

THIRD EDITION

Baxter's

THE FOOT AND ANKLE IN SPORT

DAVID A. PORTER
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The third edition of “Baxter’s The Foot and Ankle in Sport” is finally here! We are excited to enhance your knowledge and skills with this unique updated textbook. So, “What’s new?” We have created five fresh chapters and have done major revision on over 50% of the chapters. More than 50% of the authors are new to this edition and reflect diversity in experience and perspective. We have included new, younger authors, as well as retained some of the best names in orthopedic athletic foot and ankle care. To further enhance the reader’s experience, we have incorporated a video section highlighting radiographic and ultrasonographic techniques as well as physical assessment of the dancer.

This text focuses on the evaluation, decision making, treatment algorithms, and rehabilitation of the athlete at all levels. Often, the technical surgical approaches to athletes are similar to the nonathlete, but the decision making, impact on return to play, interplay with other healthcare providers and caregivers, as well as the rehabilitation of the athlete take more specific detailed education. We have attempted to elucidate this aspect of care well.

We start the book off in this edition with a thorough outline and explanation of a good, standard foot and ankle examination that we use on most all our athletes, *Evaluation of the Athlete*. This is a complete rework of the 2nd edition chapter and takes a totally different approach. To augment the chapter, we have included extensive videos on examination to better help the reader incorporate the exam into their practice. We have also integrated unique ultrasound examination of common foot and ankle ailments and dynamic radiographic examination of other foot and ankle injuries. A series of videos on assessment of the dancer is presented to enlighten our readers. We hope these

ground-breaking videos educate more about our approach and the anatomy.

The chapter on *Medical and Metabolic Considerations in the Athlete* is the most up-to-date and authoritative treatise on the subject. The full extensive chapter is available online, but a more streamlined version is in the written text. We recommend you avail yourself of the whole chapter. We have also included the chapter, *The Team in the Care of the Athlete*, to help with assessing this critical area within the care of the athlete. The chapter specifically discusses how the healthcare provider and specialist can interact with all the other providers, caregivers, patient representatives, and team administrators. We have taken an interview and key word approach and think it delivers at every level from perspective of the athlete, athletic trainer, general manager, parent, and specialist. *Tendonoscopy and Arthroscopy of other Foot and Ankle Joints* looks at the ever-expanding role of arthroscopy and endoscopy in the evaluation and treatment of the foot and ankle. *The Military Athlete* is a unique new chapter that delineates and addresses needs, circumstances, and considerations in this special population of athletes. We are not aware of another foot and ankle text that addresses this. Lastly, we have also added a chapter on the ever-expanding *Orthobiologics in Foot and Ankle Surgery*. This chapter gives up-to-date information on biological additives, subchondroplasty, cartilage substitution, and other bone and cartilage options for operative and nonoperative treatment. We think it is a must-read for all those caring for the athlete.

We hope you enjoy the book. We welcome any feedback you would like to give us. As always, we hope all your foot and ankle patients get better.

David A. Porter, MD, PhD, and Lew Schon, MD, FACS

The Basic Foot and Ankle Physical Exam

David A. Porter

OUTLINE

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The physical examination is essential to properly diagnose ailments of the foot and ankle. Subsequent to obtaining a patient's history and noting the patient's overall well-being, the medical professional conducting a routine exam of a new patient should investigate the following on the involved and uninvolved foot and ankle:

****Please view the following videos that accompany this chapter, especially the primary video of the foot and ankle exam: *Examination of the Foot and Ankle in the Athlete* (Video 1.1)**

Other mini C-arm X-ray evaluations are useful to the examination of the Foot and Ankle. (Videos 1.2-1.5)

Ultrasound videos also presented for review. Ultrasound has become more prevalent in musculoskeletal evaluation. We have provided 11 narrated ultrasound videos of common foot and ankle pathology. We recommend you avail yourselves of these videos to learn how to use ultrasound to evaluate foot and ankle pathology from the Achilles, to the peroneals, to the Anterior tibial tendon, to the posterior tibial tendon, to neuromas, and even muscle injury. (See Videos 1.6-1.16)

Please note, from the standpoint of billing and thoroughness, there may be more things you want to include in your exam, such as constitution, skin, lymphatic, psychiatric, etc., but it may not be specific to this exam.

1. Neurologic Exam

- a. The purpose of this exam is to look for light touch sensation in different nerve distributions. Underlying conditions, such as prior surgeries, diabetes, neuropathies, or prior back ailments, may cause decreased light touch sensation.
- b. While the patient is seated, the medical provider should be at the patient's level, if not slightly below. Place the patient's legs in a straight position onto the provider's lap/upper thighs. This makes it easy to examine the foot and ankle while the patient is relaxed.
- c. The nerves should be simultaneously examined bilaterally in order for the patient to differentiate the sensations he or she may feel. This allows the patient to better assess subtle differences in light touch.

d. Nerves Examined

- i. Deep peroneal nerve
- ii. Superficial peroneal nerve
- iii. Medial plantar nerve—found over great toe
- iv. Lateral plantar nerve—felt over fifth metatarsal
- e. If further investigation is indicated, the Semmes Weinstein test is performed. This assesses the patient's ability to feel pressure and examines altered sensations indicative of different neuropathies.

2. Pulses and Edema

- a. At least one pulse on each foot must be felt in order to be considered as having sufficient blood flow to the foot. Good blood flow to the foot and ankle is important for proper healing, especially in patients with preexisting conditions that could impede the return of function, e.g., diabetes and peripheral vascular disease in the older athlete.
- b. While palpating for pulses, the medical professional should place the patient's feet on his or her knees so the patient's legs are completely relaxed. In a non-relaxed position, the lower limb will begin to shake, making it hard to feel the pulses.
 - i. The following arteries are examined bilaterally:
 - I. *Pedal posterior tibial artery*—palpated just posterior to medial malleolus
 - II. *Dorsalis pedis artery*—palpated laterally to extensor hallucis longus muscle over the Lisfranc region of the bigfoot
 - ii. Pulses are reported in reference to their rhythm and quality.
 - I. "No Pulse (NP)" is reported if no pulse is felt. If no pulse is felt, the medical provider checks to make sure the foot is at least warm to the touch.
 - II. If either pulse is felt, it is reported as regular or irregular, noting bradycardia or tachycardia. A "+1" or "+2" is additionally added to describe the ease of finding the pulse; "+2" means the pulse was found quickly.

- c. **Edema** is reported on a scale of 0–2, with 2 being a substantial amount of edema. Fullness (“puffiness”) and swelling (noticeable edema) should be described along with the location of the edema.

3. Musculoskeletal Exam

- a. **Gait** is observed and reported as normal or antalgic, which means that it has changed in order to avoid pain. This is a good indication that a patient may have problems bearing weight on the involved foot and/or ankle.

- b. **Knee Alignment**

- i. It is essential to always look at knee alignment with the patient/athlete fully standing in a weight-bearing position, as some ailments are related to malalignment.
 - ii. Knee alignment is not measured in degrees. It is reported as varus (angled medially toward midline), valgus (angled laterally from midline), or neutral. The patient’s feet are placed together to get the best reading of varus and valgus alignment.

- c. **Deformity** notes the different types of deformities present and the impact they have on the function of the foot and/or ankle. One example would be a pronation deformity. All deformities should be reported as mild, moderate, or severe. Another is femoral ante version that can be associated with patellofemoral malalignment and pronation deformity.

- d. **Hindfoot Angle**

- i. This angle is measured by aligning the goniometer from the center of the calf, through the center of the Achilles tendon, to the center of the plantar heel as the patient is standing with his or her feet shoulder width apart facing away from the medical provider so the provider sees the back of the patient.
 - ii. The hindfoot angle is reported as varus, valgus, or neutral. If in varus or valgus, the angle measurement is reported with it.
 - iii. The deltoid ligament is crucial to medial instability. Like the lateral collateral ligament of the knee for varus stability, it is under tremendous tension during stance. The knee often is in varus, while the hindfoot is often seen in valgus, thus the medial tension on the deltoid.
 - iv. Common measurements for males in our experience are 2–3 degrees of valgus, while for females they are 3–4 degrees of valgus, but the variance can be as great as 3 degrees of varus up to 6–8 degrees of valgus. Dancers often have more of a neutral or sometimes varus hindfoot angle.
 - v. Hindfoot angle, type of alignment, and type of injury are all considered when deciding on the proper treatment. For example, if a patient has a hindfoot varus malalignment (walking more on the lateral heel) with chronic ankle instability, alignment must be considered and possibly fixed because a reconstruction could fail due to continued stress of malalignment.

- e. **Calf Tone**

- i. Observed during a bilateral toe raise, calf tone reports the size of the calf while looking at the contour of the muscle. It is reported as good or decreased.

