instruments horizontally to arthroscope shaft. It's important to touch arthroscope and then verticalize instruments. (**h**) Arthroscopic view: first step = fascia view. (**i**) Second step = after removal of fascia, we can view posterior talus (PL tubercle). (**j**) Third step = identification of the FHL tendon in Zone 1. (**k**) Particular aspect of the arthroscopic anatomy: on left, near mosquito clamp tip FHL and in front, posterior tibial nerve

metatarsal. The PM portal is at the cross of those markers and then much higher compared to the Van Dijk PM portal [19].

Plantar portal: under endoscopic view, we introduce through the PM portal a wessinger rod into the osteofibrotic tunnel under the sustentaculum tali. We can push the wessinger rod ahead until the plantar fascia at the navicular tubercle. On the wessinger rod impression, we lance the skin for the plantar portal. Visualization of the FHL tendon in Zone 2 is possible if changed optical and instruments in the PM and Plantar portals. If we hold the foot and ankle in plantar flexion, we increase the distance between instruments and/or arthroscope and nervous structures with less complications.

27.2 Traps and Tricks

Traps	Tricks
Iatrogenic damage of the medial nerve and vascular structures	Pay attention to surgical steps and in particular to introduction of instruments through posteromedial portal Pay attention to "operative dynamic" Not classic triangulation Pay attention to operate medially to the FHL
Iatrogenic damage of branches of sural nerve on the lateral aspect	Pay attention to preparing the PL portal. Split subcutaneous layer with mosquito clamp

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Peroneal Tendons Tendinopathy

28

Pim A. D. van Dijk and Cornelis Nicolaas van Dijk

28.1 Introduction

Peroneal tendon pathologies account for a preponderance of (posttraumatic) posterolateral ankle complaints and can cause serious disability in the longer term. In order to prevent further impairment of the tendon tissue and, moreover, to prevent chronic pain, adequate diagnosis and treatment in an early stage is essential [1–4]. Peroneal tendoscopy is an adequate instrument to confirm your clinical diagnosis or to provide insight when in doubt. Moreover, it provides opportunities for treatment of peroneal tendon pathologies. Over the past decade, the procedure has become more and more appreciated [5–8].

Given their important role as dynamic stabilizers of the lateral ankle, the peroneal tendons naturally remain under significant tension. During ankle inversion, high mechanical loads are put on the tendons, especially at the level where they curl around the lateral malleolus [9]. The amount of (over)load increases in patients with chronic ankle instability [10] or overuse [11]. Therefore, recurrent ankle sprains due to chronic lateral ankle instability are considered a typical onset of peroneal tendon pathologies [2, 7, 10, 12].

Tendinopathies linked to the peroneal tendons are generally classified into three categories: (1) tendinopathies (tendinitis, tenosynovitis, tendinosis, and stenosis), (2) (partial) tears and ruptures, and (3) subluxation and dislocation [10, 13, 14]. Other diagnosis for posterolateral ankle pain include posterior ankle impingement, chronic laxity of the lateral ankle ligaments, avulsion or calcification of the posterior talofibular ligament (PFTL), bony spurs, rheumatoid arthritis, and disorders of the posterior compartment of the subtalar joint [15]. While posttraumatic lateral

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ankle pain is a common seen clinical problem, differentiation can be challenging due to nonspecific symptoms and nonspecific MRI findings [16]. Careful patient history and clinical examination is often the key to a correct diagnosis [11]. Peroneal tendoscopy has shown to be an adequate instrument to confirm the clinical diagnosis or to provide insight when in doubt [6, 7, 17].

Treatment of peroneal tendon pathologies is primarily indicated by pain [18]. In general, the first choice of treatment is conservative. Surgical intervention is required when conservative management fails. Surgical treatment of persistent tendinopathy or tendinitis involves debridement and resection of a prominent distal muscle belly. Depending on the severity of the damage, peroneal tendon tears can be treated with debridement and tubularization, tenodesis, or grafting [16]. In case of peroneal tendon dislocation, fibular groove deepening with or without repair of the superior peroneal retinaculum is recommended [19]. Good clinical outcomes have been described after open procedures. However, traditional peroneal tendon surgeries are associated with complications such as postsurgical scar formation, adhesions of the tendon, peroneal nerve dysaesthesia, and impairment of the superior peroneal retinaculum (SPR) [3, 6, 16, 20–22]. Peroneal tendoscopy offers several advantages, as compared to open procedures, with respect to diminished complications rates and a functional aftertreatment [8, 23–27]. This chapter provides an overview of the anatomy and function of the peroneal tendons and patient history, clinical examination, and diagnostics of peroneal tendon disorders and proposes a technique for peroneal tendoscopy.

28.2 Anatomy and Function

For adequate use of peroneal tendoscopy, accurate knowledge of the anatomy and function of the peroneal tendons and its surrounding tissues is substantial. The peroneal muscles form the lateral compartment of the lower leg, better known as the peroneal compartment, and act as the primary evertors and abductors of the foot. Moreover, they play an important role in maintaining integrity of the lateral ankle stability. The peroneus longus (PL) originates at the lateral condyle of the tibia, the lateral aspect of proximal fibular head, the intramuscular septa, and the adjacent fascia. The peroneus brevis (PB) originates more distally on the fibular shaft and interosseous membrane. Approximately 3–4 cm proximal to the distal fibular tip, the PL becomes completely tendinous, whereas the muscle fibers of the PB tend to extend more distal [28]. Sometimes the PB musculotendinous junction occurs distal to the fibular tip, an anatomical variation known as a low-lying muscle belly [29, 30].

Distal to the origin of the PB, both tendons share a common tendon sheath, wherein the PB is located anteromedially to the PL and is flattened against the fibula. The tendons descend posterior to the distal fibular tip, passing through the superior retromalleolar tunnel formed by the retromalleolar groove of the fibula and buttressed by a fibrocartilagenous ridge [31]. The bottom of the tunnel is formed by the deep crural fascia, known as the fibulotalocalcaneal ligament. Both tendons are secured posteriorly to the distal fibula by the SPR, which plays a critical role in maintaining the stability of the tendons within the groove [32]. The SPR originates laterally along the posterior aspect of the distal fibula and extends to its tip where it becomes contiguous with the periosteum. Medially, the retinaculum is merged with the deep transverse fascia of the posterior compartment of the lower leg [33].

After curling around the fibular tip, the tendons are separated by the calcaneal peroneal tubercle. Here, the common tendon sheath is split into two separated sheaths and the tendons enter their individual fibrous tunnels, secured by the inferior peroneal retinaculum. The PB inserts at the base of the fifth metatarsal. The PL tendon turns plantarly at the cuboid groove and inserts at the plantar side of the medial cuneiform and the first metatarsal base (Fig. 28.1).

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 penetrate both tendons over their entire length

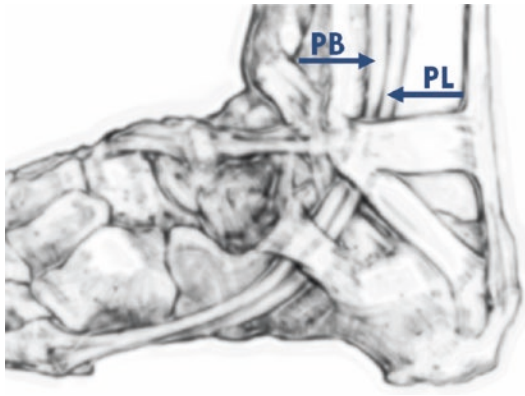

Fig. 28.1 Anatomic overview of the peroneal tendons at the level where both tendons curl around the fibular bone

along the posterolateral side [7, 34]. For many years, 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 [35]. Recent work by the first author of this chapter, however, found no evidence to support the presence of these avascular zones [34].

28.3 Patient History, Clinical Examination and Diagnostics

Due to relatively unspecific symptoms, it is challenging to differentiate between posterolateral ankle pathologies [11], but most of the times, careful patient history and physical examination will lead to a proper diagnosis of peroneal tendon disorders. Acute peroneal tendon pathologies typically present after a recent ankle inversion trauma mechanism. Chronic pathologies mostly occur after a gross ankle inversion trauma in the medical history or in patients with chronic lateral ankle ligament instability. Predisposing factors include cavovarus hindfoot deformity, rheumatoid arthritis, psoriatic arthritis, diabetic neuropathy, calcaneal fractures, fluoroquinolone use, and local steroid injections [36–41].

Typical symptoms include pain and tenderness along the course of the peroneal tendons, mostly posterior or distal to the lateral malleolus. During palpation of the peroneal tendons, a recognizable tenderness, crepitus and swelling can often be found. Passive plantarflexion and inversion or active dorsiflexion and eversion may exacerbate pain, and muscle strength is sometimes weakened when compared to the contralateral side. In tears, pain on provocation of the peroneal tendons in eversion and on acute loosening of resistance during the provocation test is typical. In case of dislocation, the patient may present with lateral instability, giving way and a popping or snapping sensation. Dislocation may be provoked during physical examination by active dorsiflexion and eversion [42], but absence of this phenomenon is not sensitive.

Additional diagnostics are often required to pinpoint the exact diagnosis. In order to rule out osseous pathologies such as fractures, spurs, or calcifications, weight-bearing radiographs are advised in anterioposterior and lateral direction. On the anterioposterior view, a small avulsion fracture of the lateral malleolus or "fleck sign" may be visible in case of peroneal dislocation [43]. MRI is the standard used diagnostic method in evaluating the peroneal tendons, SPR, and the retromalleolar groove [44]. Visible abnormalities include a C-shaped tendon, clefts, irregularity of the tendon contour, and increased signal intensity due to fluid with in the tendon sheath (Fig. 28.2a and b) [45, 46]. It must be noted, however, that abnormalities associated with peroneal tendon pathologies, like fluid within the tendon sheath, are also seen in some asymptomatic patients [47]. Furthermore, the so-called magic angle effect may overestimate peroneal tendon disorders [48]. Ultrasound (US) has the ability of dynamic evaluation of the tendons, making it easier to diagnose dynamic injuries such as dislocation, episodic peroneal subluxation and tears that are not visible on MRI. Quality of the US, however, is strongly dependent on the quality of the observer. Abnormalities visible on US include tendon thickening, peritendinous fluid within the tendon sheath, and ruptures.

Peroneal tendoscopy is gaining popularity in diagnosing peroneal tendon disorders, since it is a highly specific and sensitive method and, moreover, provides easy transition to minimally invasive treatment [17]. The primary indication for peroneal tendoscopy includes posterolateral pain

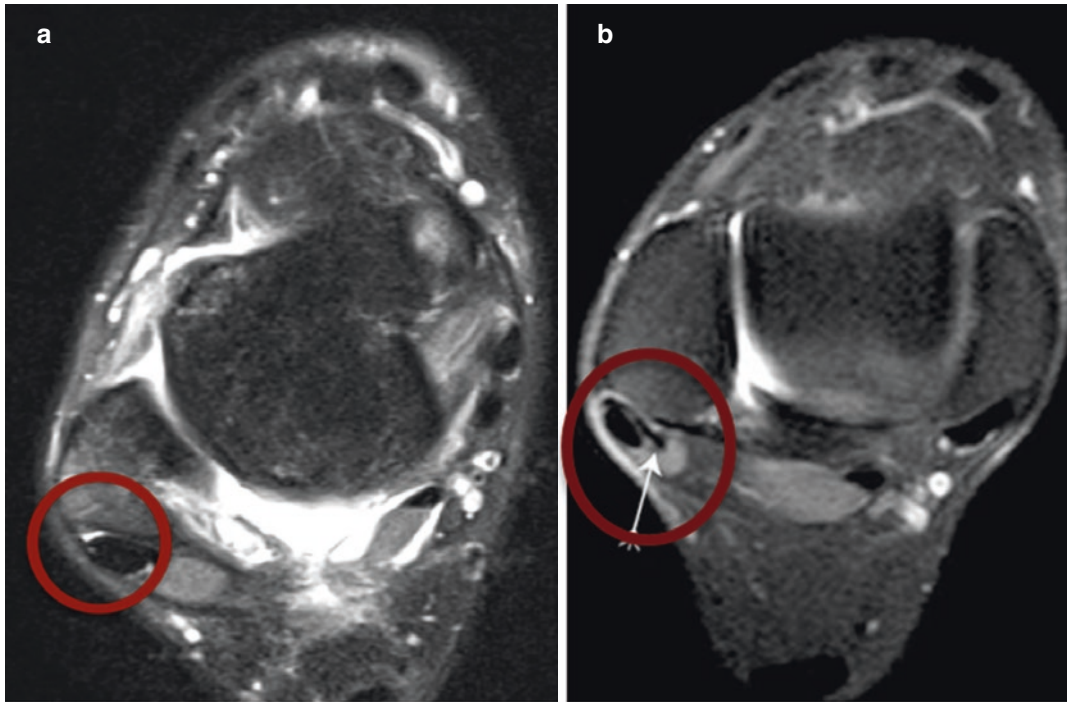

Fig. 28.2 Signs of peroneal tendinopathy on MRI. (**a**) high signal intensity indicating fluid within the tendonsheath. (**b**) c-shaped tendon and irregularity of the tendon

due to tenosynovitis, subluxation or dislocation, partial tears, or postoperative adhesion [7, 17]. Because MRI can be inconclusive for diagnosing peroneal tendon tendinopathies, peroneal tendoscopy be considered when clinical suspicion for a peroneal pathology is strong, with or without positive MRI findings [5, 49].

28.4 Tendoscopic Technique

Ideally, patients are placed in lateral decubitus position for optimal portal access. Moreover, this position allows access to both the anterior and posterior aspects of the ankle when open techniques are required. Alternatively, patients can be placed with the foot in supine position and in endorotation. When the tendoscopy is combined with an arthroscopic procedure, the patient must be placed in a semilateral position to facilitate access to the medial ankle. For free motion in the ankle during surgery, a support may be placed under the leg. To visualize the location of the peroneal tendons, patients are asked to actively evert the foot before anesthesia is administered. The course of the tendons and the location of the lateral malleolus are drawn on the skin, just as the location of both portals (Fig. 28.3). Local, regional, epidural, or general anesthesia can be used for the surgery. To optimize visualization during the procedure, a tourniquet is inflated around the proximal thigh of the affected leg.

For a peroneal tendoscopic procedure, in general two portals are created. The distal portal is made first, 2–3 cm distal to the posterior tip of the fibula. After making a superficial incision through the skin only, the tendon sheath is penetrated with an arthroscopic shaft with a blunt trocar and a 2.7 mm arthroscope with an inclination angle of 30° is introduced. A low-pressure, low-flow pump of 50–70 mmHg is recommended. Some surgeons prefer a 4 mm scope. While this affords an increased flow with a lower pressure [17], it may be challenging to pass this larger scope through the superior tunnel [6]. To maintain hemostasis, normal saline is used for tendoscopic fluid.

Inspection of the tendons starts approximately 6 cm proximal to the posterior edge of the fibular

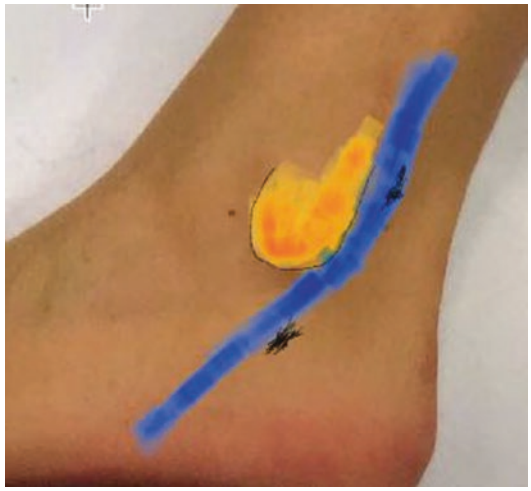

Fig. 28.3 The course of the peroneal tendons (blue), the lateral malleolus (yellow) and the location of the portals (black) are marked on the skin before anesthesia is administered

tip. At this level, a thin membrane splits the tendon sheath into two separate tendon chambers. More distally, the tendons lie in one compartment. A spinal needle is used to guide the second portal, approximately 2–3 cm proximal to the posterior edge of the fibular tip. By rotating the scope within the sheath, an overview of both tendons can be obtained and the course of the tendons can be inspected for damage and pathologies. When significant tenosynovitis is present, complete tenosynovectomy with a shaver is recommended in order to create better visualization of the tendons. This allows the surgeon to evaluate the tendons for associated pathologies, including tenosynovitis, tears, ruptures, dislocation, and stenosis [17].

When a tear is found in one of both tendons, a miniopen approach is required. The tendon is brought into the incision, degenerative tissue is debrided, and the tendon is tubularized using the buried sutures knot and running technique.