- ii. Decreased calf tone tells us that the use of the foot and/or ankle has been limited within the last few months, indicating an injury may have occurred.

- f. **Toe Raise**

- i. *Bilateral toe raise* is used to observe the quality of calf tone. It also aids in the understanding of the functioning of the posterior tibial tendon. By the nature of the heel, and the posterior tibial tendon mechanics, as the patient goes up onto his or her toes, alignment of neutral, varus, or valgus can be determined and the function of the posterior tibial tendon (PTT) is assessed. That is, normally, the heel should go from slight valgus in stance to significant varus with heel rise. Pathologic function of the PTT or if the athlete has a subtalar coalition will result in the heel staying in valgus or only getting to neutral. The medical provider should note any pain associated with this maneuver also.

- I. While in a bilateral toe raise, the patient should walk to assess for pain and strength.

- II. Additional walking tests include having the patient walk on his or her heels. This will demonstrate any extensor lag of the foot that patient may be experiencing due to a radiculopathy, or weakness/tear of the anterior tibial tendon.

- ii. *Single toe raise* is used to diagnose and assess specific injuries. The provider should note if the patient is unable to perform the maneuver or if it is considered good or poor. The alignment of varus, valgus, and neutral should also be reported. It is common to see patients limp if they are unable to perform a single toe raise because they cannot push off while walking.

- I. A single toe raise can be used to observe the functional status of the posterior tibial tendon.

- II. During the healing process of an Achilles tendon rupture, a single toe raise can assess calf size to see if there has been an increase in the use of the foot and/or ankle.

- g. **Range of Motion**

- Throughout range-of-motion testing, the provider should sit at the same level of the patient’s lower extremities, if not slightly below the patient. This allows the patient to relax his or her foot and ankle.*

- i. **Straight Leg Raise**

- I. Straight leg raise with the patient still seated, is used as a screening exam to determine if pain in the lower limb involves the back. This is a good maneuver to test for herniated disc pain/sciatica/radiculopathy.

- II. The medical provider lifts the straight leg while the patient is seated to test for shooting pain down the leg. If there is abnormality reported with this maneuver, the patient is then asked to lay supine and perform the leg raise again in order to execute a more thorough examination to assess if the pain is coming from the back instead of tight hamstrings.

ii. Dorsiflexion

- I. Maximal dorsiflexion is measured with a goniometer to assess flexibility in degrees. The angle is created with the anterior edge of the tibia and a line parallel to the plantar sole while the foot is passively positioned into dorsiflexion. We measure dorsiflexion from the medial foot/ankle for proper placement of the goniometer just for the convenience of not having to move our position or the patients (see [video 1.1](#)).
- II. If a patient presents with a severe pronation deformity, the medical provider must first correct the hindfoot alignment before passively moving the foot into dorsiflexion.
- III. Pain during dorsiflexion should be noted.
- IV. The average dorsiflexion angle is 10 degrees to 15 degrees; however, the goal is to get patients to 20 degrees.
- V. Good dorsiflexion is an important part of treatment because it can relieve general pain caused by tightness.

iii. Plantarflexion(PF)

- I. PF is primarily assessed postoperatively to ensure full range of motion has returned.
- II. It is measured with a goniometer by passively putting the foot into PF.

iv. Subtalar Motion

- I. The quality of subtalar range of motion is tested by passively inverting and everting the foot to check lateral and medial range of motion.
- II. It is reported as good or restricted, noting if inversion or eversion is impeded. If restricted, it is reported as mild, moderate, or severe. Pain with the maneuver should also be reported.

- v. Great Toe Extension (GTE)—Is assessed by measuring the angle between the dorsal border of the first metatarsal and the axis of the distal toe itself (see [video 1.1](#)). Normal GTE is 70—90 degrees and is decreased with Hallux Rigidus.

h. Stability (see [Video 1.1](#))

- i. Talar Tilt (Assesses the Calcaneofibular ligament—CFL):
 - I. Talar tilt is assessed with the ankle maximally dorsiflexed and the whole foot and hindfoot tilted into varus through the ankle joint. This is because the CFL is under maximal tension in DF.
 - II. It tests the stability of the calcaneofibular ligament by inverting the ankle joint.
 - III. It is reported as good stability or increased laxity, meaning that there is no or positive ligamentous laxity present, respectively. If increased, it is reported as mild, moderate, or severe.
- ii. Anterior Drawer (Assesses the Anterior Talofibular Ligament—ATFL).
 - I. It tests the stability of the anterior talofibular ligament by holding the tibia stable and then grabbing the whole foot and hindfoot with the other hand and pulling the complex forward to 10–15 degrees of PF.

- II. It is reported as good stability or increased ligamentous laxity. If increased, it is reported as mild, moderate, or severe.

- III. While performing an anterior drawer, the medical provider should look for a positive “suction sign.” When a torn ligament is present, there is negative pressure in the ankle with this maneuver, pulling the skin into the space. These are often accompanied with poor endpoints.

iii. Endpoints

- I. Both the talar tilt and anterior drawer are reported with an endpoint, which is where the movement from the test is halted. It is determined based on laxity during the exam by feeling a firm endpoint, mild softness, or no endpoint, with other soft-tissue structures possibly limiting excursion.
- II. An endpoint is reported as good, poor, soft, or no endpoint.

4. Further Investigation When Indicated by Patient History and Physical Exam

a. Special Tests

i. External Rotation (ER) Test (see also [Video 1.3 Evaluation of Syndesmosis](#))

- I. External rotation test is used to check for syndesmosis (high ankle sprain) injuries.
- II. The medical provider rotates the foot and ankle externally while keeping the lower leg straight. We believe this test is more sensitive, specific, and definitive than the Squeeze test.

ii. Squeeze Test

- I. The squeeze test is also used for syndesmosis injuries; however, the ER test is often considered more sensitive, specific, and definitive.
- II. The provider is looking for reported ankle pain while squeezing the proximal leg around the proximal tibiofibular joint.

iii. Thompson Test

- I. It is used to assess the integrity of the Achilles’ tendon.
- II. The patient lies in the prone position, and the examiner passively bends the knee to a 90-degree angle. As the medical provider squeezes the calf, the foot should plantarflex, showing the Achilles is intact. If there is no PF, the Achilles tendon is ruptured. This is called a positive Thompson Test.
- III. To avoid the confusion regarding what is a positive (+) Thompson test and what is a negative (–) Thompson test, we recommend stating whether there is “+ Plantarflexion with Thompson test” (Achilles intact) or “no plantarflexion with Thompson test” (torn Achilles tendon). We believe this approach is simpler and clearer (don’t have to remember, or expect those reading your report to remember, when the Thompson test is positive or negative, with or without PF).
- IV. It is reported as “+ PF” for positive plantarflexion or “No PF” if there is no plantarflexion. The presence of a palpable defect should be noted.

- iv. Abduction Stress Test
 - I. This test is used to examine Lisfranc ligamentous injuries.
 - II. To perform the test, the lateral foot is held while a valgus stress is applied to the medial foot to test for ligamentous instability of the Lisfranc.
 - III. The presence of pain while performing the test should be reported.
- b. Posterior Ankle Impingement (PAI) (See also [Video 1.4 Evaluation of Os Trigonum](#))
 - i. The provider is assessing the amount of pain with passive plantarflexion or hyperplantarflexion and palpation of the posterior process of the talus (Os trigonum or Stieda process). It is important to ask the athlete if the pain he/she reports is actually in the anterior or posterior ankle; posterior pain implies posterior ankle impingement, while anterior pain with PF implies only stretching of the anterior capsule and does not imply PAI.
 - ii. The medical provider moves the foot into plantarflexion while pushing the heel up so the posterior talus is impinged by the posterior distal tibia and calcaneus.
- c. Reflexes

Reflex examinations should be performed on athletic patients reporting athletically associated lower leg pain. If there is difficulty assessing the following reflexes, have the patient interlock his or her fingers and forcefully pull against them to block any resistance.

 - i. Achilles (S1 reflex)
 - I. It is a neurologic exam used to look at the Achilles jerk reflex.
 - II. The Achilles tendon is tapped while the foot is dorsiflexed. A positive test results in the foot jerking toward the plantar surface.
 - ii. Patellar (L4 reflex)
 - I. It is a neurologic exam used to look at the patellar stretch reflex.
 - II. The test is performed by striking the patellar tendon just below the patella while the knee is bent and relaxed. A positive test will cause the leg to extend (a goniometer itself can be used as a “reflex hammer,” since it is readily available).
- d. Strength

Strength should also be assessed in athletes who report lower leg pain. 5/5 (normal) strength is reported if the medical provider cannot change direction of foot/ankle or lower limb with resistance while the athlete actively moves the foot/ankle during each of the provocative tests.