In patients with recurrent subluxation or dislocation of the tendons, tendoscopic fibular groove deepening can be performed. The limited workspace around the fibular tip makes this procedure time consuming and challenging. Therefore, it is recommended to perform this procedure by means of a two portal hindfoot endoscopy with the patient in the prone position [50] and create a third portal 4 cm proximal to the posterolateral portal [51]. One or two probes are placed to keep the peroneal tendons out of the groove, decreasing the risk of iatrogenic damage. A concavity in the retromalleolar groove can be created using a 5.5 mm bone cutter shaver. This deepening should cross from the most proximal end of the distal fibula to the tip of the lateral malleolus. The surface of the groove is smoothened and sharp edges are rounded, to prevent the tendons from postsurgical deterioration. Subsequently, the stability of the peroneal tendons in the retromalleolar groove is tested. Only in case of persistent instability after the groove-deepening procedure, a ruptured SPR is repaired. When the SPR was stripped of the fibula, the surface should be roughened with the burr. Two of three suture anchors are then inserted to the fibular ridge and sutured to the SPR.

After finishing the procedure, the portal incisions are closed by sutures to prevent sinus formation. A compressive dressing is applied for 2 days, followed by full weight-bearing as tolerated. Immediately after surgery, functional rehabilitation with active range-of-motion is recommended. Only in case of retinaculum repair, it is favoured to place patients in a lower leg splint for 2 days followed by 12 days of non-weight-bearing in a lower leg cast. Hereafter, patients are either allowed weight-bearing in a Walker boot or in a lower leg cast for an additional 4 weeks, followed by physical therapy to regain strength and range-of-motion.

28.5 Complications

Peroneal tendoscopy is associated with a low rate of complications. The most common reported complication includes a rupture of the tendon sheath during introduction of the surgical instruments, causing reduced visibility of the tendons [5]. Other complications include iatrogenic damage of the tendons due to surgical instrument introduction, suture irritation, and nerve injuries [49, 52]. These complications may be prevented by creating more working space by increase of the fluid pressure [5].

28.6 Pearls

Proper identification of the peroneal tendons is important before creating the portals. Ask the patient to actively evert the foot and draw the course of the tendons on the ankle in order to create a clear reference for your portals (Fig. 28.3).

Before starting the procedure, mark the maximal pain spot during physical examination to have an accurate intraoperative reference point.

To prevent iatrogenic damage, identify the posterior talofibular ligament and the calcaneofibular ligament before initiating the work on the posterior distal fibular surface during a groove-deepening procedure.

Try to keep the vincula intact during the procedure in order to provide optimal vascularization of the tendons during recovery.

28.7 Pitfalls

To prevent the tendons from iatrogenic damage, introduction of the surgical instruments must be performed without any resistance. Increase of fluid pressure during the tendoscopy allows for more working space, making introduction easier.

Don't include retinacular tissue during closure of the portals in order to prevent adhesions.

28.8 Conclusions

When conservative management of peroneal tendon pathologies fails, surgical intervention is recommended. In comparison to traditional open procedures, peroneal tendoscopy offers several advantages: it allows visualization of the tendons from the myotendinous junction up to the peroneal tubercle without causing damage to the surrounding tissues or the vincula. It provides dynamical evaluation of the tendons. And, peroneal tendoscopy is associated with less morbidity, smaller scars, less postoperative pain and complications, a functional rehabilitation, and good functional outcomes, as compared to open peroneal tendon surgery [49].

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Tibialis Posterior Tendinopathy

29

Pim A. D. van Dijk and Cornelis Nicolaas van Dijk

29.1 Introduction

The posterior tibial tendon plays an essential role in the function of the hindfoot by stabilizing the lower leg [1]. Inactivity of the tendon can cause midfoot instability leading to a flatfoot deformity in the longer term. In patients with a flatfoot deformity, on the other hand, high mechanical loads are put on the posterior tibial tendon, predisposing the tendon to deterioration. Other factors associated with posterior tibial tendon pathology include direct or indirect trauma, chronic overuse, and systemic inflammatory diseases. Posterior tibial tendon pathologies must be differentiated from other causes of posteromedial ankle complaints, including pathologies to the flexor hallucis longus tendon, deltoid instability, ossicles, ankle impingement, stress fractures, a Cedell fracture, pathologies to the subtalar joint, and osteochondral defects.

In general, patients can be divided into two groups: (1) younger patients with a systemic inflammatory disease and (2) older patients with chronic overuse of the tendon [2]. Johnson and Strom proposed a classification of posterior tibial tendon pathology, based on the severity of damage and insufficiency of the tendon [3]. Grade 1 is defined by pain and swelling, without foot deformity. In grade 2, patients are not able to perform a single-leg heel raise and present with a flat foot or forefoot abduction. Grade 3 includes a complete rupture of the tendon and is characterized by a fixed subtalar joint together with the criteria of grade 2 pathologies.

Grade 1 and 2 pathologies are initially treated conservative with rest, immobilization, and physical therapy. When flatfoot deformity is present, a supportive foot orthotic may be used to correct the malalignment. Corticosteroid injections are

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contraindicated, due to the risk of tendon ruptures. If conservative management fails, surgical management is often required. In grade 3 pathologies, surgical management is the treatment of recommendation. Over the last years, tendoscopy is preferred over an open procedure, due to advantages such as lower costs, less complications, less morbidity, reduction of postsurgical pain, and the ability of outpatient care [4, 5].

29.2 Indications

Indications for tendoscopy of the posterior tibial tendon include:

- Diagnostic procedure.
- Tendon sheath release.
- Tenosynovectomy.
- Adhesiolysis.
- Resection of the vincula.
- Smoothening of the surface of the posterior tibia.
- Tendon debridement of partial tears.

Moreover, tendoscopy of the posterior tibial tendons can provide diagnostic insight for patients with a suspected partial tendon tear. Partial tendon tears can both be diagnosed and treated during a tendoscopic procedure, while avoiding large incisions, postoperative pain, and a long recovery. Moreover, in case the tear needs to be reconstructed, the tendoscopic procedure is easily converted to a miniopen approach. This is still less invasive than the traditional open procedure.

29.3 Anatomy and Function

The posterior tibial tendon is the largest tendon at the medial side of the ankle and lies closest to the posterior ankle retinaculum. It plays an important role in the stabilization of the lower leg and functions as a plantar flexor and invertor of the foot. The tendon is the primary antagonist of the peroneus brevis tendon, with more than twice as much relative strength.

The posterior tibial muscle arises from the posterior side of the tibia, the posterior side of the fibula, and the interosseous membrane. The tendon is formed in the distal third of the calf and is surrounded by a synovial sheath, starting approximately 6 cm proximal to the tip of the medial malleolus and ending at the level of the navicular tuberosity [6]. The tendon runs at the posterior side of the medial malleolus. Here, the tendon passes through a fibro-osseous tunnel and is covered by the flexor retinaculum. After the tendon curls around the medial malleolus, it becomes flattened and the amount of fibrocartilage increases [7, 8]. Eventually, the tendon fans out into several slips. The anterior part inserts onto the tuberosity of the navicular bone, the inferior surface of the cuneiform bone, and the inferior capsule of the medial naviculocuneiform joint. The plantar part inserts onto the cuneiform bone and the base of the second, third, and fourth metatarsals [9].

The tendon is innervated by the tibial nerve, and blood is supplied by vessels of the posterior tibial artery. These vessels run through a vincula, which is attached to the posterior side of the tendon and runs in all directions [4]. It ends with a free edge approximately 4 cm proximal to the posteromedial tip of the medial malleolus [10].

The presence of hypovascular zones within the tendon remains unclear. Histochemical studies found a hypovascularized zone within the retromalleolar groove, starting immediately distal to the medial malleolus and ending around 5 cm proximal to the distal insertion [11, 12]. Another study, however, did not find such evidence [13]. Therefore, the contribution of hypovascular zone within the tendon to the development of pathologies remains a topic of discussion [14].

29.4 Patient History, Clinical Examination and Diagnostics

Symptoms related to posterior tibial tendinopathy are often vague, and patients may only report posteromedial ankle pain. The early phase of the pathology is characterized by pain along the course of the tendon, and fatigue and aching on the plantar medial aspect of the foot. Weightbearing may increase pain. In case of tenosynovitis, swelling is often found along the course of the tendon. Damage to the vincula may lead to

thickening and scarring of the free edge, which can become symptomatic in the longer term. The painful thickening is typically located posterior and just proximal to the medial malleolus. The strength of the tendon is evaluated by active inversion of the foot against resistance. To evaluate possible abduction of the forefoot, the foot is visually examined from behind. When three of more toes are visible at the lateral side of the calcaneus, significant abduction is present. This is better known as the so-called too-many-toes sign. During physical examination, it is important to compare the symptomatic to the healthy side.

Despite their known limitations, additional diagnostics are useful to confirm the clinical diagnosis. In order to rule out osseous pathologies and reveal hindfoot alignment, weight-bearing radiographs in dorsoplantar and lateral direction are recommended. MRI is the standard used diagnostic method to evaluate the posterior tibial tendon. Signs of pathology include soft tissue abnormalities, bony changes, and edema within the bone. It must be noted, however, that MRI often leads to false-negative or false-positive results. A study by Miller et al. found that 20% of the posterior tibial tendon tears were not accurately diagnosed on MRI [15]. Ultrasound (US) is becoming more appreciated, since it is more cost-effective and accurate [15]. Pathologic changes found during US include hypervascularization and thickening of the peritendinous tissue. In patients with a high clinical suspicion of a posterior tibial tendon pathology, with or without positive MRI and US findings, tendoscopy has proved to be an adequate instrument to confirm the diagnosis or to provide insight when in doubt.

29.5 Tendoscopic Technique

Tendoscopy of the posterior tibial tendon is performed in the outpatient clinic, under local, regional, or general anesthesia. Patients are placed in a supine position. Before anesthesia is administered, the patient is asked to actively invert the foot for palpation of the posterior tibial tendon and to mark the location of the portals. Moreover, the location of maximal tenderness is marked on the skin. A tourniquet is placed on the affected leg and a small support is positioned under the leg to support easy movement of the ankle during the procedure.

In general, access can be obtained along the whole course of the tendon. As preferred by the senior author, the distal portal is made first, 2 cm distal to the posterior edge of the medial malleolus. An incision is made through the skin only and the tendon sheath is opened with a mosquito clamp to subsequently introduce a blunt trocar. A 2.7 mm arthroscope with an inclination angle of 30° is then introduced, and the tendon sheath is filled with saline.

A spinal needle is used to indicate the location of the second portal, approximately 2–4 cm proximal to the posterior edge of the medial malleolus. This proximal portal is used to introduce instruments such as a probe, knife, scissors, and shaver during the procedure.

By rotating the scope over and around the tendon, the course of the tendon can be inspected, from the level of the insertion to approximately 6 cm proximal to the medial malleolus. In order to avoid iatrogenic damage to the neurovascular bundle, it is important to stay within the tendon sheath. The sheath in between the posterior tibial tendon and the flexor digitorum longus is relatively thin, so it is important to regularly check which tendon is being inspected by passive flexion and extension of the toes. In case the tendon sheath of the flexor digitorum longus is entered, the tendon is moving up and down on passive motion of the toes. During inspection of the posterior tibial tendon, the vincula is carefully evaluated and excised in case it is thickened or scarred. Adhesions of the tendon sheath are removed or released. When total synovectomy is required, a third portal is created distal from the distal portal and a 3.5 mm shaver may be used. Tears and ruptures frequently require a miniopen surgical approach.

In order to prevent sinus formation, all portals are sutured. A compressive dressing is applied for 2 days with full weight-bearing as tolerated and functional aftertreatment with active range-of-motion immediately after surgery [16]. In case a retinaculum or tendon tear repair is performed, it is recommended to apply a lower leg splint for 2 days followed by 12 days of non-weight-bearing

in a lower leg cast. Thereafter, patients are allowed weight-bearing for an additional 4 weeks in either a Walker boot or in a lower leg cast, followed by physical therapy to regain strength and range-of-motion.

29.6 Outcomes and Possible Complications

In an early case series published by the senior author, clinical success and high patient satisfaction were reported. Sixteen patients with a posterior tibial tendinopathy were tendoscopically treated, without any complication at a mean follow-up of 1.1 years (range: 4 months to 2 years). In four patients, a symptomatic thickening of the posterior tibial tendon was successfully removed.

A later published series included nineteen procedures in seventeen patients [14]. In nine cases, a tendoscopic procedure was performed for several reasons. No complications were observed at a minimum follow-up of 1 year (range: 1–5 years). In the other eight patients, a tendoscopic synovectomy procedure was performed for chronic tenosynovitis due to rheumatoid arthritis. Three procedures were combined with an arthroscopic synovectomy of the ankle or a hallux valgus correction. In this group, two patients had a recurrence of the tenosynovitis without a rupture of the tendon. A successful second tendoscopic procedure left both patients symptom-free. One other patient underwent a hindfoot arthrodesis 2.5 years after the tendoscopy due to hindfoot arthritis and one patient had some remaining complaints, without development of a flatfoot.

29.7 Pearls

In order to optimize the outcome of a tendoscopic procedure, it is important to have accurate knowledge on the local anatomy and to use routine portals when possible.

Tendoscopy has shown to be an adequate tool for both diagnosis and treatment of posterior tibial tendon pathologies. Moreover, it provides easy transition to a minimal invasive miniopen procedure.

29.8 Pitfalls

Since the sheath in between the flexor digitorum longus and the posterior tibial tendon is relatively thin, make sure that you're inspecting the posterior tibial tendon. To differentiate between the two tendons, passively move the toes up and down. In case the tendon sheath of the flexor digitorum longus is entered, you will see the tendon moving up and down.

29.9 Conclusions

The advantageous character of a tendoscopic procedure as compared to open treatment has made tendoscopy an important tool in the diagnosis and treatment of posterior tibial tendinopathies. It has shown to provide great clarification in diagnosis and assists in diminishing surgical aggregation. Moreover, it provides shorter rehabilitation, allows visualization of the tendons without causing damage to the surrounding tissues, and is associated with less morbidity, smaller scars, and less postoperative pain as compared to open surgery. Not only does it provide a minimal invasive technique with a low complication risk, it also results in high patient satisfaction.

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Haglund Disease

30

Marcello Lughi

When we talk about Haglund disease, we refer to a pathology that causes hindfoot pain and in particular we identify a pathological condition that affects the retrocalcaneal bursa.

Retrocalcaneal bursitis often is associated with the deformity and hypertrophy of the posterior superior tubercle, of the posterolateral portion of the calcaneus, and to insertional tendinopathy of the Achilles tendon configuring Haglund's Syndrome [1–3].

Retrocalcaneal bursitis more frequently is the result of continued impingement between the Achilles tendon ventral portion and the posterosuperior calcaneal tuberosity and is especially is more frequent in repetitive activity and when the ankle and foot are in forced dorsal flexion [4, 5].

The fundamental clinical signs are pain of the hindfoot and particularly near the Achilles tendon often associated with posterolateral swelling.

The Squeeze Test triggers pain from retrocalcaneal bursitis and consists of pinching the preinsertional area of the Achilles tendon with the thumb and index finger.

It is positive when it unleashes pain that derives from an inflammatory condition of the pre-Achilles' space busy by retrocalcaneal bursa.

Digital X-ray, in latero-lateral projection, could give the diagnosis.

In this projection the posterosuperior profile of the calcaneus is very evident, the insertion of the Achilles tendon with any intratendinous calcifications and especially a well-defined area of uniform radiolucency that corresponds to the Kager triangle.

Modifications of this radiolucent aspect are typical of retrocalcaneal bursa inflammation [4–17].

On the digital latero-lateral X-ray, we can do various measurements in order to highlight bone factors predisposing bone to osteotendinous impingement.

None of the measuring methods proposed, however, has a diagnostic accuracy equal to 100%.

There are three measuring methods that are often used. We can evaluate posterior calcanear anatomical configuration with Pavlov's parallel inclined lines, Fowler Philips' angle, and Calcanear pitch angle.

With MRI soft tissue and tendinous structures are well evaluated [15–17] (Figs. 30.1, 30.2, 30.3, and 30.4).