 - i. Inversion strength
 - I. The purpose of this test is to examine the strength of the posterior tibialis.
 - II. The provider applies medial force while the patient inverts the foot against the force.
 - ii. Eversion strength
 - I. This test examines the strength of the peroneus longus and brevis.
 - III. The provider applies a lateral force while the patient everts the foot against the force.
 - iii. Extension of Knee Strength—While the knee is fully extended, the provider applies force to try to move knee into flexed position.
 - iv. Flexion of Knee Strength—The provider tries to disrupt flexion of the knee by applying an anterior force to the calf in order to extend the knee.
 - v. Abductor Strength—With a patient in a neutral position, the provider pushes the lateral thigh toward the midline while the athlete tries to move the leg away from the midline (Abduction).
 - vi. Adductor Strength—With the athlete in a slightly abducted position, the provider tries to move the athlete’s leg away from the midline (abduct) the leg while the athlete tries to move the leg toward the midline (adduct).
 - vii. Hip Flexor Strength—With the athlete in a seated position with the knees in flexion, the athlete should lift one knee at a time against downward resistance force applied by the provider.
- e. Palpation of Nerve Pain
 - i. Deep peroneal nerve (DPN)
 - I. Located lateral to first metatarsal and medial to second metatarsal (the soft space interval between these two metatarsals), it can be palpated in the soft space distal to the Lisfranc ligament.
 - II. Pain in this area is characteristic of deep peroneal nerve entrapment.
 - III. The athlete does not always have numbness, tingling, or light touch deficits with DPN entrapment.
 - ii. Superficial peroneal nerve (SPN)
 - I. It can be palpated approximately four finger-breadths above the distal tip of the fibula, just anterior to the fibula (can be coming out of the anterior or lateral compartment). Typically seen traversing the ankle over the anterolateral ankle joint with the ankle PF and inverted.
 - II. Localized pain can occasionally radiate to the top of the foot.
 - III. Pain to palpation that reproduces the athletes discomfort is the most common manifestation of SPN entrapment.
 - iii. Medial plantar nerve (MPN)—Palpate the nerve just inferior to the abductor hallucis muscles at the level of the medial navicular
 - iv. Lateral plantar nerve (LPN)—This nerve is hard to palpate, but with isolated LPN entrapment, one notes pain with palpation of the PT (posterior tibial) nerve posterior to the medial malleolus. (This entrapment can occur with an accessory flexor tendon in the tarsal tunnel—it can abut the LPN only and cause lateral plantar foot pain.) **See Chapter 11 section on Tarsal tunnel.**
 - v. Posterior tibial nerve
 - I. Palpated posterior to posterior tibial tendon and flexor digitorum longus tendon in the tarsal tunnel.

- II. Pain with or without palpation in this area can be indicative of neuralgia or tarsal tunnel syndrome. Further testing is required to determine cause. **See Chapter 11 section on Tarsal tunnel.**
 - vi. Sural nerve
 - I. It is located posterior to the fibula, and it is often palpated along the lateral malleolus, or just posterior to it and anterior to the Achilles.
 - II. Though uncommon, sural nerve entrapment can occur with pain in this localized area.
 - III. Also, sural nerve can be associated in rare cases after chronic calf strains with excessive scarring. In this scenario, the pain is in the central calf between the medial and lateral heads of the gastrocnemius where the sural nerve runs at the level of the musculoskeletal junction.
 - f. Suspicion of Neuroma
 - i. If nerve pain is suspected, the provider should separately palpate each individual web space on the dorsal and plantar surface of each foot noting any difference in sensation, radiation into the toes, or pain.
 - ii. The Mulder Click, noted with medial and lateral compression of the forefoot while simultaneously palpating the webspace. We have noted this is a common finding due to movement of local tissues (tendons and bursae) even in those without any other evidence of a neuroma. Therefore, we do not put a lot of weight on this finding.
 - iii. It is important to palpate each space and not just the symptomatic interspace. If a patient is symptomatic in all web spaces, it could be indicative of neuralgia or tarsal tunnel syndrome. Pain, primarily localized to one interspace (2-3 or 3-4 interspace) is suggestive of a symptomatic neuroma. Doing this discriminating exam can help differentiate nerve pain caused by a neuroma or nerve pain caused by neuralgia or tarsal tunnel.
 - g. Dorsal Foot Pain
 - i. The medial provider should specifically examine the deep peroneal nerve found between the first and second metatarsals. The first and second metatarsals along with the soft space in between should be palpated.
 - ii. The metatarsophalangeal joints (MTP), especially the second MTP joint and occasionally the third MTP, should be moved passively into plantarflexion to assess for MTP synovitis. Passive PF of the joint tightens the capsule and, with synovitis, can result in severe pain.
 - h. Great Toe Pain (see also [Video 1.2 Evaluation of Turf Toe](#))
 - i. If the athlete reports plantar great toe pain, both sesamoids should be examined. If the pain is acute with a history of GTE injury and swelling, then turf toe must be considered and evaluated.
 - ii. The provider can often palpate well between the tibial sesamoid and fibular sesamoid, and the plantar plate (turf toe injury) is just distal to the sesamoids.
 - i. Lower Leg Pain
 - i. Palpation for Musculoskeletal Pain—Stress Fracture
 - I. Anterior crest of the tibia—anterior tibial stress fracture
 - II. Proximal tibial stress fracture—posteromedial tibial metaphysis
 - III. Distal tibial stress fracture—posteromedial tibial metaphysis and distal tibial diaphysis (medial tibial stress syndrome [MTSS] will have pain over larger area, stress fracture has focal pain)
 - IV. Distal fibula
 - a. The fibula is subcutaneous over the distal 8-10 centimeters. The SPN can be confused with the distal fibula stress fracture. The SPN exits its fascial compartment 3-4 finger breadths above the ankle. The SPN is usually more anterior to the fibula at this level, and to differentiate between SPN entrapment and fibula stress fracture, the provider palpates the posterior border of the fibula, which will be pain free in SPN but still painful with fibular stress fracture.
 - ii. MTSS (pain at soleus bridge and soleus/gastroc soft tissue attachment to medial tibia)
 - I. We want to differentiate the pain from a distal tibial stress fracture and this soft-tissue attachment that is akin to a “tennis elbow of the leg.”
 - II. Starting at the distal posterior tibial tendon on the medial side, the provider palpates up the medial tibia, marking where pain is felt. The provider then starts proximally and moves distally along the medial tibia noting the location of pain.
 - III. To ensure accurate location of pain, it is recommended that a mark with a pen be made at the two extents of pain. Typically, the pain is over a 8-15-cm segment of the posteromedial tibia. We measure and document how far each mark is from the distal tip of the medial malleolus.
 - IV. MTSS has a much larger area of pain (8-15 cm), and distal tibial stress fracture has a very focal and small area of pain (3-6 cm).
- In addition, the medical provider should note any bruising, abrasions, varicosities, or masses that are seen throughout the physical examination. Pain and tenderness with palpation and movement should be described by their location and severity.

Video Legends - <https://www.kollaborate.tv/link?id=5c9d1f8590f70>

- ▶ **Video 1.1** Title: General Complete Physical Exam of the Foot and Ankle Legend: This video demonstrates a complete and detailed physical examination of the foot and ankle.
- ▶ **Video 1.2** Title: Mini C-arm Evaluation for Turf Toe Legend: Mini C-arm of the great toe with hyperextension to assess if the sesamoids move with movement of the toe itself.
- ▶ **Video 1.3** Title: Mini C-arm Evaluation of the Syndesmosis Legend: Mini C-arm of ankle syndesmosis with abduction stress demonstrating widening of the medial clear space and the syndesmosis. External rotation stress shows more subtle rotational instability.
- ▶ **Video 1.4** Title: Mini C-arm Evaluation of the Os Trigonum. Legend: Mini C-arm of the Os trigonum shows movement of the Os-trigonum with plantarflexion and dorsiflexion, and with needle injection, the bone moves also.
- ▶ **Video 1.5** Title: Mini C-arm Evaluation of an Apophyseal Nonunion of the 5th Metatarsal Legend: Mini C-arm of the lateral foot with a sterile needle demonstrates that the apophyseal nonunion is actually loose and will likely remain symptomatic without removal.
- ▶ **Video 1.6** Title: Ultrasound Evaluation for Base 5th Metatarsal Fracture. Legend: Ultrasound over lateral foot to demonstrate a 5th metatarsal Jones stress fracture.
- ▶ **Video 1.7** Title: Ultrasound Evaluation for Anterior Tibial Tendon Pathology. Legend: Ultrasound over the anterior ankle is utilized to assess the anterior tibial tendon. It can evaluate for tendinopathy or tear.
- ▶ **Video 1.8** Title: Ultrasound Evaluation for Achilles Tendon Tendinopathy. Legend: Ultrasound over the Achilles shows the thickening of the midsubstance consistent with tendinopathy.