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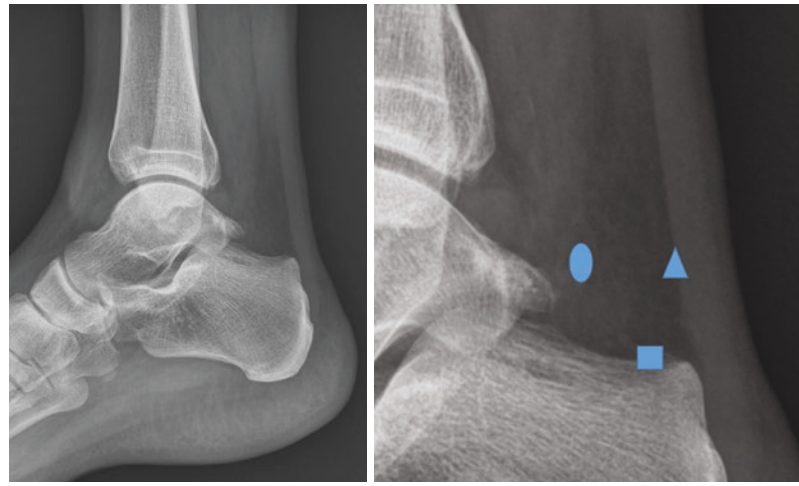

Fig. 30.1 Radiolucent of the Kager's triangle at digital X-ray. Blue oval: ankle-flexor hallucis longus limit. Blue triangle: Achilles tendon. Blue square: posterosuperior tuberosity and superior calcaneus margin with radiolucent of a normal retrocalcaneal bursa

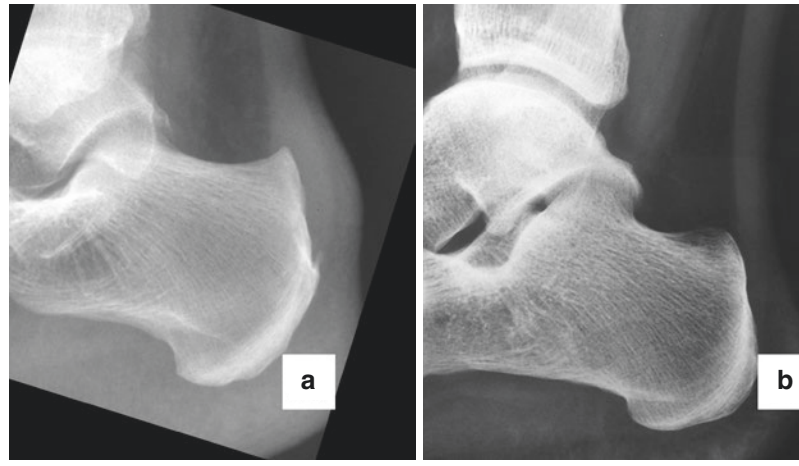

Fig. 30.2 X-ray appearance of the posterosuperior calcanear tuberosity. (**a**) Vertical appearance of the posterosuperior tuberosity. (**b**) Normally convex appearance of the posterosuperior tuberosity

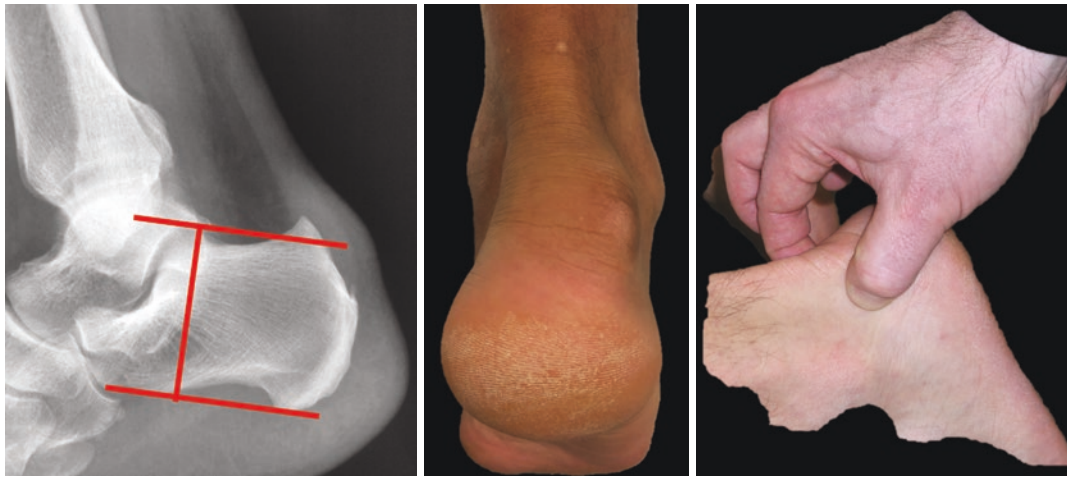

Fig. 30.3 On the left side: X-ray and Pavlov's line. The centre: Evident posterolateral swelling. On the right side: Squeeze test

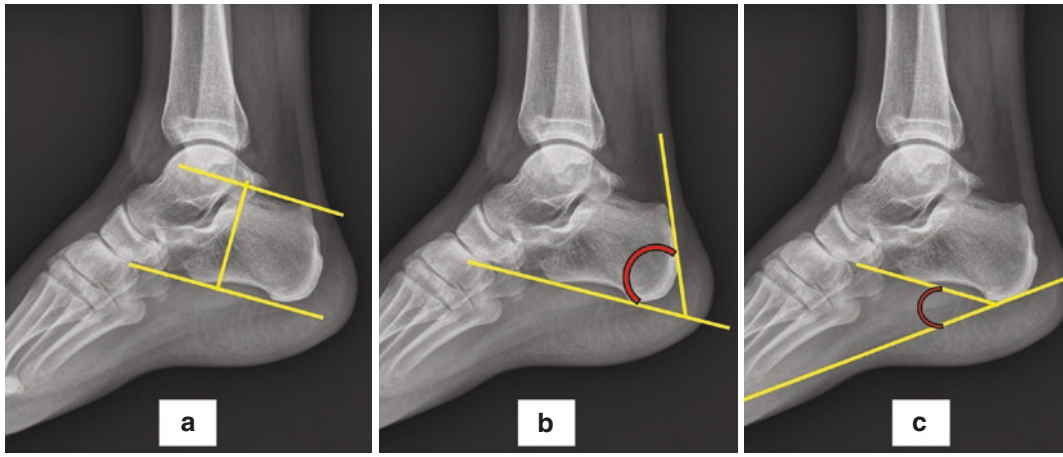

Fig. 30.4 X-Ray measuring methods. (**a**) Pavlov's lines. (**b**) Fowler Philips' angle. (**c**) Calcanear pitch angle

30.1 Treatment

The first therapeutic option is conservative treatment. Only if the pain continues after 6–8 months of conservative treatment, we would propose surgical treatment to the patient.

30.1.1 Conservative Treatment

The basic elements of the conservative treatment are represented by using less restrictive footwear in the rear and a heel rise, reduction of physical activity and rest, anti-inflammatory drug therapy, ice, physical therapy (ultrasound, laser), infiltrative therapy of only the bursal space and not in the Achilles tendon, stretching exercises of the gastrocnemius-soleus complex, and baropodometric study and possible use of orthotics [3].

30.1.2 Surgical Treatment

The ideal surgical treatment is one that resolves pain with the least incidence of complications.

There are two surgical options: open or minimally invasive endoscopic surgery [18–29].

When comparing the two surgical techniques, it is evident that minimally invasive techniques bring good results with a low rate of complications and minor recovery time.

It is important to establish the correct indications and distinguish the limits of the surgical techniques.

Among the OPEN techniques, we find the Zadek or Keck and Kally surgical procedure. It is a rare application of surgical technique that involves cuneiform osteotomy of the posterior calcaneal portion. It has the objective of changing the calcaneus anatomy and decreasing osteotendineous impingement [18].

In consideration of Achilles tendon degeneration with or without intratendinous or enthesic calcification associated with hypertrophic posterosuperior tuberosity and with retrocalcaneal bursa inflammation, an open procedure is indicated, which provides tendon recovery and bone and bursa removal [23].

The simple retrocalcaneal bursitis and the hypertrophic posterosuperior tuberosity removal is possible with an endoscopic technique.

With this minimally invasive technique, a selective treatment is possible saving important anatomical structures (like the pre-Achilles tendon vessels).

Under endoscopic vision, it is possible to operate "adapting yourself" to the Achilles preinsertional district, which has unique anatomical features. It is important to know that the ret-

Table 30.1 Correlation between pathologic anatomy and surgical treatment

Pathologic anatomy conditions	Surgical treatment (preferable)
Retrocalcaneal bursitis	Endoscopic procedure
Retrocalcaneal bursitis Posterior superior tuberosity hypertrophy	Endoscopic procedure
Retrocalcaneal bursitis Posterior superior tuberosity hypertrophy Achilles tendinopathy	Open pricedure

rocalcalneal bursa has a crescent form and the posterior superior tuberosity could be distorted in its posterolateral part.

A para-achilleus approach, beyond the limits of visualization and with greater incidence of complications, does not permit adequate treatment of the bone deformity.

In Table 30.1 correlations between pathologic anatomy conditions and preferable surgical treatment.

30.1.2.1 Endoscopic Calcaneoplasty

Van Dijk has published articles about the posterior endoscopic approach to the ankle and hindfoot where he clarified and standardized a surgical technique that through the years, it has proven to be reproducible, with few complications and obtains excellent results [22, 23].

Surgical Technique Step-by-Step

The patient is in prone position with a pneumoischemic lace at the thigh root.

The operated foot exceeds the end of the operating bed to allow free foot movement in flexion and extension during the surgical procedure. It is useful that the operated foot be raised above the contralateral to be favorites during surgery with the use of the shaver.

Skin Markers and Portals

The skin markers are: the tip of the fibular malleolus, cutaneous projection of the posterosuperior calcaneal tuberosity on the lateral and medial side, Achilles tendon borders, and a line that goes from the tip of the lateral malleolus perpendicular to the lateral margin of the Achilles tendon.

The first portal is posterolateral para-achilleus immediately above the cutaneous projection of the posterosuperior calcaneal tuberosity. Posteromedial portal is made at the same level of the posterolateral portal or under direct endoscopic vision. I prefer the first method if I am not in face with an asymmetric hypertrophy of the posterosuperior tuberosity (e.g., more pronounced posterosuperior/posterolateral hypertrophy). In 2011, Van Sterkenburg highlighted that the portal levels are different, depending on whether you are in front of a normal, cavus, or flat foot. He used as a reference point for portal marker a line parallel to the foot sole tangent to the tip of lateral malleolus (with the foot at 90°).

In a normal foot, the posterolateral portal is about at 2 cm from this line; in a cavus foot, it is more distant, whereas in a flat foot, it is closer [26].

Extra care is taken to avoid injury of the sural nerve, saphenous vein, and posteromedial neurovascular structures.

A 4.5 mm arthroscope is used at 30°. Liquid inflow occurs by taking advantage of gravity force and not with mechanical systems.

The initial configuration, with arthroscope through the PL portal and instruments through the PM portal, can be interchanged in order to obtain the largest and best field of vision and operational capacity possible.

We start to work in this configuration removing the retrocalcaneal bursa and the fibrous and periosteum calcanal layers.

Bone tissue removal must be accurate in particular at the lateral and medial portions of the posterosuperior calcaneal tuberosity. Without close attention to the removal, we could create a "crater" in the middle part of the tuberosity, which could cause a possible recurrence of symptoms (Fig. 30.5).

How Much Bone Must We Remove?

It is important to do good preoperative planning. We can use the thickness of the burr (usually 5 mm) as a reference of how much bone we have just removed, and compare it with the posterior portion of the tuberosity that we have left intact.

Another method involves Kirshner wire application, under fluoroscopic view, at the limit of the bone resection that we have established on the X-ray. In endoscopy, we must remove much bone to highlight Kirshner wire.

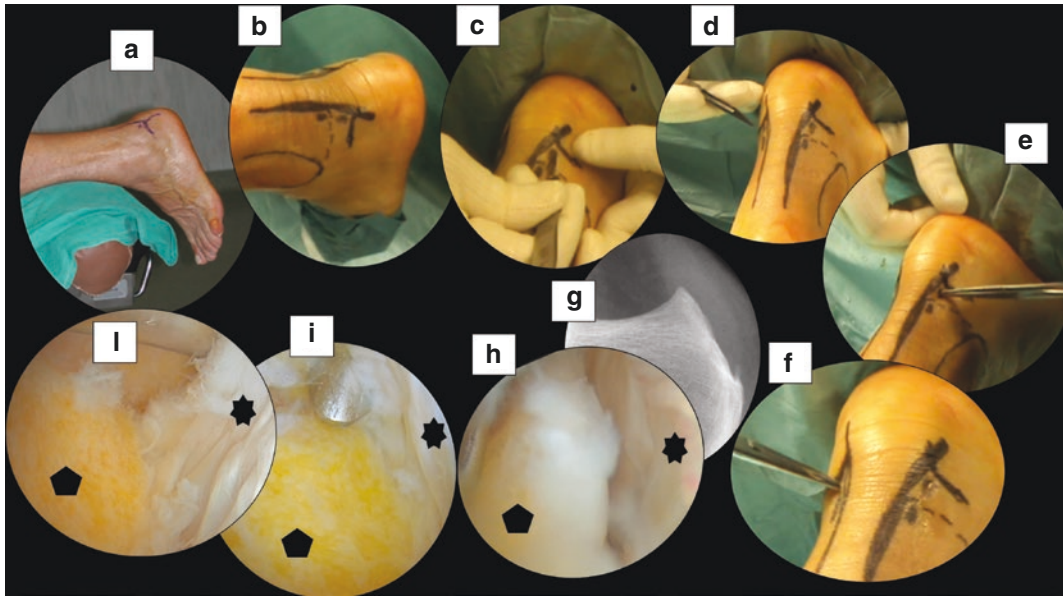

Fig. 30.5 (A) Foot position. (B) Posterolateral marker: note the difference between the level of the PL portal for endoscopic calcaneoplasty and for endoscopic procedure in the hindfoot. (C) Skin incision only. (D) Posterolateral and posteromedial portal at the same level. (E, F) Portal preparation with mosquito clamp. (G) X-ray. (H) Endoscopic vision: black pentangle: posterosuperior tuberosity; black star: Achilles tendon. (I) Posterosuperior tuberosity removal with spherical burr. (L) Final aspect

Table 30.2 Pitfalls and Tricks

Pitfalls and traps	Tricks
Iatrogenic damage of the sural nerve and medial vascular and nervous structures	Pay attention to surgical steps and in particular to portals preparation
Insufficient bone removal in the medial and lateral portions of the deformity	Initial surgical configuration with arthroscope through the PL portal and instruments through the PM portal could be changed for a more suitable and greater field of vision and for the best operative ability possible

We could use also fluoroscopic view for evaluation of the result.

In the postoperative period: Elastic compressive bandage, ice, elevation, weight-bearing to pain tolerance, active mobilization of the ankle and foot in flexion-extension 3–4 times a day.

30.2 Traps and Tricks

See Table 30.2.

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Tendinopathy of the Achilles Tendon

31

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31.1 Introduction

In the past three decades, the incidence of Achilles tendinopathy has risen as a result of greater participation in recreational and competitive sporting activities [1]. However, this condition does not involve only athletes: in one series of 58 patients, nearly one-third did not participate in vigorous physical activity [2].

Tendon pathology has become particularly relevant only in the last few years, principally for the diffusion of competitive sports at a high level and for the different approach to tendon problems, since a better knowledge on the physiopathology and molecular structure of tendons is now available.

31.2 Anatomy of Tendon

The Achilles tendon is formed by the confluence of the gastrocnemius and soleus muscles. The soleus muscle 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 posterosuperior 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 vascularized, and it is responsible for the tendon's blood supply [3], 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 at about 12–15 cm proximal to its insertion, becoming more marked in the distal most 5–6 cm. The tendon spirals approximately 90°, with the medial fibers rotating posteriorly, and

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the posterior fibers 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 [3].

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. 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 fibers along the long axis of tendon [4].

Tendon innervation arises from three main sources:

- Cutaneous nerve trunks.
- Muscular nerve trunks.
- Peritendinous nerve trunks.

Nerve fibers cross and enter the endotenon septa at the musculo-tendinous junction. Nerve fibers penetrate the epitenon from plexuses in the paratenon. Most nerve fibers do not actually enter the main body of the tendon, but they terminate as nerve endings on its surface. Nerve endings of myelinated fibers function as specialized mechanoreceptors to detect changes in pressure or tension. Unmyelinated nerve endings act as nociceptors, sensing and transmitting pain. Both sympathetic and parasympathetic fibers have been identified in tendons [5]. Autonomic peptides such as neuropeptide Y and vasoactive intestinal peptide, which regulate vasoactivity, act in tendons [5, 6].

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 [4]. As collagen fibers deform, they respond linearly to increasing tendon loads [7]. The configuration is initially lost when the stretch exceeds 2% but is regained if the strain placed on the tendon remains at less than 4%. If strain exceeds 8%, macroscopic rupture will occur [8, 9]. The tensile strength of tendons is related to 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 [10].

31.3 Terminology

The terminology of Achilles tendon pathology has become inconsistent and confusing, because of changes in the terminology throughout the years. This has resulted in a large number of confusing definitions and terms. Uniform and clear terminology is necessary for proper research, diagnostic, and treatment. A new terminology has been proposed; the newly described definitions include anatomic location, symptoms, clinical findings, and histopathology.

31.3.1 Historical Perspective

31.3.1.1 Midportion Achilles Tendinopathy

Philip Verheyen, a Dutch surgeon, was the first in 1693 to give to the Achilles tendon its name [11]. Before that, it was known as the "tendo magnus of Hippocrates." In 1883, Raynal described the first case of "cellulite peritendineuse of the Achilles tendon" [12]. In 1950, Lipscomb proposed new definitions for the inflammatory processes of tendons: he defined "paratendinitis" as an inflammatory process of tendons or portions of tendons without a tendon sheath, "tenosynovitis" as an inflammatory process of tendons with a sheath, and "peritendinitis" as a general term to refer to either "tenosynovitis" or "paratendinitis" [13]. In 1976, Perugia et al. introduced a new terminology for the inflammatory pathology of the Achilles tendon [14]. They based their definitions on histological findings, and differentiated among pure peritendinitis, peritendinitis associated with tendinosis, and pure tendinosis. Peritendinitis was characterized by the presence of inflammation of the peritendinous sheaths, without any pathological changes in the tendon itself. Peritendinitis associated with tendinosis involved degenerative features of the tendon associated

with inflammation of the sheaths. Pure tendinosis was characterized exclusively by degenerative phenomena, often associated with foci of osteocartilaginous metaplasia.

The discussion about the nomenclature of Achilles tendon's disorders continued throughout the years [15, 16]. In 1992, Clain and Baxter divided the definitions of Achilles tendinitis into insertional and noninsertional [17].

In 1998, Maffulli et al. [18] suggested to change the confusing terminology concerning overuse of tendon conditions. They proposed to use "tendinosis" [19], "paratendinitis", and "tendinitis" only after microscopic examination of excisional biopsies, since these conditions involve specific histopathological conditions, such as the presence of inflammatory cells [18]. They also proposed to give the name of "tendinopathy" to the clinical syndrome characterized by pain, swelling (diffuse or localized), and impaired performance.

31.3.1.2 Pathology Around the Achilles Tendon Insertion

Albert [20] was the first to describe "Achillodynia" in 1893, but the underlying pathology was not determined [21]. In 1895, Rossler [22] found that the cause of "Achillodynia" was an inflammation of the bursa between the insertion of the Achilles tendon and the posterosuperior tuberosity of the calcaneus. Three years later, Painter found out that the exostoses were most probably manifestations of an osteoarthritic process. Haglund suggested that the term "Achillodynia" was not specific: for this reason, he divided patients into three groups:

- Achillotendinitis ossificans.
- Bursitis Achillea.
- Children with calcaneus'epiphysis growth problems.

He found described two bursae: one located between the calcaneus and the skin (inferoposterior bursa Achillea), and one between the Achilles tendon and the calcaneus (superoanterior bursa Achillea). Haglund stated that superficial bursitis were caused by incorrect shoe-wear for 'Kulturmenschen' (civilized people), and the other (deep bursitis) by either acute or chronic trauma. The inferoposterior bursitis had, in his opinion, no clinical relevance, but the superoanterior bursitis, caused by impingement between the Achilles tendon and a bony prominence on the calcaneous, caused clinically relevant signs and symptoms [23, 24].

In 1954, the term "pump bump" arose. Dickinson et al. described the "pump bump" as an enlargement of the posterolateral aspect of the heel at the site of insertion of the Achilles tendon, and Achilles tendon bursitis, mostly associated with wearing high-heel shoes [25].

Until 1982, the inflammation of the retrocalcaneal bursa was called "retrocalcaneal bursitis," and, in the same year, Pavlov et al. described the "Haglund's syndrome," which was characterized by a painful soft tissue swelling at the level of the Achilles tendon insertion. They described a prominent calcaneal bursal projection, retrocalcaneal bursitis, thickening of the Achilles tendon, and a convexity of the superficial soft tissues at the level of the Achilles tendon insertion using a lateral heel radiograph [26].

Haglund's deformity was then described by Vega et al. as a tender swelling in the region of the Achilles tendon with visible prominence of the posterolateral aspect of the calcaneus.

In 1993, Haglund's disease was added to this list of the tendon pathology [27], although it was earlier described as being an osteochondrosis of the accessory navicular bone [28–30].

Sella et al. thought that all these names were confusing, and proposed separate definitions for them:

- Haglund's disease, which was referred to an osteochondrosis of the accessory navicular bone.
- Haglund's syndrome, which was used when symptoms were present with or without the deformity, and may involve the retrocalcaneal bursa as well as the Achilles tendon and the superficial Achilles bursa.
- Haglund's deformity, which was referred to chronic and sometimes painful distortion of the posterosuperior and lateral portion of the calcaneus [31].

This proposal was, however, not commonly adapted, and in 1998, the Federative Committee on Anatomical Terminology and the International Federation of Associations of Anatomists

published a general anatomic terminology [32]. This "Terminologia Anatomica" defines the retrocalcaneal bursa as "bursa tendinis calcanei" and the superficial calcaneal bursa is called the "bursa subcutanea calcanea" [32].

31.3.2 Proposed Terminology

31.3.2.1 Midportion Achilles Tendinopathy

It is a clinical syndrome characterized by a combination of pain, swelling, and impaired performance. The swelling can be diffuse or localized. Typically, the nodular swelling is located at 2–7 cm from the insertion onto the calcaneus; this part of tendon has also been described as the "main body of the Achilles tendon." This syndrome involves an isolated pathology of the tendon, and includes, but is not limited, to the histopathological diagnosis of tendinosis [18]. Tendinosis implies histopathological diagnosis of tendon degeneration without clinical or histological signs of intratendinous inflammation, and it is not necessarily symptomatic [33]. However, it should be kept in mind that, although the histological term "tendinosis" is widely used, the proper lesion of tendinopathy is not of a degenerative nature: it has the features of a failed healing response, in which the tendon attempts to heal, but, for some reason such as continuous inappropriate mechanical stimuli, the healing process appears to be not finalized.

31.3.2.2 Achilles Paratendinopathy

It is defined as an acute or chronic inflammation and/or degeneration of the thin membrane around the Achilles tendon. Exercise-induced pain and local swelling around the tendon's midportion are the most important symptoms. Acute paratendinopathy is characterized by edema and hyperemia of the paratenon, with infiltration of inflammatory cells, possibly with the production of a fibrinous exudate that fills the space between the tendon sheath and the tendon itself, causing palpable crepitations at the physical examination. In chronic Achilles paratendinopathy, exercise-induced pain is the major symptom, while crepitations and swelling are less pronounced. Histopathologically, the paratenon becomes thickened as a result of fibrinous exudate, prominent and widespread proliferation of (myo) fibroblasts, formation of new connective tissue and adhesions between tendon, paratenon, and the crural fascia [34].

31.3.2.3 Insertional Achilles Tendinopathy

It occurs at the insertion of the Achilles tendon onto the calcaneus, and it can cause the formation of bone spurs and calcifications at tendon's insertion site. Patients report pain, stiffness, and localized swelling. At examination, the tendon insertion (located at the mid-portion of the posterior aspect of the calcaneus) is painful. A swelling may be visible and a bony spur may be palpable. Histopathologically, there is an ossification of enthesial fibrocartilage, and sometimes, small tendon tears occur at the tendon-bone junction [35].

31.3.2.4 Retrocalcaneal Bursitis

It is defined as an inflammation of the bursa in the recess between the anteroinferior side of the Achilles tendon and the posterosuperior aspect of the calcaneus (retrocalcaneal recess), resulting in a visible and painful soft tissue swelling, medially and laterally to the Achilles tendon at the level of the posterosuperior calcaneus.

Histopathologically, the fibro-cartilaginous bursal walls show degeneration and/or calcification, with hypertrophy of the synovial infoldings, and accumulation of fluid in the bursa itself [36].

31.3.2.5 Superficial Calcaneal Bursitis

It is regarded as an inflammation of the bursa located between a calcaneal prominence of the Achilles tendon and the skin, resulting in a visible, painful, solid swelling and discoloration of the skin. It is frequently located at the posterolateral aspect of the calcaneus. It is frequently associated with the use of shoes with a rigid posterior portion. The Achilles tendon is usually not involved. Histopathologically, the subcutaneous bursa is an adventitious bursa, which is acquired after birth, and develops in response to friction. It

is covered by hypertrophic synovial tissue, and fluid. A superficial calcaneal bursitis can be further specified by its location (i.e., posterior, posterolateral, or posteromedial).

31.4 Etiology of Tendinopathy

Tendon injuries can be either acute or chronic, and they are caused by intrinsic or extrinsic factors, either alone or in combination. In acute trauma, extrinsic factors predominate, while in chronic cases, intrinsic factors also play a role. These factors are associated with the onset of overload pathology of tendons, though there is not a specific relationship between cause and effect. Interaction between intrinsic and extrinsic factors is common in chronic tendon disorders.

31.4.1 Intrinsic Factors

Intrinsic factors directly affect the health and composition of the tendon from the local environment in which it forms.

- Age: recent studies show a correlation between full-thickness tears and increasing age; cells frequently shift from aerobic to more anaerobic energy production, and this leads to an increase in matrix metalloproteinase (MMP) production, leading to an imbalance between tendon catabolism and anabolism, with an increase in matrix degeneration and a decrease of strength [37]. In addition, cells gradually lose their ability to undergo apoptosis and turnover, becoming older and less robust, less capable to be replaced by new cells. Furthermore, the vascular supply to the tendons and the nutritional resources from the blood are decreased.
- Body structure and composition: they affect tendons in multiple ways [38]. In 2007, a cross-sectional study [39] showed that a high BMI (in both the overweight and obese range) significantly increased the chances of posterotibial and peroneal Achilles tendinopathy. Similarly, in a 10-year retrospective analysis performed by Klein et al. [40] on 944 subjects, the mean BMI was significantly higher in patients with Achilles tendinopathy compared to the control group.

  Injuries can also result from a sudden increase in repetitive workload, such as when the intensity or frequency of an exercise regimen is increased. If tendons have not enough time to allow cell turnover and injury repair, their structure may undergo further injury with progressively worsening tendinopathy [41].
- Nutrition: tendons depend on nutritional sources for their metabolic cellular processes and turnover. While tendons use approximately 15% as much oxygen as skeletal muscle, they are aerobic structures that require oxygen. Ischemia is tolerated for a relatively long time to allow for long periods of loading and tension. Prolonged hypoxic stress can cause decreased cells function, cells' death, increase of free radicals, oxidative stress, and tissue damage.
- Metabolic diseases: Holmes and Lin [42] in 2006 studied some metabolic risk factors (diabetes, hypertension, use of estrogen, and exposure to steroids) to define and quantify their possible etiological role in Achilles tendinopathy.
- Diabetes is a well-documented cause of tendinopathy. It can alter cellular metabolism, increasing edema and decreasing both the ability of cells to undergo growth and to tolerate ischemic and oxidative stress. Advanced glycosylation end-products are increased: they form crosslinks within collagen fibers, altering their structure, increasing proinflammatory pathways, and modifying the proteins, which are involved in cellular regeneration. In 2009, Maffulli et al. suggest that normal, but in the high range of normal, increasing plasma glucose levels may be a risk factor for rotator cuff tear [43].

  Dyslipidemia may cause disorganization of collagen fibrils and a decrease in their density, resulting in a decreased overall strength.

  Severe hypercholesterolemia, with abnormal fat deposition, can occur macroscopically in tendons with tendon xanthomas.

Inflammatory arthropathies, such as rheumatoid and psoriatic arthritis, can cause direct damage and inflammation of tendons and impair healing.

In 2015 Maffulli et al. demonstrated a correlation between the high plasma non-protein-nitrogen (NPN) levels with rotator cuff tears [44].

– Genetics: it plays an important role in both tendon strength and ability to resist and repair injury. Different structures could be involved, depending on different genetic loci; variations may be seen in normal population variance or as part of distinct clinical pathologies or disorders [45, 46]. The genes currently associated with tendon and ligament injuries include gene encoding for collagen, tenascin, matrix metallopeptidase, and growth factors [47]. For example, blood groups have a correlation with the overall risk of tendon rupture, with blood groups O and A/O more likely to experience a tendon rupture [18, 48].

31.4.2 Extrinsic Factors

Extrinsic factors influence tendon's growth environment from the outside. They can cause injury and impair healing in both acute and chronic situations [18].

– Excessive loading: it can cause microscopic damage to the tendon, either with a direct force or with repetitive excessive loading.
– Fatigue loading: injury can also result from repetitive loading within the physiological range. Repeated microtrauma can cause stress and alter tendon's mechanical properties. Tendon's mechanical loading, within physiological limits, stimulates fibroblast proliferation and collagen synthesis and realignment, promoting appropriate fiber organization. However, if the tendon is subjected to repeated trauma without enough time to repair itself, or without adequate biological mechanisms to undergo regeneration, the cumulative effects may lead to tendinopathy and, eventually, to rupture.
– Improper loading: it results when an abnormal load is given to the tendon because of abnormal positioning. The eccentric positioning of forces acting on a tendon can result in frictional forces between the fibrils, causing microtrauma and/or direct damage. It can lead to abnormal healing and disorganization in fibrils' localization, increasing the predisposition to rupture.
– Disuse: if a tendon is not subjected to physiological levels of stress, they undergo degeneration, and show a decrease in mechanical properties [49]. Microscopic changes include a decrease in cell number and tensile modulus, more disorganized collagen fibers, and changes in cellular morphology and reparative ability [50].
– Compression: tendons typically undergo tensile stresses, but some compressive forces also act in some areas [51]. The tendon-bone junction has a transition zone, which is mineralized, and where tendinopathies frequently occur. Altered loading can increase compressive forces, requiring the upregulation of fibrocartilage formation. This can cause decreased tissue properties and predispose the structure to tendinopathy [52].
– Exogenous damage: it occurs from the outside of the body, either from systemic or local causes [53]. Fluoroquinolones are, for example, a proven cause of tendon damage and rupture; they interact with tenocytes, impairing cellular turnover and healing ability, and they also have a cumulative effect [54, 55]. Tobacco use also impairs the capacity of the tendon to heal and remodel, even when tendons are subjected to a physiological load. A direct mechanical injury (such as laceration) can be caused by multiple conditions, representing a large proportion of tendon injury.

31.5 Histology of Achilles Tendinopathy

Microscopic examination of abnormal tendon tissues shows a noninflammatory degenerative process [56]. Histopathology of Achilles tendinopathy

shows degeneration, disordered arrangement of collagen fibers, and increased vascularization [57], with absence of inflammatory cells and tendency to poor healing [58]. An angioblastic reaction is present, with a random orientation of blood vessels, sometimes at right angles to collagen fibers [59].

Inflammatory lesions and the presence of granulation tissue are uncommon and, if present, they are associated with tendon ruptures [58]. At least 6 different subcategories of collagen degeneration have been described [60], but Achilles tendon degeneration is usually either "mucoid" or "lipoid" [61]. The characteristic hierarchical structure of collagen fibers is also lost [62].

In 163 patients (75% of whom participated in nonprofessional sports, particularly running) with classical symptoms and signs of Achilles tendinopathy for a median of 18 months, changes in collagen fibers' structure, with loss of the normal parallel bundles, were evident [63].

Achilles tendon degeneration is evident with an increased signal on magnetic resonance imaging (MRI) [64], and with hypoechoic regions on ultrasound (US) [65]. These areas of abnormal imaging correspond to areas of altered collagen fibers' structure, and increased interfibrillar ground substance, which have been shown to consist of hydrophilic glycosaminoglycans [60, 66].

In the paratenon, mucoid degeneration, fibrosis, and vascular proliferation, with a slight inflammatory infiltrate, have been reported [67, 68].

31.6 Clinical Aspects

Clinical examination plays a key role in both diagnosis and management of Achilles tendinopathy. The onset of pain, duration, and aggravating factors should be documented. Information on previous treatments is also important. Achilles tendinopathy typically occurs with pain 2–6 cm proximal to the tendon insertion after exercise. As the pathological process progresses, pain may occur during exercise, and, in severe cases, the pain interferes with activities of daily living [69]. There is a correlation between the severity of the disease and the degree of morning stiffness. Runners also experience pain at the beginning and at the end of training sessions, with a period of diminished discomfort in between [70].

Clinical examination starts by exposing both legs from above the knees, and the patient should be examined both standing and prone. Foot and heel should be inspected for any malalignment, deformity, asymmetry in tendon size, and localized thickening. The Achilles tendon should be palpated to detect tenderness, heat, thickening, nodularity, and crepitation [71].

The "painful arc" sign helps to distinguish between tendon and paratenon lesions. In paratendinopathy, the area of maximum thickening and tenderness remains fixed in relation to the malleoli, from full dorsiflexion to plantar flexion, whereas lesions within the tendon move with the ankle motion [72]. Patients with chronic tendinopathy may have greater difficulty in performing the test than patients with acute tendinopathy [71].

Four types of nerve endings can be identified in tendons: Ruffini corpuscles, free nerve endings, Pacini corpuscles (mainly at the tendon site), and Golgi tendon organs (mainly at the muscle site) [73]. The source of pain is still under investigation: classically, pain has been attributed to inflammatory processes, but now it is evident that tendinopathies are not classical inflammatory conditions. Recently, the combination of mechanical and biochemical causes has become more interesting [64, 74]. Tendon degeneration, with mechanical breakdown of collagen fibers, could theoretically explain the pain mechanism. The biochemical model has become appealing, as many chemical irritants and neurotransmitters may produce pain in tendinopathy. High concentrations of the neurotransmitter glutamate have been found in patients with Achilles tendinopathy [75]. Tendons, in these patients, showed no signs of inflammation, as indicated by the normal prostaglandin E2 levels [75]. Substance P and chondroitin sulfate may also be involved in pain generation in tendinopathy [64, 74].