Video 1.9 Title: Ultrasound Evaluation for Achilles Tendon Partial Tear. Legend: Ultrasound over the Achilles shows the partial tear. Note there is partial tear but not a full-thickness tear. ▶

Video 1.10 Title: Ultrasound Evaluation for Dislocating Peroneal Tendon. Legend: Ultrasound over the lateral ankle demonstrates subluxation/dislocation of the peroneus longus over the posterolateral fibula. ▶

Video 1.11 Title: Ultrasound Evaluation for Intrasheath Peroneal Tendon. Legend: Ultrasound over the lateral ankle demonstrates subluxation of the peroneal tendon side-to-side on each other within the expanded sheath. ▶

Video 1.12 Title: Ultrasound Evaluation of Posterior Tibial Tendon Tear. Legend: Ultrasound over the medial ankle shows an abnormal posterior tibial tendon consistent with a degenerative posterior tibial tendon tear. ▶

Video 1.13 Title: Ultrasound Evaluation of the Plantar Fascia. Legend: Ultrasound over the plantar medial heel demonstrates chronic plantar fasciitis. This can be used diagnostically and also to direct needle injection. ▶

Video 1.14 Title: Ultrasound Evaluation of the Peroneal Tendons. Legend: Ultrasound over the lateral ankle demonstrates a longitudinal split tear of the peroneus brevis tendon. The peroneus longus tendon is found to be free of tears. ▶

Video 1.15 Title: Ultrasound Evaluation of the Peroneal Muscles Legend: Ultrasound over the lateral ankle demonstrates degeneration of the peroneus brevis muscle belly with a normal-appearing gastrocnemius and soleus muscle. ▶

Video 1.16 Title: Ultrasound Evaluation of the Plantar Foot for Neuroma. Legend: Ultrasound over the plantar foot demonstrating a 3-4 interdigital neuroma, showing the subluxating neuroma between the 3-4 metatarsal with squeezing of the foot. ▶

Impingement Syndromes of the Ankle

Michel A. Taylor, Annunziato Amendola

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INTRODUCTION

Impingement is derived from the Latin verb *impingere*, meaning “to force against,” where an anatomic structure becomes entrapped causing pain, bony and soft tissue injury, decreased range of motion, and dysfunction.^{1–3} When impingement occurs, one bone repetitively strikes another, which over time can stimulate the deep layer of the periosteum to form osteophytes that further exacerbate the impingement and alter the normal mechanics of the ankle joint. Osseous or bony impingement most commonly occurs following spur formation along the anterior margin of the distal tibia and talus or as a result of a prominent posterolateral talar process—the os trigonum (OT). Soft tissue impingement usually results from scarring and fibrosis associated with synovial, capsular, or ligamentous injury and most often occurs in the anterolateral gutter, the medial ankle, or in the region of the syndesmosis.⁴ Conservative treatment such as activity modification and retraining should be aimed at breaking this repetitive cycle in order to decrease the inflammation and provide relief. However, the sporting limitations are often limiting, and if conservative management fails, surgery is usually indicated.

GENERAL TECHNIQUE TIPS FOR OSTEOPHYTE REMOVAL

1. Obtain adequate exposure and visualization prior to attempting removal, to ensure complete excision
2. Careful placement of skin incisions in order to avoid painful neuromas and hypersensitive scars.
3. The surgeon should be meticulous with osteophyte removal. Care should be taken to remove all of the impinging osteophyte. Intraoperative fluoroscopy can be utilized to confirm complete removal.

Studies have shown that preoperative computed tomography (CT) scan may improve the characterization of the osteophytes and lead to more complete removal at the time of surgery.⁵

The Ankle

In order to understand and appropriately manage ankle impingement conditions, one must first understand the anatomy of the ankle joint. The talus articulates inferiorly with the os calcis, and the talar axis is in line with the first web space of the foot, while the axis of the os calcis is in line with the fourth web space (Fig. 2.1). In dorsiflexion, bony impingement occurs anteromedially between the neck of the talus and the anterior lip of the tibia. In plantarflexion, bony impingement occurs posterolaterally between the os calcis and the posterior lip of the tibia. Therefore, anteromedial and posterolateral impingement is usually bony in nature while anterolateral and posteromedial impingement is usually a soft tissue condition. Impingement conditions are most commonly seen in the anterior ankle; however, they can also occur in all quadrants of the ankle joint, including anterior, lateral, posterior, and medial.

Anterior (Medial, Central, Lateral) Impingement

Anteromedial Impingement

Anterior ankle impingement is relatively common in the athlete. Repetitive forced dorsiflexion can lead to frequent impaction injuries as the talar neck and hypertrophied synovial tissue impinge on the anterior lip of the tibia leading to the formation of osteophytes over time.⁶ Recurrent supination injuries in the form of inversion ankle sprains, seen in chronic ankle instability, can also cause inflammation and hypertrophic changes of the synovium between the talus and tibia.⁷ If the instability is not addressed, synovitis and scar tissue will accumulate most commonly in the anterolateral gutter leading

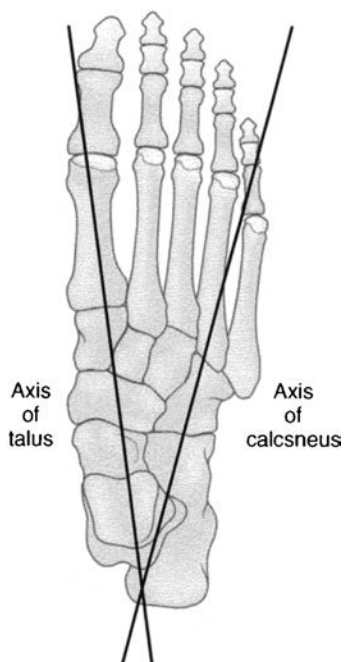


Fig. 2.1 Axis of talus versus axis of os calcis.



Fig. 2.2 Lateral impingement and stress fracture.

to pain, swelling, continued impingement and lateral column overload, and stress fractures (Figs 2.2, 2.3). Anteromedial ankle impingement occurs when the anterior portion of the medial malleolus impinges against a spur on the medial shoulder of the talus. The medial joint spurs form following injuries to the deltoid ligamentous complex. In addition, the anterior tibiotalar ligament becomes thickened and can impinge on the anteromedial corner of the talus. Patients typically complain of anteromedial joint line pain, which is aggravated by walking and running as well as planting and pivoting during sporting activities. They also report medial clicking and limited, painful dorsiflexion. Patients often have anteromedial ankle swelling and pain over the anterior aspect of the deltoid ligament, and the spur can often be palpated on physical exam. Anterior osteophytes are often seen on standard lateral weight-bearing plain x-rays. The “plié view,” which is a lateral weight-bearing view in maximal dorsiflexion, can be useful to demonstrate anterior impingement. With regards to the anteromedial talar and tibial bone spurs, plain anteroposterior and lateral radiographs can often miss these osteophytes. Studies have shown that spurs over 7 mm in size can be missed in these locations due to projectional issues and the relative positions of the



Fig. 2.3 Oblique stress fracture.

lateral talar neck and body and anterolateral tibia.⁸ An oblique anteromedial impingement view (AMI) can be obtained by plantar-flexing the foot and externally rotating the leg to 30 degrees with the x-ray beam tilted 45 degrees cranial. This view has been shown to be especially useful in detecting anteromedial bone spurs.⁸ During arthroscopy, the anteromedial ankle should be adequately visualized and inspected for the presence of an impingement spur, which should be debrided when present. Holding the ankle in dorsiflexion will help with visualization and any required debridement.

Another form of anterior impingement was described by Amendola et al., as cam-type impingement of the ankle, similar to the impingement that has previously been described in the femoral neck. This form of impingement occurs when the sagittal contour of the talar dome forms a non-circular arc with anterior talar flattening and creates pathologic contact with the anterior aspect of the tibial plafond with the ankle in dorsiflexion (Fig. 2.4). On lateral weight-bearing foot x-rays, the cam ratio is measured by drawing a line under the lateral process of the talus parallel to a line drawn along the navicular, cuneiform, and metatarsal bones. From this line, the widest and narrowest portions of the talus are measured distal to the apex of the talar dome (Fig. 2.5). The cam ratio is then determined by dividing the narrowest point by the widest. The α -angle is determined for both the medial and lateral talar domes. A circle is drawn to match the curvature of the tibial plafond. This circle is then overlaid with the curvature of the talar domes. The α -angle is positive when the radius of the talar dome exceeds the radius of the circle anteriorly. The authors found that the medial dome was most prominent and anterior in cases of impingement. The authors also found that cam-type ankles were associated



Fig. 2.4 CAM lesion. From Amendola N, Drew N, Vaseenon T, Femino J, Tochigi Y, Phisitkul P. CAM-type impingement in the ankle. *Iowa Orthop J.* 2012;32:1-8.