31.7 Imaging

US and MRI are the best diagnostic tools in patients with Achilles tendinopathy.

US: Archambault et al. [76] used a simple US grading scheme for patients with Achilles tendinopathy: grade 1, normal tendon; grade 2, enlarged tendon; grade 3, a tendon containing a hypoechoic area, regardless of size. The visualized hypoechogenic regions can be nodular, diffuse, or multifocal. The ability to visualize the paratenon and intratendinous areas depends on the frequency probes used. High frequencies (7.5, 10, 13, 15 MHz) are more accurate in visualizing abnormalities in the main body of the Achilles tendon and the paratenon [77].

The hypoechoic regions (Fig. 31.1) correlate well with macroscopic pathology seen at surgery [63, 78]. However, ultrasonography cannot differentiate partial tendon ruptures from focal degenerative areas [65]. Hyperechogenic areas can represent focal accumulation of calcium deposits (Fig. 31.2).

The hypoechoic areas showed a very abnormal tendon structure, including an increased amount of proteoglycans.

MRI: the normal Achilles tendon is usually dark on all imaging sequences [79]. In patients with pain in the main body of the Achilles tendon, MRI may show thickened paratenon, peritendinous fluid, edema of the Kager's fat pad, thickening of the tendon (commonly in a fusiform shape), focal or diffuse intratendinous intermediate or high signal, and interrupted appearances of tendon tissue [80, 81]. There is a significant overlap of MRI findings in symptomatic and asymptomatic Achilles tendons. Furthermore, a wide spectrum of diseases is present in symptomatic tendons, ranging from slightly intratendinous and peritendinous tears to complete tendon tears [82].

The sensitivity to detect pathological Achilles tendon tissue can be increased by shortening the echo time [83] and by enhancement with a gadolinium-contrast agent [66]. Under optimal imaging conditions, tendon's structure can be evaluated.

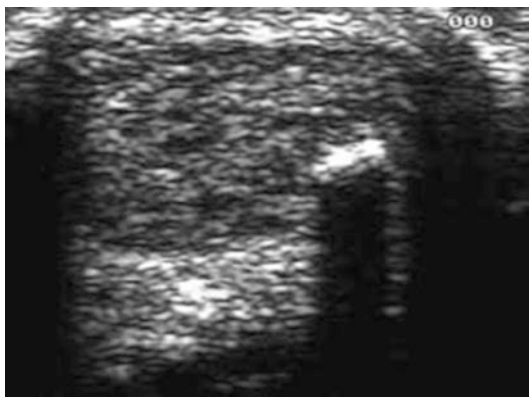

Fig. 31.1 Ultrasonographic appearance of Achilles tendinopathy in a 28-year-old male soccer player at presentation. The longitudinal scan shows that the tendinopathic tendon is thicker than the asymptomatic contralateral one. The normal, well-ordered fibril distribution is lost

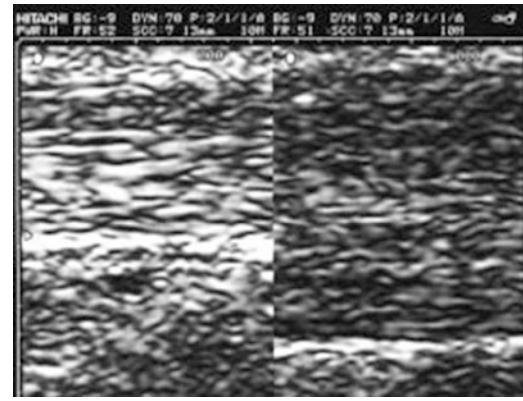

Fig. 31.2 Same patient as in Fig. 31.1. The transverse scan shows an area of intratendinous calcification

31.8 Conclusions

Although Achilles tendinopathy has been widely studied, there is a clear lack of properly conducted scientific research to clarify its causes, pathology, and optimal management plan. Most patients respond to conservative treatment if the condition is recognized early. Teaching patients to control the symptoms may be more beneficial than leaving them to believe that Achilles tendinopathy is fully curable. Progressive eccentric training has been reported with encouraging short-term results. Since the biology of tendinopathy is still under study, more effective management regimens could be provided in the future, improving the success rate of both conservative and operative management.

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Part VIII

Plantar Fascia

Endoscopic Treatment of Plantar Fasciitis

32

Francesco Allegra, Giovanni Corsini, and Carlo Paglialunga

32.1 Introduction

Plantar fasciitis is a common cause of heel pain in adults. It is a soft-tissue disorder first described by William Wood in 1812 [1] and known by many pseudonyms such as jogger's heel, heel spur syndrome, plantar fascial insertitis, calcaneal enthesopathy, subcalcaneal bursitis, subcalcaneal pain, neuritis, and calcaneodynia. This lifestyle-limiting condition can be defined as a localized inflammation of both perifascial structures and plantar fascia at the proximal attachment on the medial tuberosity of the calcaneus. It is thought to be caused by biomechanical overuse from prolonged standing or running, resulting from chronic repetitive microtears and degeneration secondary to overuse or mechanical and congenital disorders [1]. Plantar fasciitis affects up to 10% of the general population and accounts for 10–15% of all foot pain symptomatology; it is experienced at least once in the lifetime of the afflicted population [2]. Although the etiopathogenesis of plantar fasciitis is poorly understood, it is actually considered multifactorial: caused by multiple microtears resulting from an increase in stress and repeated fascia stretching, it exceeds the repair capacity of the body. Diagnosis of plantar fasciitis is based on patient history and risk factors. Among them are referenced excessive congenital disorders such as foot pronation (pes planus), high arch (pes cavus), tightness of the Achilles tendon, and intrinsic foot muscle and leg length discrepancy, while being important in daily activities such as working with prolonged standing, walking occupations (e.g., military or police personnel), excessive running, obesity with a body mass index greater than 34.5–35, and poor motor activity. Some studies have shown that plantar fasciitis is better described as plantar fasciosis because its histological changes at the calcaneal enthesis are consistent with a chronic inflammatory condition, myxoid degeneration, noninflammatory degeneration, fibroblastic hypertrophy, and chaotic vascular hyperplasia, indicating a particular histological picture known as fasciosis [3].

32.2 Anatomic Aspects

Anatomically, the plantar fascia is divided into three bands: medial, central, and lateral. The central band attaches proximally to the medial tuberosity of the calcaneus, separating distally into five individual small bands whose divisions attach to the sesamoids and to the plantar plate of

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the second through the fifth toes. First described by Hicks in 1954 as the *windlass mechanism*, the plantar fascia tenses during the terminal stance to toe-off phases of gait. This tension elevates and reinforces the medial longitudinal arch, which in turn allows the foot to work as a rigid lever for forward propulsion [4].

32.3 Findings and Physical Examination

Plantar fasciitis needs primarily a clinical diagnosis, being the onset of inferior heel pain that is insidious and worsens over time with standing and walking. The sharp pain is usually localized to the plantar-medial aspect of the heel or over a small area near the proximal insertion of the plantar fascia at the medial tuberosity of the calcaneus. Most patients suffer heel pain and tightness after standing up from bed in the morning or after a prolonged time seated, the heel pain improving with ambulation. Occasionally pain can intensify day by day, so that the patient may be obliged to continue walking or standing even for a long time. Findings suggestive of plantar fasciitis include reproducible pain with palpation of the plantar medial aspect of the heel and pain with passive dorsiflexion of the ankle and toes (windlass test). The imaging techniques useful in confirmation of plantar disorder are ultrasound (US), plain radiographs, magnetic resonance imaging (MRI), bone scintigraphy (BS), and elastography. Ultrasound (US) is often to be considered in the first step of diagnosis, remaining lower in cost, noninvasive, safe, portable, radiation free, and easy to administer compared to the other modalities. Radiographic images have been used in the process of diagnosing, relative to those cases where the presence of calcaneal bone spurs need to be confirmed. However, subcalcaneal spurs may be present also, and it is generally fundamental to evaluate the presence of a congenital or acquired deformity of the longitudinal plantar arch, which acknowledgment can represent a direct relationship to a diagnosis of plantar fasciitis. MRI has been confirmed as a reliable and validated tool to effectively diagnose plantar fasciitis, being considered a perfect means to assess and differentiate any abnormalities in its thickness.

32.4 Management and Treatment

Most patients recover without surgery, with only lifestyle modification. Patient education into the expectation and duration of treatment from the outset is fundamental to achieve collaboration at the maximum level. Plantar fasciitis is an overuse phenomenon, and avoiding high-impact activities seems very sensible at its beginning, although patients may consider this change frustrating because this condition has an indolent course. From 85% to 90% of patients affected by plantar fasciitis can be successfully treated with conservative treatment without surgery. Although treatment may be required for 6 months or longer, about 80% of patients treated conservatively have no recurrence of pain for a long time [2]. Initial management should include sufficient foot rest with analgesic and antiflammatory drug use, as nonsteroidal antiinflammatory drugs (NSAIDs) have been shown to represent an adequate means of pain control, especially when used in combination with other kinds of treatment [5, 6]. The intermittent application of ice seems to provide some benefit to patients in association with or during other treatments. Many physical therapies are available that may mitigate and relieve heel pain associated with fascial disorder: these modalities include manual therapy, such as stretching exercises of the calf muscles. Iontophoresis, ultrasound therapy, extracorporeal radial shock wave therapy (ESWT) [7], and laser therapy [8] could be useful in pain control. Also, monophasic pulsed currents support efficiency in reducing inferior heel pain and tenderness and improving functional activities levels associated with plantar fasciitis [9]. These treatments evaluated in comparison have been demonstrated to be effective for maintaining the improvements

in pain and functional ability among the patients treated with two different therapies until follow-up at 12 months [7–10]. Osteopathic manipulative treatment techniques may occasionally provide immediate improvement of plantar fasciitis symptoms; however, the continued maintenance of these results is still being discussed. In some cases, night splinting, customized inserts, shoe modification, and plantar fascia taping could be beneficial [11]. A wide variety of orthotic device options are available for the management of plantar fasciitis: the use of shoe inserts, such as silicone heel pads, felt pads, and a rubber heel cup, in combination with stretching exercises, has been shown to provide superior short-term improvement in relieving symptoms compared to stretching alone [11].

Local injections should be considered: the use of corticosteroid must be evaluated with caution, because of the potentially unfavorable risk–benefit ratio for patients and the briefness of pain relief. Although the local corticosteroid injection (CSI) technique has significantly better treatment outcomes because it is immediately available and easy to administer, both CSI and extracorporeal radial shock wave therapy (ESWT) could be preferred as the primary treatment of patients with acute plantar fasciitis [12]. Early results of platelet-rich plasma (PRP) local injection have been promising: at 12 months, PRP is significantly more effective than steroids, providing better and more durable results than CSI as a treatment option [13]. Platelet-rich plasma is produced via centrifuged autologous blood. The plasma collected is rich with platelets, which release growth factors to stimulate healing in degenerative tissue; currently, some slight evidence supports the use of platelet-rich plasma for nonhealing tendinopathies [14]. On the other hand, a systematic review and meta-analysis found corticosteroid efficacy as compared to autologous whole blood to be marginally superior in relieving pain intensity in patients at 2 and 6 weeks [15]. When conservative therapies have been ineffective and significant symptoms persist, associated with further complications such as chronic pain and arch collapse, surgery is often considered as the last resort, and it can be suited to the patient needs.

Fasciotomy results in significant changes in joint position. A decrease in arch height in a quite wide extension is not suggested for patients with preexisting pes planus because possible consequences may lead to further deterioration of foot position [16]. Overall, these biomechanical effects on the foot are still unclear. Plantar fasciotomy may appear to lead to changes in arch stability in cadaveric specimens and models, thus suggesting modifying the procedure from complete to partial release. However, what effect this may have in the longer term on human subjects in vivo has not been conclusively demonstrated. Again, surgery for plantar fasciitis is not always clinically successful, with 10–50% of patients left unsatisfied following surgery, and with poorer outcomes being obtained in those with more severe symptoms and with a longer duration of preoperative symptoms [17, 18].

A change in operative technique has been noted in the literature, with endoscopic plantar fascia release (EPFR) becoming a viable alternative to the previously established open procedures. Based on the findings of noninflammatory vascular dysfunction, newer treatments for recalcitrant plantar fasciitis have been aimed at focal stimulation and localized angiogenesis. Because it is considered a fasciosis, radiofrequency coblation has been successfully proposed as a minimally invasive treatment modality for chronic plantar fasciitis. Radiofrequency microtenotomy is demonstrated as effective at relieving pain, improving function, achieving patient satisfaction, and meeting patient expectations as plantar fasciotomy [19]. Plantar fasciotomy is perceived as a risky procedure leading to deterioration in foot stability in time. Surgery for plantar fasciitis remains an appropriate treatment option for a patient who does not respond to conservative therapeutic options, achieving reasonable success in the long term, with stable results that do not seem to deteriorate over time even when surgery

is conducted within 1 or 2 years from symptom onset [20]. The disadvantages of surgery include incision care, immobilization, and potential complications such as arch flattening, nerve injury, calcaneal fracture, length of recovery time, and reflex sympathetic dystrophy.

32.5 Endoscopic Procedure

To assess endoscopic treatment, the patient is in supine position with the foot outside the external border of the surgical bed. Spinal or triple-nerve block anesthesia is performed. A tourniquet is recommended to avoid the risk of lack of visualization because of bleeding, which comes from the deep venous vein net, strongly represented on the plantar side of the foot. The instrumentation is the same for carpal tunnel release: a slotted cannula, a blunt trochar, a retrograde hook scalpel, and a regular 30° scope. Skin demarcation of the posterior aspect of medial malleolus and anterior calcaneus, plantar arch, plantar fascia insertion, and small venous vessels should be outlined before surgery (Fig. 32.1). The first portal is placed medially at the upper insertion of the plantar fascia. Through this portal the blunt trochar is passed (Fig. 32.2) until it emerges under the skin

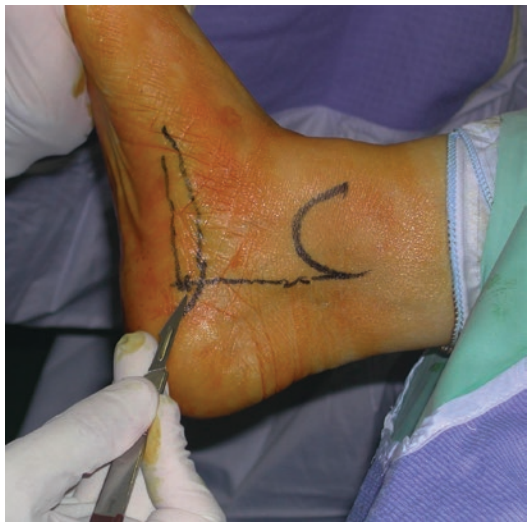

Fig. 32.1 Skin demarcation is essential to put the portal in the right place, avoiding the risk of a misplacement and of injury to closer structures

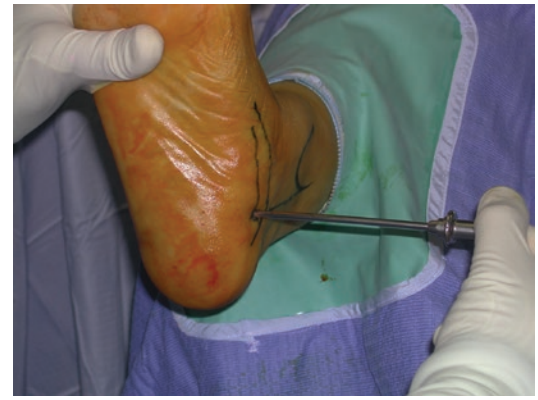

Fig. 32.2 The blunt trochar is inserted into the portal and advanced toward the lateral aspect of the heel

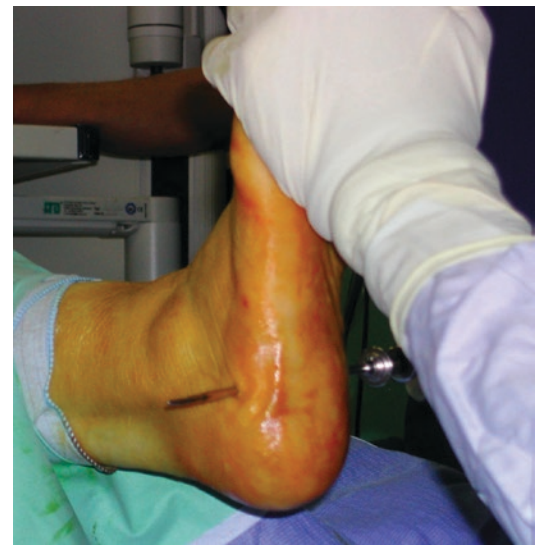

Fig. 32.3 The tip of the blunt trochar is seen emerging under the skin on the lateral heel side, taking care to maintain the trochar perpendicular to the longitudinal axis of the foot

projection of the fascia insertion on the lateral side of the heel (Fig. 32.3). The lateral portal is now opened to permit the trochar to advance. The slotted cannula is advanced alongside the trochar, from lateral to medial until emerging medially (Fig. 32.4). When the trochar is removed and the cannula cleaned, the arthroscope is placed to present a visualization of the entire fascia (Fig. 32.5). The central toward the medial portion of the plantar fascia is explored with a hook