Fig. 2.5 CAM measurement. From Amendola N, Drew N, Vaseenon T, Femino J, Tochigi Y, Phisitkul P. CAM-type impingement in the ankle. *Iowa Orthop J.* 2012;32:1-8.

with higher cam ratios and positive α sign when compared to control ankles. The authors suggest that if unrecognized and untreated, cam-type impingement, like femoroacetabular impingement, can lead to recurrence as well as degenerative arthritic changes.⁹

Anterocentral Impingement

Anterocentral impingement is the classic location when describing anterior ankle impingement. The locations of the spurs have been described in four typical patterns:

1. Spurs found primarily on the lip of the tibia. This type is ideal for arthroscopic management. Under direct vision, the impinging lip of the distal tibia can be easily removed with a burr or thin osteotome. The osteophyte is removed while holding the ankle in maximal dorsiflexion while using an osteotome with blunt edges to avoid damaging the dome of the talus.¹⁰

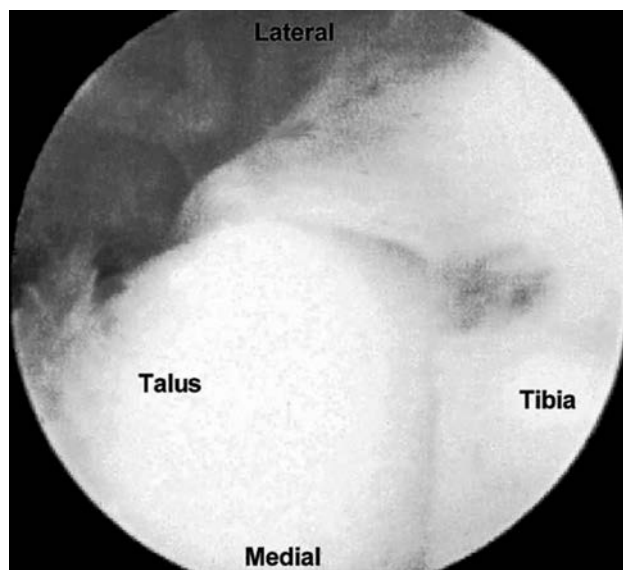


Fig. 2.6 Bassett's ligament. (Arthroscopic view seen from the medial portal.)

2. Spurs found primarily on the talus. This pattern of impingement is more difficult to treat with only an arthroscopic technique. The osteophytes are typically found within the capsular insertion on the talar neck, and it becomes necessary to strip the capsule distally in order to visualize the pathology. In addition, the osteophytes are easily missed, and therefore intraoperative imaging may be needed to ensure adequate removal.
3. Spurs are present on both the lip of the tibia *and* the talar neck. This pattern is also associated with loose bodies that have broken off the osteophytes. This pattern is common and the most technically challenging. Surgical management often involves arthroscopy with an associated mini arthrotomy.
4. Multiple anterior osteophytes are present secondary to advanced ankle osteoarthritis. The benefit of arthroscopic debridement in these cases is questionable and should probably not be performed.

Anterolateral Ankle Impingement

Anterolateral ankle impingement is usually secondary to soft tissue impingement, as the tibia and talus do not come together in this location. Impingement in this location is usually a result of one of two pathological conditions: Bassett's Ligament and synovial impingement.

Bassett's Ligament. Bassett's ligament is an abnormal thickened distal fascicle of the anterior tibiofibular ligament (AITFL). Rather than being a distal component of the AITFL, Bassett's ligament represents a separate ligament that is separated by a fibrofatty septum¹¹ and extends distally on the lateral malleolus. It can lead to impingement of the lateral shoulder of the talus when the ankle is plantarflexed (Fig. 2.6).¹² Patients typically present with anterior ankle pain and a remote history of an ankle sprain. Physical exam findings include anterolateral talar dome and AITFL point tenderness exacerbated with ankle eversion and dorsiflexion. The ankle is usually found to be stable on physical exam. Treatment is usually nonoperative

and should be attempted for approximately 6 months prior to considering any kind of surgical intervention. The surgical management of this condition involves the surgical excision of the ligament and has been associated with good to excellent results.

Synovial Impingement. Synovial impingement or Ferkel's disease is the chronic accumulation and entrapment of scar tissue and synovitis in the anterolateral gutter of the ankle, which is usually preceded by trauma in the form of an inversion injury.¹³ The patient typically presents with symptoms similar to Bassett's ligament impingement. Pain with palpation over the anterolateral gutter with the ankle plantarflexed typically reproduces the symptoms. Plain films are usually normal but can be used to rule out other bony pathology. Advanced imaging modalities such as CT scan and conventional magnetic resonance imaging (MRI) have shown moderate sensitivity and specificity but often rely on the experience level of the reader.^{14–18} Magnetic resonance (MR) arthrogram, however, has been associated with a sensitivity of 96%, specificity of 100%, and accuracy of 100% in the assessment of anterolateral impingement when clinical signs are present.¹⁹ Like Bassett's ligament, arthroscopic debridement is the surgical treatment of choice after a trial of nonoperative management has failed, with good to excellent results in approximately 94%–96% of patients.^{20,21}

Anterolateral ankle pain can also be caused by anterior syndesmosis pathology. Although this is not as a result of true impingement, it can be exacerbated by ankle dorsiflexion as the widened anterior aspect of the talus engages the malleoli and places tension on the anterior tibiofibular ligament. There are three types of anterolateral syndesmosis pathology: a sprain of the syndesmotic ligaments and interosseous membrane, also known as a high ankle sprain; the Tillaux fracture, which is an avulsion fracture of the insertion of the AITFL usually on the distal tibia; and the herniation of synovium into rents in the tibiofibular ligament.

Results of arthroscopic anterior ankle debridement have been reported as good to excellent by numerous authors, with success rates of approximately 67%–88%.^{2,13,22,23} A systematic review looking at the results of anterior ankle arthroscopy found good or excellent results in 64%–100% of patients while most studies had outcomes greater than 80%. Improved postoperative outcomes were seen in patients with mostly soft tissue impingement compared to bony impingement.²⁴ Studies comparing open and arthroscopic debridement for anterior ankle impingement found significant postoperative clinical improvements in both groups with shorter hospital stays seen in the arthroscopic group and earlier return to sports.¹⁰ Multiple studies have consistently found that patients undergoing arthroscopic or open debridement in the presence of arthritic changes have inferior clinical outcomes.^{10,23–25}

Lateral Ankle Impingement

The lateral ankle is complex and the causes of pain and discomfort are varied, therefore obtaining an accurate diagnosis can often be difficult. Symptoms in this area are also often preceded by ankle sprains. The original trauma can often be mild

and appear inconsequential at first, such as a first-degree ankle sprain with no residual lateral instability. Other conditions to consider in the evaluation of lateral ankle impingement and pain are:

1. *The “meniscoid” of the ankle* – Thought to be soft tissue trapped between the lateral shoulder of the talus and the lateral malleolus. This lesion was described in four soccer players with a history of frequent ankle sprains who underwent arthroscopic debridement after failing nonoperative treatment.²⁶ After appropriate rehabilitation, all four had complete resolution of symptoms and returned to competition.
2. *Fracture of the lateral process of the talus*²⁷ – Can be a source of impingement beneath the lateral malleolus. The fracture is also known as a “snow-boarder’s fracture” due to the increased incidence in this particular athletic population. It is often misdiagnosed as an ankle sprain, therefore a high index of suspicion is required. Routine plain radiographs of the foot and ankle can often miss the subtle fracture, and a CT scan is the study of choice. Surgical treatment options range from bony excision to open reduction internal fixation (ORIF), depending on the size of the fragment.²⁸
3. *The symptomatic os subfibulare* – An accessory ossicle that was previously asymptomatic can loosen or fracture following an injury and become symptomatic.
4. *Distal fibula avulsion fractures* – The tip of the fibula can often become trapped at the insertion site of the calcaneofibular ligament (CFL) and become symptomatic. If the fragment is small, it can be excised and the stump of the ligament can be sutured to the tip of the lateral malleolus. If it is large, it often can be reattached with a screw or K-wire. Infrequently a similar fracture can occur at the anterior edge of the lateral malleolus at the insertion of the anterior talofibular ligament (ATFL) (Fig. 2.7).
5. *Os Calcis fractures* – Previously healed or malunited fractures of the os calcis can present with lateral ankle pain and subfibular impingement, which is often difficult to differentiate from subtalar joint pain and dysfunction. A small injection of local anesthetic beneath the tip of the lateral malleolus, but not into the subtalar joint, can help elucidate the cause. If there is significant pain relief with local anesthetic, fragment excision may be considered prior to recommending subtalar arthrodesis.
6. *An avulsion fracture of the anterior process of the os calcis*²⁹ – an avulsion fracture of the origin of the EDB and EHB and not a true impingement syndrome. It can usually be seen on x-ray (Fig. 2.8) and suspected on physical examination by point tenderness over the site exacerbated by pronation-supination of the forefoot. If symptoms persist despite nonoperative treatment, excision of the fragment is warranted (see Fig. 2.8).
7. *Accessory anterolateral talar facet* – The accessory anterolateral talar facet was first described by Sewell in 1904 who found it to be present in 10.2% of cadaveric tali.³⁰ A case series by Martus et al. described the association between an accessory talar facet and the anterior process of the calcaneus leading to talocalcaneal impingement and symptomatic rigid flatfoot. Patients typically presented around the age of



Fig. 2.7 Fracture of the tip of the fibula trapped under the lateral malleolus.