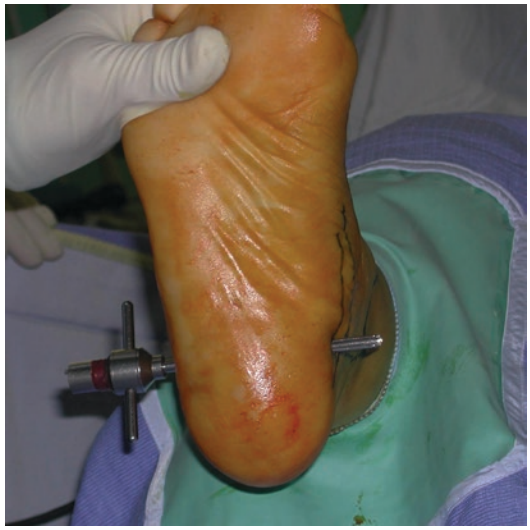

Fig. 32.4 After incision, the lateral portal is opened, positioning the trochar in the correct lateral heel side

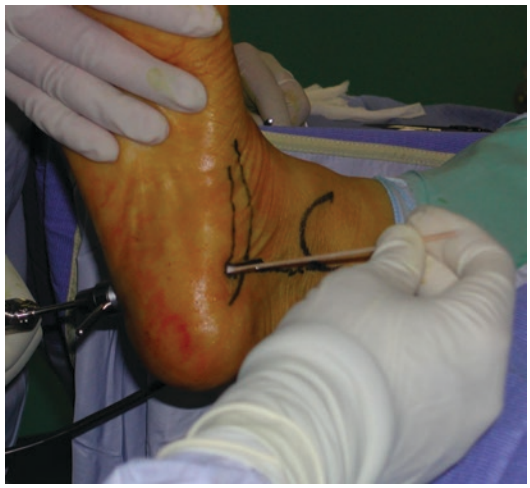

Fig. 32.5 The slotted cannula is advanced along the trochar from lateral to medial and rotated to provide a complete visualization of the plantar fascia

probe (Fig. 32.6) and then resected from lateral to medial, keeping the scope placed laterally (Fig. 32.7). After a final cleanup of the cannula, resection is evaluated with the hook probe detecting the integrity of the flexor hallucis brevis muscle by moving the toe (see Video 32.1). Skin portals are closed with sutures. An ankle bandage

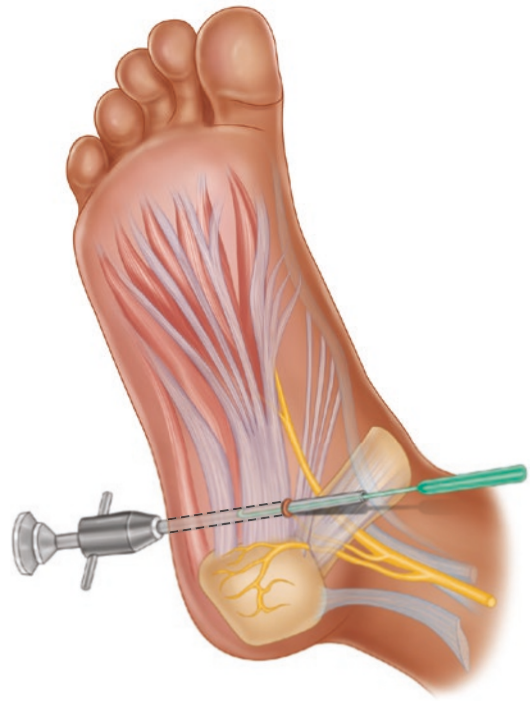

Fig. 32.6 The slotted cannula is placed in its own right position, to permit the instruments to easily proceed transversally on the plantar fascia, which is resected by the hook knife positioned on the medial side of the heel

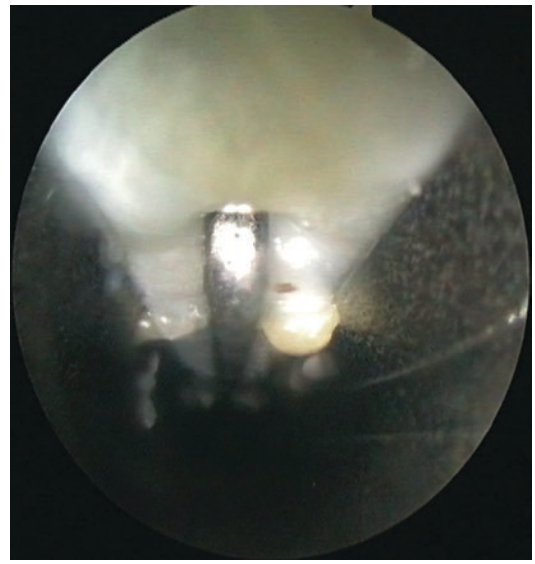

Fig. 32.7 Endoscopic visualization of the hook knife while the section of the plantar fascia is being concluded

is applied for 1 day. Partial weight-bearing is possible with crutches for 2 weeks until complete weight-bearing is permitted.

Traps	Tricks
Do not consider surgery as first therapeutic choice	Primarily make a correct clinical diagnosis
Patient requires therapy with immediate effect on pain	Consider in advance changes in lifestyle and type of shoes
Difficult blind procedure with scope if not performed without blood loss	Use of tourniquet makes a low time-consuming procedure
Frequent postoperative heel discomfort makes patient unsatisfied	Orthotic devices such as silicone heel pads, felt pads, and rubber heel cup should be indicated for a long period after surgery

Key Points

- Plantar fasciitis is primarily a clinical diagnosis and requires a physical examination for differential diagnosis.
- Female patients, middle-aged patients, long-distance runners, military personnel, obese patients, and individuals with a cavus or planus foot deformity are at increased risk for developing plantar fasciitis.
- Lifestyle changes are strongly recommended as first treatment.
- Conservative therapies are recommended for the initial management of plantar fasciitis.
- Among symptomatic patients with low satisfaction and high expectations, 10% can be submitted to plantar surgical release, by fasciotomy or radiofrequency microtenotomy.

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Part IX

Great Toe Disorders

33 Etiology, Classifications, and Treatment by Arthroscopic Procedures

Alberto Marangon, Yoshiharu Shimozono, Michele Montanari, Angelo Bertelli, Paolo Sembenini, and Piergiuseppe Perazzini

33.1 Introduction

Arthroscopy of the first metatarsophalangeal (MTP) joint is still relatively rare, and has been considered to have limited application and to be difficult to accomplish. However, consensus is growing among orthopedic surgeons that a proven surgical technique and the availability of dedicated instruments are making the surgery possible.

Historically the first case dates back to 1985 with Watanabe, Ito, and Fuji [1]. In 1988 Bartlett published the treatment of an osteochondral defect of the head of the first metatarsus of an adolescent boy, entirely by arthroscopy [2]. Ferkel made an important contribution in 1993 when he gave a detailed description of the methodology, specifying the access portals and the intraoperative diagnostic pathway [3].

Initially, arthroscopy of the great toe was used solely as a diagnostic method, but following numerous scientific contributions relating to the technique and its usefulness, it became a treatment method.

Conservative treatment, such as pharmacological therapy and physiotherapy associated with the use of customized orthoses, is the first choice, with surgery being considered only after its failure.

The results obtained in the treatment of major joint pathologies led to the cure of the first MTP joint above all because arthroscopy is less invasive, with minimal bleeding, leaves fewer scars, and is characterized by more rapid functional recovery (especially in athletes), as well as improved esthetics and lower costs as less time is spent in the clinic.

33.2 Indications, Etiology, and Classification

This arthroscopy is no longer 'new-generation surgery,' as it could be called, as it is undergoing continuous development by refining of the technique and the broadening of therapeutic indications.

Up to now, the various pathologies of the great toe have been treated with the classical open methods; arthroscopy is not intended to replace these methods completely, but it is a valid and less aggressive alternative with undoubted advantages.

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The conditions that should prompt the surgeon to consider arthroscopy are persistent pain and swelling that are impervious to physiotherapy and pharmacological treatment, reduced articular function in dorsal and plantarflexion (range of motion), with some episodes of articular blocking.

33.2.1 Major Indications

- Hallux rigidus (Figs. 33.1 and 33.2) (first–second in the Coughlin and Shurnas classification) [4].
- Focal osteochondral lesions (Fig. 33.3).
- Synovitis (Fig. 33.4).
- Soft-tissue impingement.
- Loose bodies (Figs. 33.5 and 33.6).

When treating hallux rigidus, for example, any axial defects of the first ray should be investigated. Dorsal osteophytes, restricting dorsiflexion, must be identified in their position and size, and the degree of the pathology should be established because a serious degenerative arthritic state is one of the counterindications (third–fourth according to the classification of Coughlin and Shurnas) [4].

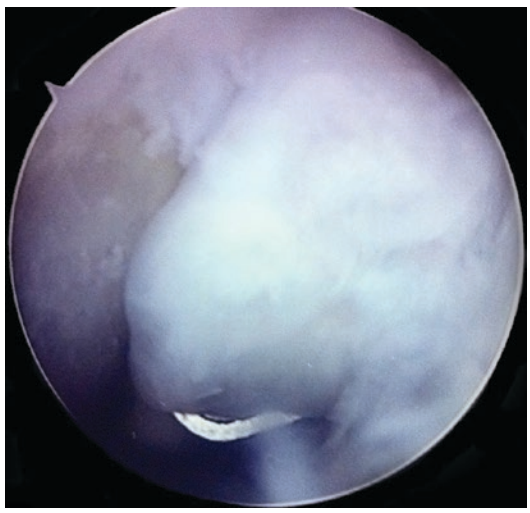

Fig. 33.1 Osteophyte on dorsal edge of head of first metatarsal ray

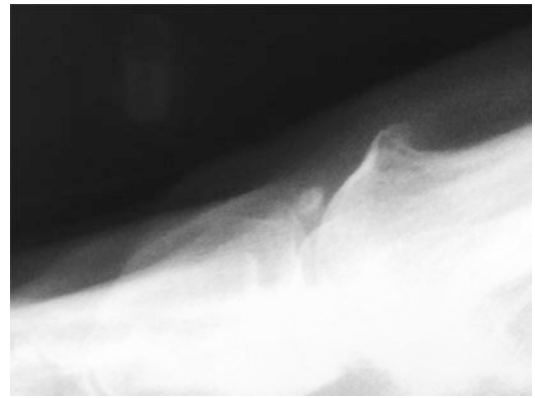

Fig. 33.2 X-ray (lateral view) in a hallux rigidus (first degree) plus intraarticular loose body

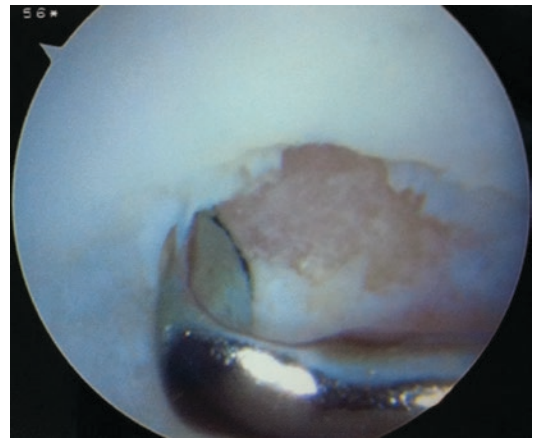

Fig. 33.3 Osteochondral lesion of the first metatarsal head

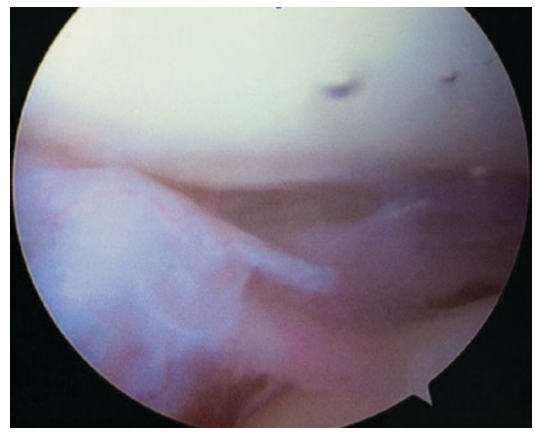

Fig. 33.4 Arthrosynovitis of the first metatarsophalangeal

Fig. 33.5 Loose body

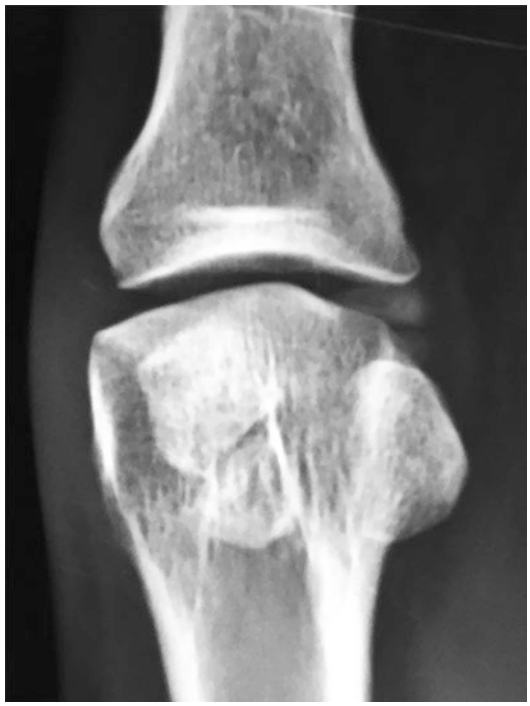

Fig. 33.6 X-ray (frontal view) of first metatarsophalangeal joint with a loose body

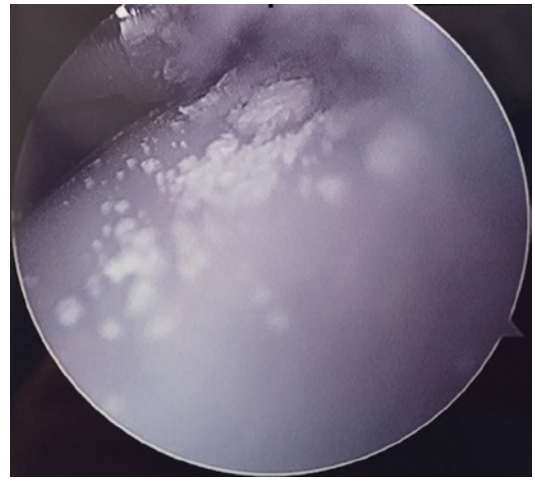

Fig. 33.7 Gout and its intraarticular deposits

This requirement also applies to the chondral and osteochondral lesions that generally affect young sports players. These lesions are derived from osteochondritis dissecans, or may be posttraumatic or microtraumatic osteochondral lesions, sometimes with loose bodies. The size and location of the osteochondral lesions are important and, as stated in the literature, the measurement limit for treatment should be 50 mm^2 or less [5].

33.2.2 Controversial Counterindications

- Endoscopic distal soft-tissue procedure for hallux valgus correction [6].
- Gout and its intraarticular deposits (Fig. 33.7).
- Sesamoiditis and sesamoid fractures.
- Early forms of osteoarthritis.

33.2.3 Absolute Counterindications

- Advanced arthritis stages (end stage; third–fourth in Coughlin and Shurnas classification) [4].
- Severe axial misalignment of the first ray, requiring osteotomies or other corrective procedures.

– Poor vascular status.
– Infection.
– Severe swelling of soft tissues.

33.3 Anatomy

The proper surgical approach requires precise knowledge of the anatomy of the joint. The first MTP joint is essential for walking, bearing twice the load of the other toes; it has been calculated that, at each step, the maximum load on the great toe is between 40% and 60% of the person's weight, and this is much more significant during running and jumping [7].

Anatomically, this joint comprises the distal portion of the first metatarsal bone (head and neck) and the proximal part of the proximal phalange (ball-and-socket joint). In the plantar region, there are two small sesamoid bones, separated by a central ridge known as the crista: the tibial one is larger and the fibular one is smaller. The two bones are embedded in the flexor hallucis brevis of the great toe and are held together by the intersesamoid ligament. Their function is to change the direction of a muscle pull, to modify pressure and diminish friction.

Arising in the dorsal region, on the median line, the extensor hallucis longus tendon of the great toe is inserted at the base of the distal phalange, and laterally is accompanied by the extensor hallucis brevis that finds its insertion at the base of the proximal phalange.