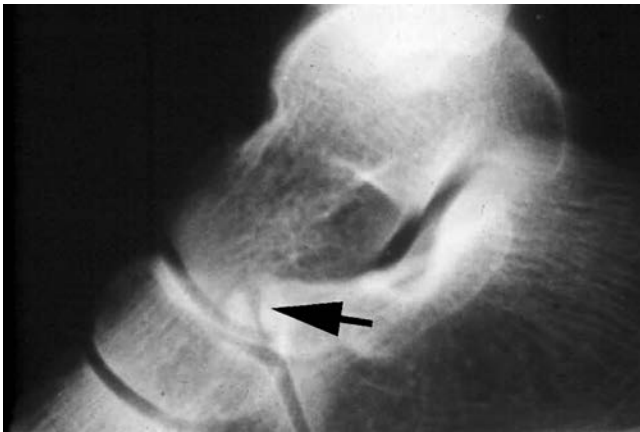


Fig. 2.8 Fracture of the anterior process of the os calcis.

15 with lateral sided ankle and hindfoot pain and symptoms consistent with a rigid flatfoot deformity. The patients were overweight with an average body mass index (BMI) of 34.6, and symptoms had been ongoing for almost 2 years. Lateral radiographs typically showed a broadening of the antero-inferior lateral talar process suggestive of an accessory antero-lateral facet. CT scan and MRI were performed and ruled

out the presence of a tarsal coalition, confirm the presence of the accessory talar facet, dorsal talar beaking was frequently present, and MRI showed bone marrow edema localized to the anterolateral talar facet and adjacent calcaneus. The authors stipulate that symptoms are caused by the combined effect of increased body mass, subtalar joint eversion in the presence of an accessory talar facet leading to impingement and associated painful flatfoot deformity. In the absence of subtalar arthrosis or an associated coalition, treatment consists of isolated resection of the accessory facet through a sinus tarsi incision with or without gastrocnemius recession, peroneal lengthening and medial displacement calcaneal osteotomy.³¹

8. *Peroneal tendon dysfunction* – although not true impingement, can also cause lateral ankle pain and should always be considered in the differential diagnosis. This can include peroneal tendon subluxation, longitudinal tears,³² and fractures of the os perineum with retraction of the peroneal tendon (Fig. 2.9).³³

Posterior Ankle Impingement

Posterior ankle impingement, also known as talar compression syndrome, os trigonum syndrome, posterior triangle pain, and posterior tibiofibular impingement,³⁴ is relatively common in athletes such as dancers, gymnasts, soccer players, and skaters who engage in repetitive and forceful ankle plantarflexion. A working knowledge of the anatomy of the posterior ankle and its key structures is important in order to understand and appropriately manage the impingement syndromes found in this area (Tables 2.1 and 2.2). The posterior talar anatomy is the main determining factor underlying posterior ankle impingement. The posterior aspect of the talus has two tubercles: the medial and the lateral tubercles (Fig. 2.10). The lateral tubercle is the origin of the posterior talofibular ligament (PTFL). When the lateral tubercle is large, it is referred to as the posterior process of the talus or Stieda's process. In 7% to 11% of the population, this posterior process is separate from the talus and connected by a fibrous synostosis; in these cases, it is referred to as an OT. The OT is the second most common accessory bone in the foot, behind the accessory navicular or os navicularum. Anatomical variants like these ones in combination with a traumatic injury such as an ankle sprain or repetitive microtraumas as is seen with certain sports can lead to posterior impingement syndrome.

There are also important soft tissue structures of the posterior ankle that can contribute to posterior ankle impingement such as the flexor hallucis longus (FHL) tendon, the posterior ankle ligaments, the joint capsule, and synovium. The FHL tendon passes in a groove on the posterior tibial plafond and through a fibro-osseous tunnel between the medial and lateral posterior tibial tubercles (Fig 2.11). It runs from its proximal origin on the fibula to its insertion on the distal phalanx of the hallux. Chronic tendinitis and dysfunction within this tunnel can produce posterior medial pain.^{34–36} The most common sources of posterior ankle impingement pain in the athletic population is trigonal impingement laterally and FHL tendinitis medially.

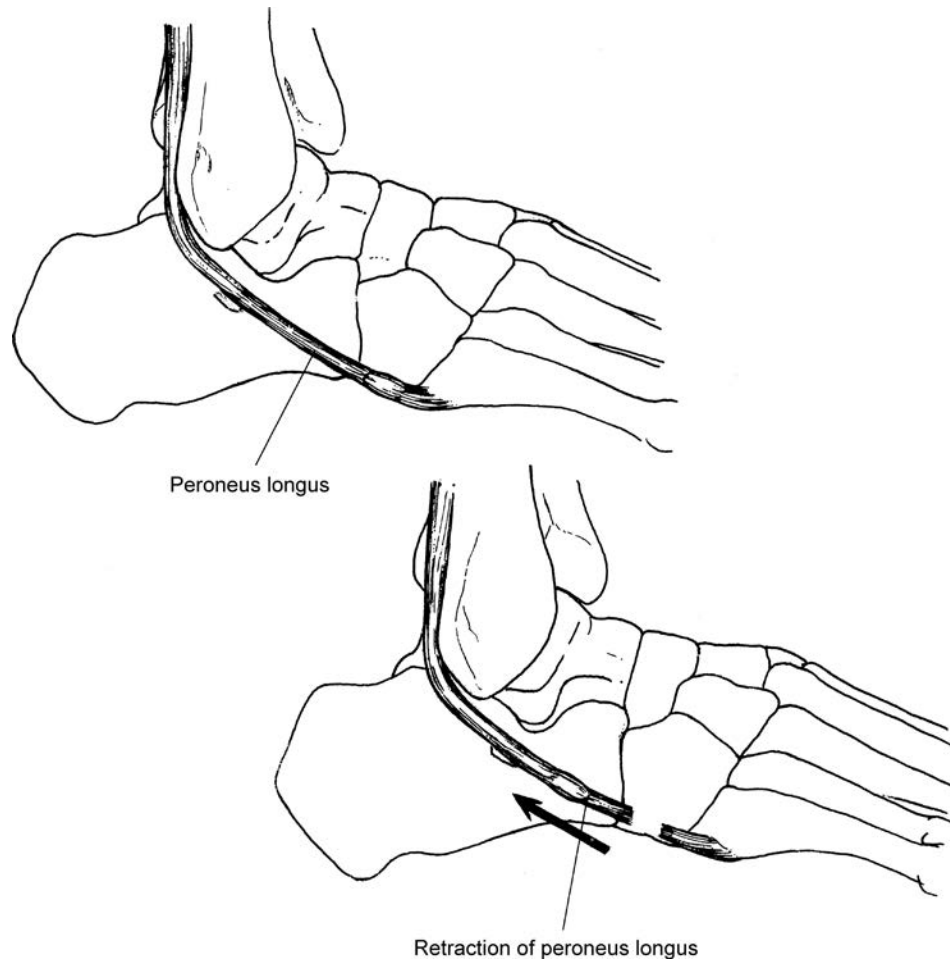


Fig. 2.9 Retraction of the os peroneum (arrow), following rupture of the peroneus longus tendon.

TABLE 2.1 Flexor Hallucis Longus (FHL) Tendonitis Versus Posterior Impingement of the Ankle

FHL tendonitis	Posterior impingement
Posteromedial	Posterolateral
Tenderness over FHL tendon	Tenderness behind fibula
Pain or triggering with motion of the hallux	Pain with plantar flexion of the ankle
±Thomassen's sign ²⁸	Plantar flexion sign
Mistaken for PT tendonitis	Mistaken for peroneal tendonitis

FHL, flexor hallucis longus; PT, posterior tibial.