Articular stability is provided by a capsule–ligament complex and the shape of the articular surfaces [8].

The joint capsule has a strengthening system, the medial and lateral collateral ligaments, added to the metatarso-sesamoid ligaments ending at the plantar plate. Capsule support is also provided by the tendons of the abductor and abductor of the great toe.

Blood supply is by the dorsalis pedis artery with its dorsal and plantar ramifications.

Sensitivity of the great toe is provided by the superficial peroneal nerve, which, in the ankle, obliquely crosses the midfoot and forefoot until it reaches the first MTP joint, located about 4 mm medially from the extensor hallucis longus, innervating the medial and plantar portions. On the other hand, the lateral region is sensitized by the interdigital nerve coming from the medial plantar nerve.

33.4 Physical Examination

The clinical evaluation of the great toe includes looking at the integrity of the cutaneous coating, identifying swollen areas, and any skin discoloration and dorsal subcutaneous osteophytes or peri-articular calluses. Any axial defect of the big toe, typical of the hallux valgus, should be investigated, along with any associated deformities of the lesser toes and the rest of the foot. It is important to palpate the painful points, evaluating the range of motion of the great toe and its stability.

The first MTP joint has a range of movement with active extension of 50–60° and 30–40° active flexion. Passive extension, indispensable in the last phase of the step, reaches or exceeds 90°, compared to 45–50° for passive flexion [9]. Decrease of the normal range of motion of the first MTP joint is one of the features of hallux rigidus.

During the objective examination, the patient should be assessed standing to evaluate the morphotype of the foot and any alterations in walking, including the ability to walk on the toes or heels. Shoes should be checked to assess any deformities of the sole.

Any hypermobility of the first ray should be considered along with any instability on the transverse or sagittal axes.

33.5 Imaging

In the diagnostic exams, the first step is to take an X-ray standing, with anteroposterior radiographs with additional lateral, oblique, and tangential views for the sesamoid bones. This imaging enables detection of osteophytes and assessment of the degree to which the joint line has been reduced, with associated sclerotic areas.

Magnetic resonance imaging (MRI) is very useful; it can be carried out if osteochondral lesions or synovial reactions are suspected. It is useful to assess the type of chondral lesion and to identify loose bodies, sesamoiditis, ligament injuries, plantar plate tear, or osteophytes. Computed tomography (CT) is typically used to identify the location and size of osteophytes and to assess any subchondral cystic areas.

It is important to choose the appropriate imaging examination to make the correct surgical choice, that is, arthroscopic versus open.

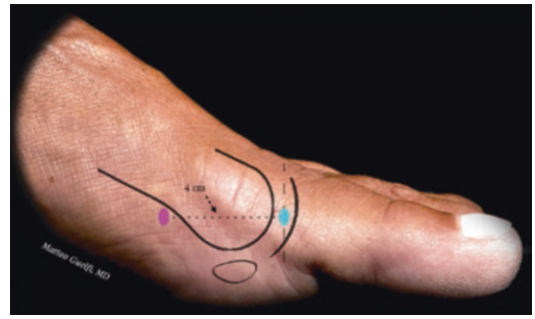

Fig. 33.8 Arthroscopic portals (lateral view) (courtesy of Matteo Guelfi MD)

33.6 Portals

In the literature, six arthroscopic portals to the first MTP joint are described:

1. The dorsal medial, the dorsal lateral, and the medial portals.
2. The medial plantar and the proximal medial portals.
3. The lateral toe web portal.

The portals most commonly used are the dorsomedial and the dorsolateral; others include the medial portal and lateral toe web [10].

When preparing surgical access portals, it is useful to draw the anatomic landmarks (dorsal joint line, the course of the extensor hallucis longus, and the medial dorsal nerve branch) to reduce the risk of causing lesions to the dorsal nerve branch coming from the superficial peroneal nerve (Figs. 33.8 and 33.9).

The first portal to be established is the dorsolateral, located on the joint line 2 mm lateral from the extensor hallucis longus. The second portal is dorsomedial, created under arthroscopic control, and is located on the joint line about 2.0 mm distant medially from the extensor hallucis longus and 4.0 mm from the nerve branch.

The remaining two portals, the plantar medial portal and lateral toe web, are used to view the plantar region of the joint and the two sesamoids [11]. The first is located 4.0 mm proximal to the joint line, between the abductor and medial head of the flexor hallucis brevis,

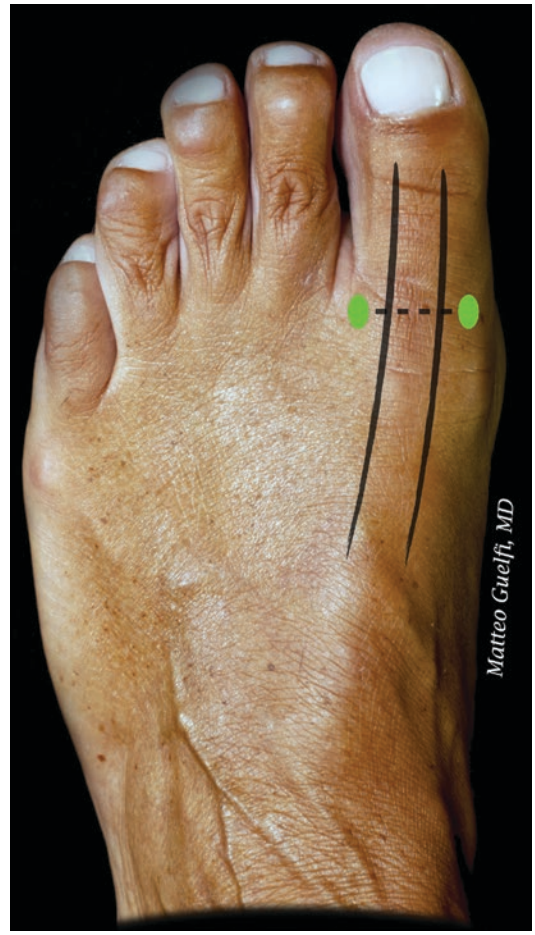

Fig. 33.9 Arthroscopic portals (frontal view) (courtesy of Matteo Guelfi MD)

and the toe web portal is on the dorsal surface of the first web, approximately 4.0 mm proximal and medial [12].

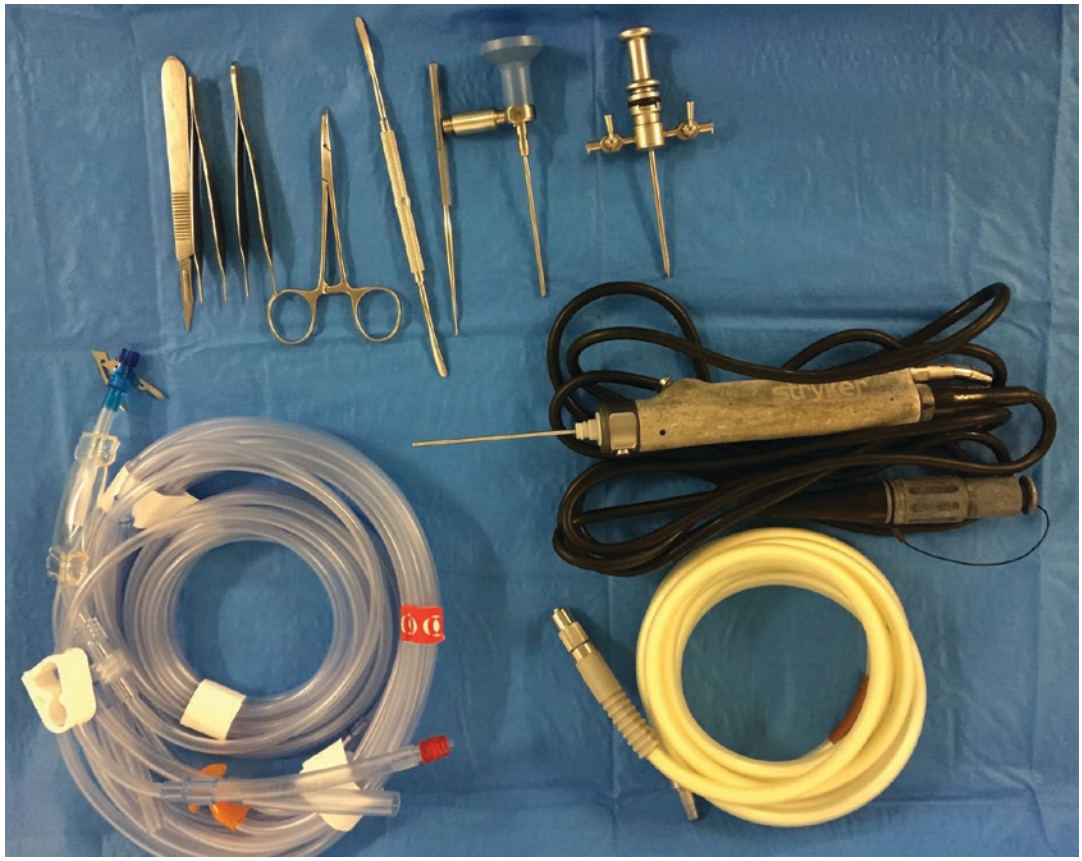

Fig. 33.10 Dedicated arthroscopic instruments

33.7 Equipment

Dedicated instruments include an arthroscope (diameter 1.9, 2.5, or 2.7 mm) with an inclination of 30° or 70° and a set of instruments for small joints with diameter varying from 1.6 to 2.3 mm (freer, basket, grasper, probe) (Fig. 33.10). The authors use an irrigation system (pressure, 45 mmHg).

To remove soft tissues, typical in arthrosynovitis, a 2.5-mm shaver is used. In the treatment of hallux rigidus and for osteochondral lesions, a 2.5-mm bone resector is used to debride the osteophytes and the damaged cartilage; a 2.0-mm curette and a microfracture set or 1.0 mm Kirschner wire is used to stimulate the subchondral bone for bone marrow stimulation (BMS).

For the treatment of osteophytes in hallux rigidus, it is advisable to have a brightness amplifier on hand to check the proper removal of bone spurs.

33.8 Preoperative Preparation and Operative Technique

The patient is placed in supine position on the operating table with the heel on the edge of the table and the foot straight. Anesthesia may be local/regional (epidural anesthesia) or general. The authors prefer to use a tourniquet on the thigh. It is advisable to apply it above the ankle and calf to prevent blockage of the extensor hallucis longus (EHL) or flexor hallucis longus (FHL) tendon.

Arthroscopic treatment of small joints is often difficult because of the reduced working space and the skeletal joint architecture, so the surgeon may use an invasive or noninvasive distractor. The first option is to use an external fixator; the noninvasive systems include manual traction, which usually provides a good view, or

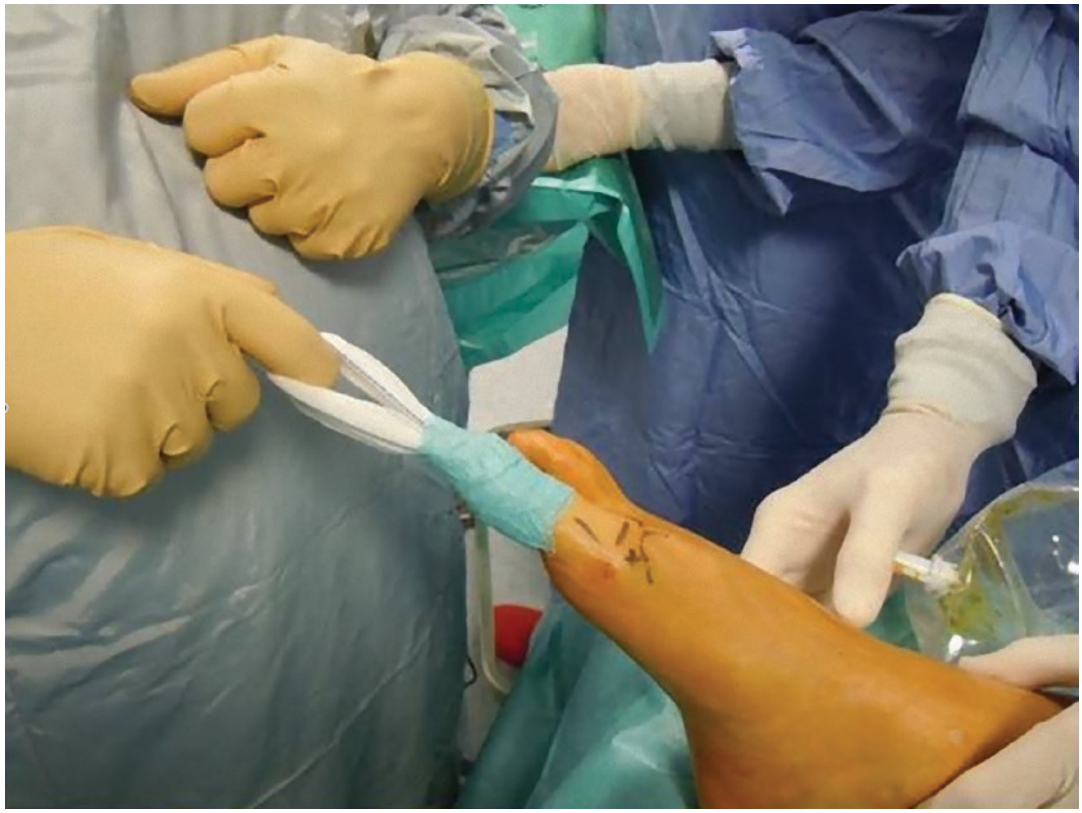

Fig. 33.11 Manual distraction systems with a finger trap

the use of a sterile finger trap applied to the great toe and hooked up to a shoulder holder system (Fig. 33.11).

The position of the surgeon relative to the patient may vary according to preference: he can sit at the foot of the table or by the side of the patient, working with the foot in a frontal position. The viewing monitor should be placed accordingly. After drawing the anatomic landmarks (joint line, extensor hallucis longus, cutaneous nerve branch), mark the arthroscopic portals (Fig. 33.8 and 33.9).

The dorsolateral portal is carried out first. The first step is to introduce into the joint 5.0 ml saline solution using a syringe with #22 needle, to create the working space (Fig. 33.12). Using an 11-blade scalpel, vertically cut only the skin (3 mm), make a subcutaneous blunt dissection, and open the capsule with a hemostat or mosquito clamp, taking care not to damage the cartilage (Figs. 33.13 and 33.14).

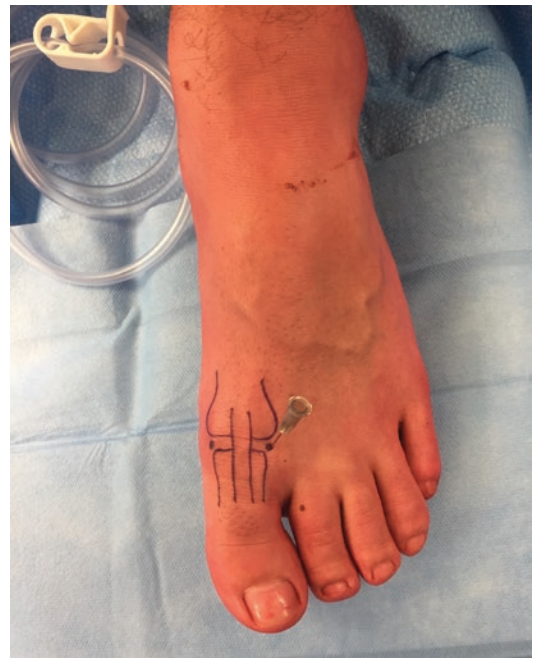

Fig. 33.12 Creation of dorsolateral portal using a #22 needle without finger trap

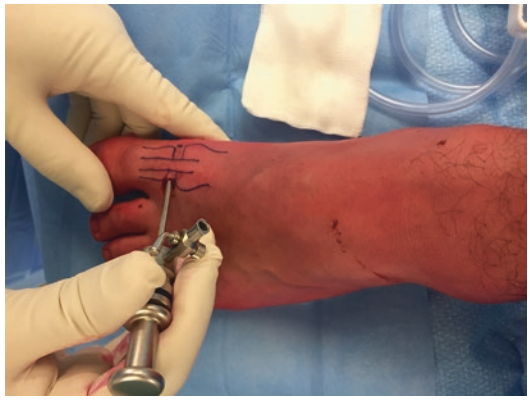

Fig. 33.13 Introduction of shaft of the arthroscope into the joint

Fig. 33.14 Introduction of the arthroscope into the joint

The dorsal medial portal is created using the same 22 G needle, under arthroscopic control and via trans-illumination, a useful system that enables the surgeon to avoid lesions to the vascular structures and articular cartilage [12].