TABLE 2.2 Medial Versus Lateral Posterior Ankle Pain in Athletes and Dancers

Posteromedial	Posterolateral
FHL tendinitis	Posterior impingement (OT syndrome)
Soleus syndrome	Fx. trigonal process (Shepherd's fracture)
PT tendonitis	Peroneal tendonitis
Posteromedial fibrous tarsal coalition	Pseudomeniscus syndrome

FHL, flexor hallucis longus; Fx., fracture; OT, os trigonum; PT, posterior tibial.

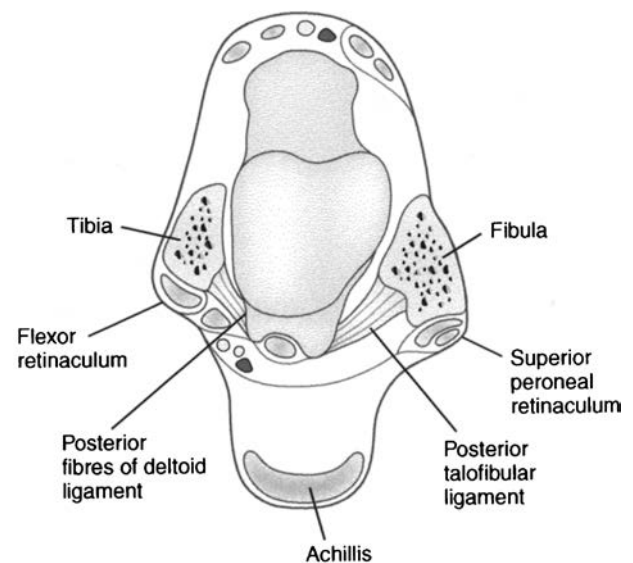


Fig. 2.10 Anatomy of the posterior talus.

Posterolateral Ankle Impingement

A common cause of posterolateral ankle impingement is from the natural consequence of full weight bearing in maximal plantarflexion of the ankle in the *demi-pointe* or full *pointe* position, especially when an OT is present. In these cases, the



Fig. 2.11 Flexor hallucis longus (FHL) posteromedial scope.

OT is compressed between the posterior lip of the tibia and the superior portion of the os calcis (Fig. 2.12) causing posterolateral ankle impingement and pain. The chronic repetitive impingement events can lead to inflammation of the OT. It can be confirmed on physical examination with patients experiencing point tenderness posterior to the peroneal tendons behind the lateral malleolus, which is exacerbated with forced passive plantarflexion of the ankle known as the “plantar flexion sign.” This pattern of impingement can often be mistaken for peroneal tendinitis, which can also occur in association. It is best seen on a lateral view of the ankle *en pointe* or in full plantar flexion (see Fig. 2.12). MRI findings often include bone marrow edema in the posterior talus (Fig. 2.13) or within the OT and/or a prominent posterior calcaneal process or a downward slope to the posterior tibia. It can also be associated with traumatic osteochondral defects of the talus (Fig. 2.14, 2.15). If necessary, the diagnosis can be confirmed by injecting 0.5 ml of a local anesthetic into the posterior soft tissues behind the peroneal tendons. It is important to keep in mind that most people who have an OT are asymptomatic and that posterolateral impingement syndrome is uncommon in most athletes. Unfortunately, due to the often dramatic appearance of the OT on x-ray, the condition is frequently overdiagnosed by practitioners, who may then recommend surgery unnecessarily.

Treatment of posterolateral impingement syndrome begins with conservative management options such as activity modification, avoiding exacerbating plantarflexed ankle positions, nonsteroidal antiinflammatory drugs (NSAIDs), and physical therapy. In addition, an injection of 0.25 to 0.5 ml of a mixture of a long-acting and a short-acting corticosteroid may provide significant relief of symptoms, and the accuracy of the injection can be improved with sonography.

Although uncommon, if nonoperative treatment has failed and the OT remains symptomatic, surgical excision becomes indicated. If the pathology is isolated to a symptomatic OT with no medial symptomatology, it can be approached



Fig. 2.12 Posterior impingement on the os trigonum.

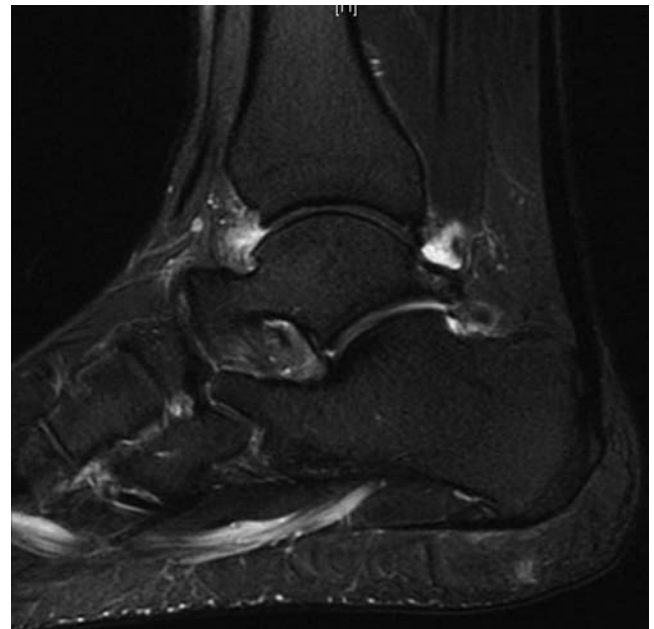


Fig. 2.13 Edema OT.

posterolaterally in the interval between the FHL and the peroneal tendons while protecting the sural nerve. However, posterolateral impingement is often associated with FHL tendinitis (Video 2.1 “Os Trigonum and FHL”), and in this case,

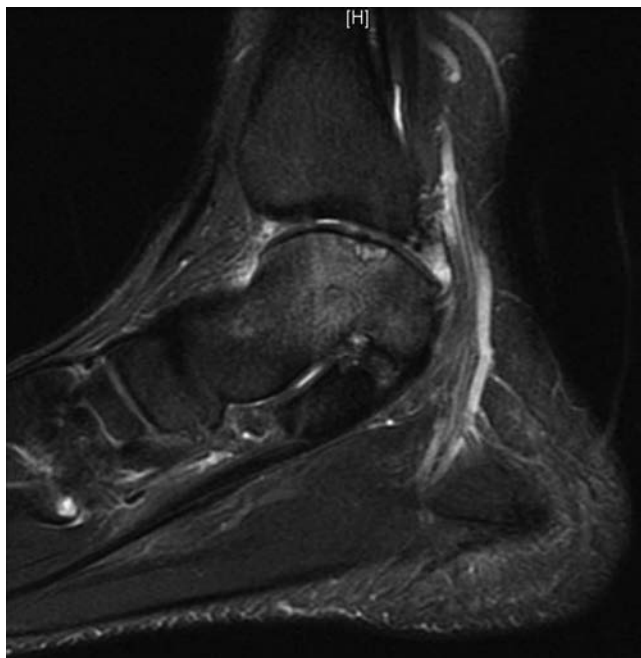


Fig. 2.14 MRI osteochondral defect (OCD) sag.

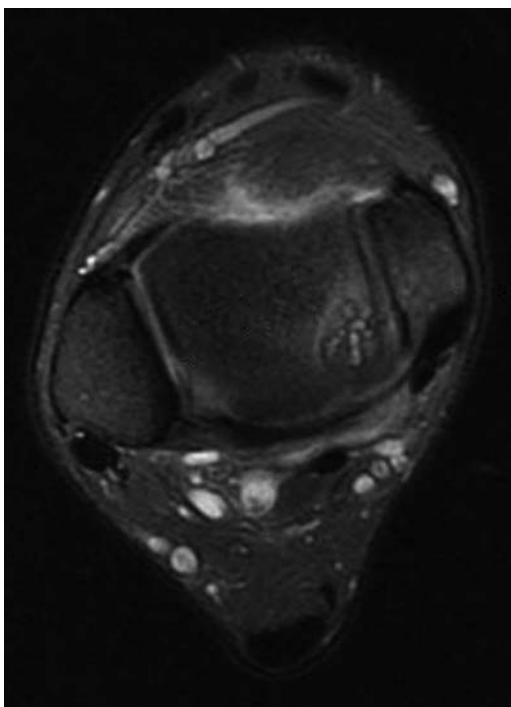


Fig. 2.15 MRI sag axial.

the posteromedial approach is utilized in order to adequately develop and protect the neurovascular bundle and perform a tenolysis of the FHL after excising the adjacent OT. Results of OT resection using a posterolateral approach have shown significant improvement in APFAS scores, with a complication rate of approximately 20% including sural nerve injury, superficial wound infection, and reflex sympathetic dystrophy.³⁷ Ankle arthroscopy using two posterior portals can also provide excellent visualization of the posterior structures of the ankle and good access to the OT, loose bodies, and FHL tendon sheath.³⁸

Posterolateral impingement can also occur following an ankle sprain where the lateral ligamentous structures are disrupted. The resulting instability causes the talus to slide forward and the posterior lip of the tibia to come to rest on the os calcis. In this case, the treatment is to correct the anterior drawer by reconstructing the lateral ligamentous complex (ex: Brostrum-Gould).^{34,39}

A posterior pseudomeniscus or plica in the posterior ankle, with or without an OT, can cause posterior impingement syndrome in the absence of an OT or ligament laxity. Bucket-handle like tears in this structure can cause locking and other mechanical symptoms similar to what is typically seen in the knee.³⁴

The differential diagnosis of posterolateral ankle pain can also include acute trauma including fractures of the posterior process of the talus, avulsions of the PTF, fracture of the trigonal process also known as a Shepherd's fracture or of the os trigonium synchondrosis.