Before diagnostic examination, a shaver may be used to remove the intraarticular synovial tissue because it often blinds the vision. The shaver should be switched in the two portals, controlling the tip to not damage the surrounding tissues. The joint should be assessed in line with Ferkel guidelines by identifying the 13 points via the two classic dorsal portals [13]:

1. Lateral gutter.
2. Lateral corner of the metatarsal head.
3. Central portion of the metatarsal.
4. Medial corner of the metatarsal head.
5. Medial gutter.
6. Medial capsular reflection.
7. Central bare area.
8. Lateral capsular reflection.
9. Medial portion of the proximal phalanx.
10. Central portion of the proximal phalanx.
11. Lateral portion of the proximal phalanx.
12. Medial sesamoid.
13. Lateral sesamoid.

33.8.1 Osteochondral Lesions

During the diagnostic examination, feel the condition of the cartilage coating, looking for chondromalacic areas and their staging. As described by Kim, osteochondral lesions of less than 50 mm^2 can be treated with BMS after careful debridement of the diseased cartilage tissue and with stimulation of the subchondral bone with small-diameter micropicks for microfracture, penetrating into the subchondral bone no more than 3 mm [5]. After carrying out the microfractures, the tourniquet is released and the water inflow closed off, to ensure the bone tissue bleeds properly, allowing clotting rich in mesenchymal cells.

33.8.2 Hallux Rigidus

In treating hallux rigidus, the authors recommend a variant of the position of the patient on the operating table. The knee, supported by a leg holder, is bent, providing a stable support for the foot on the operating table, and traction is manual (Fig. 33.15).

Fig. 33.15 Position preferred by the authors in the treatment of hallux rigidus

After creating two classic dorsal portals, carry out the needed synoviectomy to locate and estimate the size of the osteophytes. Importantly, use a Freer to clear the portion of the articular capsule adhering to the osteophytes. After creating a working space, remove excess bone on both sides. The portals should be inverted to provide an unobstructed view. After cheilectomy, assessed with a brightness amplifier, check the recovery of the range of motion, above all the dorsal flexion, excluding elements of impingement (Fig. 33.16).

33.8.3 Synovitis, Loose Bodies, and Sesamoiditis

Synovitis is one of the most common causes of joint pain, so is necessary to remove the synovial soft tissue carefully, using a shaver, and always paying attention to the presence of loose bodies in the joint: these must be removed.

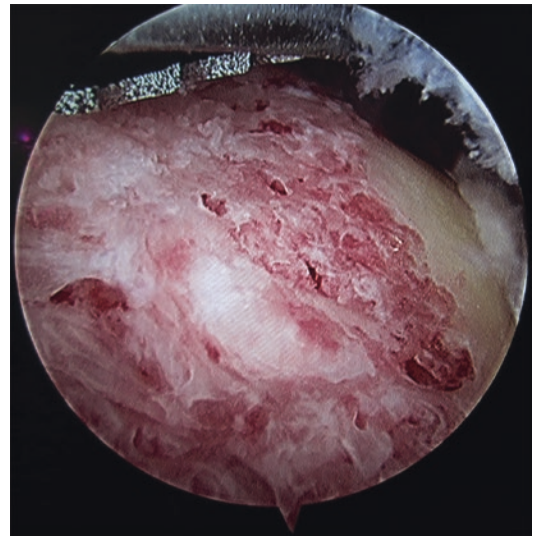

Fig. 33.16 First metatarsal head after cheilectomy

Inspection of the sesamoids and plantar plate is carried out via medial plantar access, as already described. The portals are sutured with nonabsorbable 3.0 nylon thread. The dressing is completed with sterile gauze near the surgical access points.

33.9 Postoperative Management

The patient is treated by outpatient procedures. During the first week, a short Walker or orthopedic footwear with a rigid sole should be used. Walking should be allowed if pain free. Antiinflammatory medications are recommended to control pain. Dressing of surgical injuries should be repeated every 2 days, and the stitches removed after 10 days.

For rehabilitation, start early with mobilization (active and passive) of the toe, carrying out gentle movements of circumduction and flexion/extension. Normal footwear can be used after a few weeks, when the patient is able to walk without pain.

The patient may return to noncontact sports (swimming, spinning) immediately after healing of the wounds. Contact sports should be avoided for at least 2 months.

33.10 Complications

Numerous complication from the surgery are described in the literature, such as nerve injuries and iatrogenic cartilage injuries. Van Dijk reported that neuropraxia of the dorsal medial cutaneous nerve is the most frequent [14]. In 2006 Debnath et al. recorded two temporary nerve lesions among 20 patients undergoing arthroscopy of the first metatarsophalangeal joint [15].

Portals should be carried out precisely, ensuring the proper size of the instruments and their proper intraarticular handling to avoid iatrogenic damage to the cartilaginous surfaces. One complication is the risk of surface and deep infections [16].

When treating hallux rigidus, a brightness amplifier or use of surgical devices such as a Kirschner wire is absolutely necessary, enabling the surgeon to properly remove the osteophytes shaping the articular profiles. These devices should be used during the learning curve, where there is a risk of carrying out excessive or insufficient resections, aggravating the articular damage.

33.11 Pitfalls and Tricks

- Carry out a precise clinical assessment of the patient and the pathology to be treated, informing him/her of all the risks and benefits of the surgery. Arthroscopy of the metatarsophalangeal joint cannot be carried out in all cases.
- The major indications for arthroscopic treatment of the first MTP joint are hallux rigidus (first–second in the Coughlin and Shurnas classification), focal osteochondral lesions, synovitis, soft tissue impingement, and loose bodies.
- Position the patient correctly on the operating table.
- Highlight the arthroscopic portals and mark the position of the anatomic structures involved (joint line of first metatarsophalangeal joint, extensor hallucis longus, medial dorsal cutaneous nerve branch).
- The joint should be assessed in line with Ferkel's guidelines.
- Careful use of the instruments is necessary to avoid iatrogenic damage to the joint tissues. If the portals are inadequate, it is advisable to use accessory portals, above all for the plantar sesamoid region.
- For osteochondral lesions, carefully debride the diseased cartilage tissue and stimulate the subchondral bone with small-diameter micropicks for microfractures.
- In the treatment of hallux rigidus, a brightness amplifier is absolutely necessary, enabling the surgeon to properly remove the osteophytes.

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Part X

Rehabilitation

34 Rehabilitation Following Ankle Arthroscopy

Alessandro Corsini and Gian Nicola Bisciotti

34.1 Introduction

Ankle arthroscopy (AA) is a surgical technique widely used for many diseases [1]. The first description of AA was reported in 1931 by Burman [2, 3]. Unfortunately, this first attempt for ankle joint arthroscopy in vivo failed. The author justified the failure with the fact that, in his opinion, the ankle joint was not suitable for arthroscopy because its interarticular access was too much narrow. We had to wait until the 1970s, when the technology concerning the optic fibers, which are part of the arthroscopy devices, substantially improved. Consequently, this technical improvement was described by Watanabe [4] in the early 1970s in the first AA series, which consisted of 28 surgical interventions. Several more publications followed, and nowadays AA represent an irreplaceable diagnostic and therapeutic instrument. In orthopedic practice, the most frequent AA indications are the treatment of anterior impingement syndrome, talar osteochondral defects, removal of loose bodies and ossicles, adhesions, and synovitis [1, 5, 6].

34.2 Rationale of Rehabilitation Following AA

The rationale that guides the rehabilitation following AA should be based on three main points:

1. Improving joint stability and proprioception.
2. Improving muscle strength.
3. Improving range of motion.

These three points are strongly interconnected and interdependent. In fact, during all movements such as running, sprinting, jumps, and change of direction, the ankle joint and its extrinsic and intrinsic muscle–tendon units are subjected to stresses that require an optimal proprioception, a high level of coordination, a significant production of strength, and a full range of motion (ROM). Thus, a loss in any of these biomechanical characteristics may cause a restriction or a true deficit during sport activities. For this reason a rehabilitation program following AA should not only necessarily be based on the these three points but the rehabilitator should be able to identify the point/s that show/s more deficit and build a specific rehabilitation path.

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34.3 Improving Joint Stability and Proprioception

Proprioceptive training (PT) represents a fundamental rehabilitation method after AA [7]. Nevertheless, PT is usually based on proprioceptive exercise performed in a semistatic condition or, at least, in low dynamic conditions (i.e., exercises performed in single-limb stance, balance-board exercises, and coordination exercises performed on teeter boards, etc.) [8, 9]. However, there are no precise indications in the literature, or any kind of consensus, indicating the exact dosage, intensity, and frequency of the PT [10]. However, it is generally accepted that to achieve a positive outcome, PT should be performed for a minimum period between 4 and 6 weeks, for three to five weekly training sessions [8, 9]. But at this point, in our opinion, it is important to offer a constructive criticism concerning PT in its classic interpretation. As previously mentioned, PT is usually performed at low execution speed, easily controllable by the subject, and, sometimes, in visual feedback conditions with the use of specific computerized equipment. These training situations are not only facilitated by, but enormously different from, those that may be found during sports activities. Indeed, it is important to underline that the muscular responses against an external stress are of three types:

1. A "reflex type muscular response" that shows a medium latency response and needs about 60 ms for its execution.
2. A "reflex type muscular response" that shows a long latency response and needs about 140 ms for its execution.
3. A "voluntary type muscular response" that requires not less than 200 ms for its execution.

In addition to this, it should be remembered that all human movements can be managed in two different modalities: open circuit modality (OCM) (Fig. 34.1) and closed circuit modality (CCM) (Fig. 34.2) [10]. During the movement realized in CCM, the information transmitted to

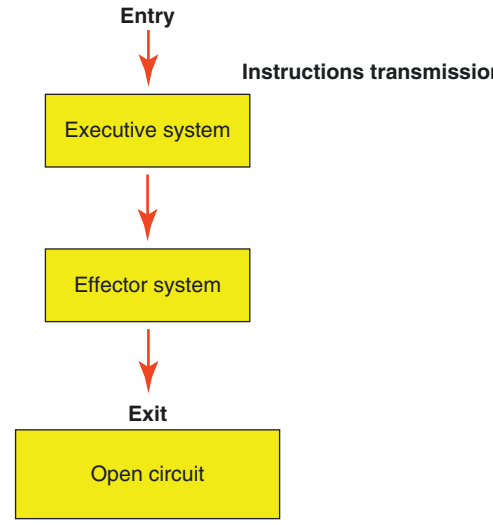

Fig. 34.1 The open circuit modality (OCM) is typical of very fast action. In OCM the transmission shifts directly from the execution system to the effector system, without being able to perform a corrective feedback mechanism

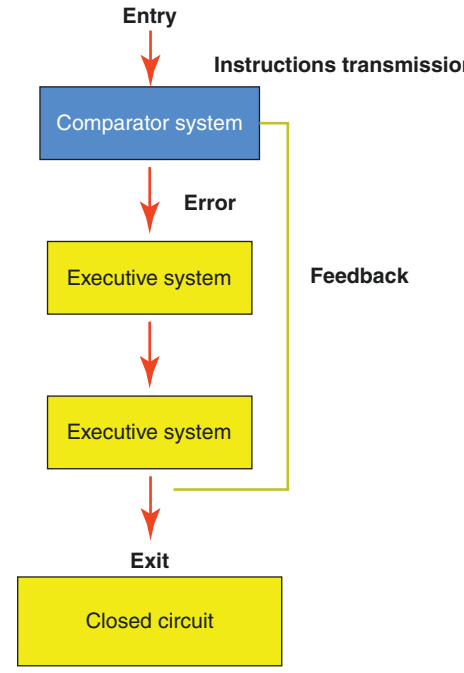

Fig. 34.2 In the closed circuit modality (CCM), the outgoing movement is continuously returned for the control of action to the comparator system that, through a feedback mechanism, is able to correct the movement during its execution

the central nervous system (CNS) is sent directly to the effector system (i.e., the muscle–tendon unit, MTU) without correction during the movement. Conversely, during a movement realized in OCM the information transmitted to the MTU is filtered by the so-called comparator system (CS). The CS compares the movement execution during its course and, if necessary, changes the movement in intinere. It is important to underline that only the movement performed with a voluntary muscular response (>200 ms) may be carried out by OCM. For this reason, if we consider that if an external force is applied on the ankle joint, the "failure time" (i.e., the time required to the mechanical rupture) for the anterior talofibular ligament is approximately 30 ms and the time required to perform a voluntary response able to counteract the inversion movement is approximately 215 ms [10, 11], it is clear that the protective effect offered by a classic PT is ineffective. In such dynamic conditions the only effective solution for stabilizing the ankle joint is to increase the stiffness of the ankle MTU. The increase in stiffness may be obtained through the systematic execution of dynamic proprioceptive training (DPT) [10]. You can find some example exercises in Video 34.1, scanning via QR code. The DPT is based on proprioceptive exercise, performed under a single form or circuit, inducing a preactivation of the MTU ankle joint and increasing its stiffness. This stiffness increase makes the ankle joint most stable and best suited to withstand violent and sudden mechanical stresses. It is clear that DPT represent the last step of PT and must be proposed only after a correct progression training. An example of DPT is observable in the following films.

34.4 Improving Muscle Strength

The foot has a total of 32 muscles, 13 of which are intrinsic and 19 are intrinsic. The intrinsic muscles are those most implicated in the force generation of the talotibial and the subtalar joints [12]. The movements generated by the foot muscular complex are dorsal and plantar flexion and the inversion and eversion movements. Furthermore, the soleus and gastrocnemius muscular complex shows the most important propulsive function and consequently are the strongest plantar flexors [12]. Many sport activities require great foot muscle force level [13]. During sport activities based on running, sprinting, jumps, and cutting movements, both maximal strength and stamina are required [14]. Furthermore, it is important during the rehabilitation plan to obtain an optimal balance between agonist and antagonist muscles. Indeed, an incorrect muscular balance between eversion and inversion muscles (with the eversion muscles too weak in comparison to the inversion ones) may predispose the subject to lateral ankle ligament injuries [15]. We must consider that the immobilization period after AA and the resulting reduced muscle activity cause a sharp weakening of the foot muscles. The degree of muscle atrophy and weakness is related to the duration of the immobilization period and the position in which the immobilization is performed: with the muscles immobilized under tension, atrophy is significantly less than when they are immobilized in a relaxed position [16]. It is important to note that a joint effusion also may cause a reduction in muscular strength as well as in pain [17]. For all these reasons, strength training should start as soon the clinical condition of the patient permits the beginning of muscular application.

Isometric strength training could start when the patient is pain free. The strength training in concentric and eccentric modalities should be started only when the patient is pain free and shows full ROM. In any case, it is important to remember that a reduction in muscle strength may persist for years after the AA because of the mechanism of reflex inhibition caused by disuse [17].

34.5 Improving Range of Motion

A reduction of ankle ROM may penalize participation in many sports activities and even make some of them impossible. In any case, a strong

reduction of ankle ROM may interfere with normal daily living activities. For example, if the subject is not able to dorsiflex the ankle joint at least 10° beyond the neutral position, lameness will be seen during normal walking. In the literature, the passive ROM of the talotibial joint (TTJ) varies in relationship to the different methods used in the different studies. The TTJ dorsiflexion values are between 10° and 23° and the plantarflexion values vary between 23° and 48° [18]. It is important to remember that the most important restraint concerning passive dorsiflexion is the stiffness of the Achilles tendon, whereas in passive plantarflexion the main restraint is represented by both the stiffness of anterior MTUs and posterior bony impingement [18]. Regarding the subtalar joint (STJ), it important to note that it rotates around a biomechanical axis that is continually changing during ankle and foot movement. In the sagittal plane this axis shows an inclination equal on average at 42° with a medial deviation; if observed in the horizontal plane, it is equal on average at 32°. The STJ passive ROM is on average 30° in inversion and 10° in eversion [19]. If the purpose of AA was to remove the cause or causes of ROM restriction, the purpose of the rehabilitation plan will be to restore the normal ROM. In any case, is important to underline the fact that any type of AA causing a period of relative immobilization (that causes capsular contracture and an increase in passive muscle/tendon stiffness) reduces both passive and active ankle ROM. For this reason, ROM recovery is an important step of the rehabilitation following AA. The recovery of the passive ROM is based on so-called joint manipulations (JM). In general, the JM techniques such as the Maitland mobilizations [20], the Mulligan mobilizations [21], and high-velocity/low-amplitude thrusts [22, 23] seem to give the most important evidence of effectiveness concerning the ankle joint. Some studies report an immediate improvement of the ankle passive and active ROM following a single session of manipulative therapy carried out immediately after the removal of the immobilizing cast [24]. However, we must remember that in the current literature studies that compare the effectiveness of the various mentioned manipulative therapies are lacking. Finally, we would like to report that the weight-bearing lunge test (WBLT) is an interesting and reliable test to measure ankle dorsiflexion during weight-bearing [25].

34.6 Conclusions

The rehabilitation plan following AA must necessarily be based on the need to improve stability, proprioception, strength, and ROM of the ankle. Furthermore, it is particularly important that the rehabilitation plan is based on the joint biomechanical requirements of the sports activity carried out by the patient. Unfortunately, to date there are no studies in the literature comparing the effectiveness of the various rehabilitation plans used. For this reason, more studies with good evidence that clarifies the various aspects of the problem are required.

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