Posteromedial Ankle Impingement

Bony impingement does not typically occur in the posteromedial ankle as the tibia and talus do not come together. Therefore, posteromedial impingement is mainly due to soft tissue causes. The posteromedial flexor tendons, joint capsule, and tibiotalar ligament are the main contributors of posteromedial impingement. The FHL passes through a fibro-osseous tunnel behind the talus between the medial and lateral tubercles to the level of the sustentaculum tali like a rope through a pulley. As it passes through this pulley, it is easily strained. When this occurs, rather than moving smoothly in the pulley, it begins to bind. This binding causes irritation and swelling. Chronic inflammation and hypertrophy of the musculotendinous unit within this tunnel can lead to a painful stenosing tenosynovitis, analogous to de Quervain disease in the wrist. Tendinitis of the FHL tendon behind the medial malleolus of the ankle is also known as dancer's tendonitis but can also occur in other athletes as well. If a nodule or partial tear is present, triggering of the big toe may occur; this is known as hallux saltans (Fig 2.16). In extreme cases, the tendon may become completely frozen in the sheath, causing pseudo hallux rigidus. Physical exam findings include localized tenderness and swelling over the FHL sheath behind and lateral to the medial malleolus. Palpation of the sheath with active and passive motion of the hallux will mimic the patient's symptoms. Dorsiflexion of the first metatarsophalangeal (MP) joint can be reduced or absent when the ankle is in maximum dorsiflexion and the muscle fibers of the FHL are drawn into the FHL tunnel, producing a functional hallux rigidus (Thomassen's sign). This finding is not always pathological, as it may be present in asymptomatic patients. Usually there is no pain with forced plantarflexion of the ankle. FHL tendinitis is often misdiagnosed as posterior tibial or Achilles tendonitis, and it typically responds to the usual conservative measures such as rest (no pointe work), activity modification, and NSAIDs. Steroid injections should be avoided in this location. In refractory cases that have failed conservative management, surgical release of the fibro-osseous is indicated.

FHL tendinitis usually occurs behind the medial malleolus, but occasionally it can be isolated to the knot of Henry where the flexor digitorum longus (FDL) crosses over the FHL, and

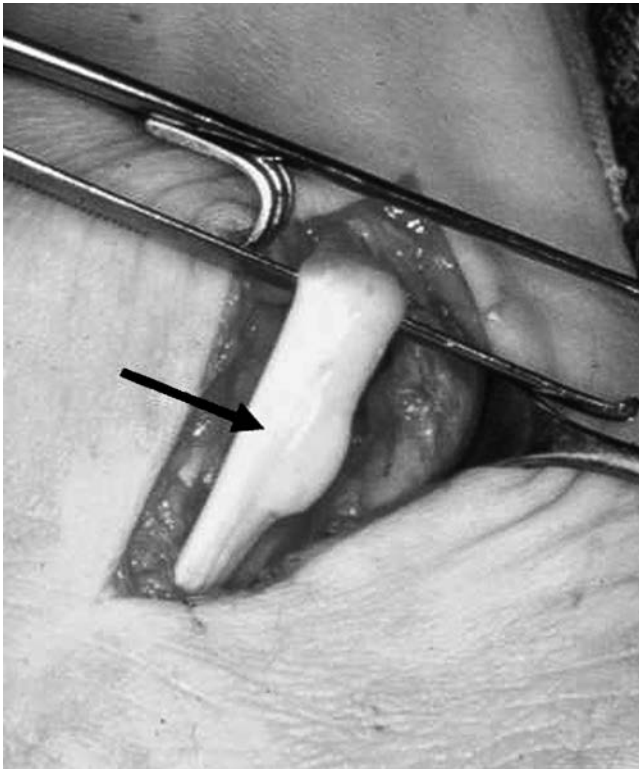


Fig. 2.16 A nodule in the flexor hallucis longus (FHL) tendon causing triggering of the great toe; “hallux saltans.”

under the head of the first metatarsal where it passes between the sesamoids. A fibrous subtalar coalition may be present in the posteromedial ankle, mimicking FHL tendinitis or tarsal tunnel syndrome. This condition should be suspected when limited subtalar motion is seen on physical examination.

In the management of posteromedial ankle impingement, operative treatment is indicated when conservative therapy has failed. A posterior-based approach can be made on either the medial or lateral side of the Achilles tendon. The lateral approach should be utilized if the patient has isolated posterior impingement without a history of FHL tendinitis or medial-based pathology. However, if the patient presents with posterior impingement and FHL tendinitis with or without an incidental OT, a medial incision should be performed. The medial incision is preferred as it avoids the sural nerve and provides access to the lateral structures while allowing direct access to the posteromedial neurovascular bundle.

Open Medial Approach—FHL Tenolysis and Excision Os Trigonum

This procedure can be performed with the patient prone or supine with a bump under the contralateral hip in order to provide external rotation of the hip and knee. A well-padded tourniquet is placed around the thigh. A curvilinear incision is made following the neurovascular bundle beginning just above the superior border of the os calcis and ending just posterior to the tip of the medial malleolus (Fig. 2.17A). Blunt dissection is performed and the deep fascia is then divided carefully to avoid damage to the posteromedial neurovascular bundle. The tibial motor branches to the os calcis are more variable in

the posterior aspect of the neurovascular bundle, therefore the interval between the medial malleolus and the anterior aspect of the neurovascular bundle should be developed at this point. The bundle can then be taken down off the malleolus by blunt dissection. In this region, there are several small vessels that will need to be ligated and the bundle can then be mobilized and held with a blunt retractor. The surgeon should examine the tibial nerve and artery and note the location where they divide into medial and lateral plantar branches as they leave the tarsal canal. It is not unusual for either the artery or the nerve, or both, to divide above this area, leading to reduplication within the tunnel. There also may be reduplication of the FHL tendon. With the neurovascular bundle retracted posteriorly, the FHL is identified by moving the hallux. The thin fascia overlying the FHL is opened proximally and tenolysed from proximal to distal. Usually the fascial sheath is stenotic and rigid, and the FHL can be seen entering at an acute angle. Care should be taken distally, as the FHL tunnel and the nerve are in close proximity. As the tendon approaches the sustentaculum tali, the fascial sheath becomes exceedingly thin. At this point, the tendon should be inspected for nodules or longitudinal tears and, if such are present, should be carefully debrided or repaired. Following this, the FHL and neurovascular bundle can be retracted posteriorly allowing access to the OT or trigonal process on the lateral side of the FHL tunnel. If the posterior aspect of the talus cannot be visualized, a capsulotomy should be performed. If there is difficulty in visualizing the OT, it helps to identify the superior border of the os calcis and the subtalar joint (by moving the os calcis into adduction and abduction). The subtalar joint is then dissected from medial to lateral, and this will take the surgeon underneath the OT. Once identified, it can be removed by circumferential dissection. Care should be taken to stay on the bone when performing this part of the procedure. This can be somewhat difficult, especially if the OT is large. Once it is removed, the posterior ankle joint should be inspected for bone fragments, loose bodies, soft tissue entrapment, or the large articular facet on the upper surface of the os calcis that often articulates with the OT. If this articulation is large, it may need to be removed with a thin osteotome. The FHL sheath should not be closed. The wound is then irrigated and the ankle is put in plantarflexion to check for any residual impingement. The wound is closed in layers with the ankle in neutral flexion. Full weight bearing with crutches can begin immediately with swimming and physical therapy started at approximately two weeks once the wound has healed. Ankle range of motion is started as early as possible to prevent residual stiffness. If only the tenolysis is performed without excision of the OT, the recovery period is approximately 6 weeks. When the tenolysis is performed with OT excision, the recovery time is approximately 8 to 12 weeks.

Posterior Ankle and Hindfoot Arthroscopy^{38,40,41}

This is our preferred technique to deal with posterior ankle impingement and pathology. The patient is positioned prone and a well-padded tourniquet is placed around the thigh. Although used infrequently, ankle distraction is achieved with a tensioned wired can be placed transversely in the calcaneus, the operative knee is cephalad to the break in the operating

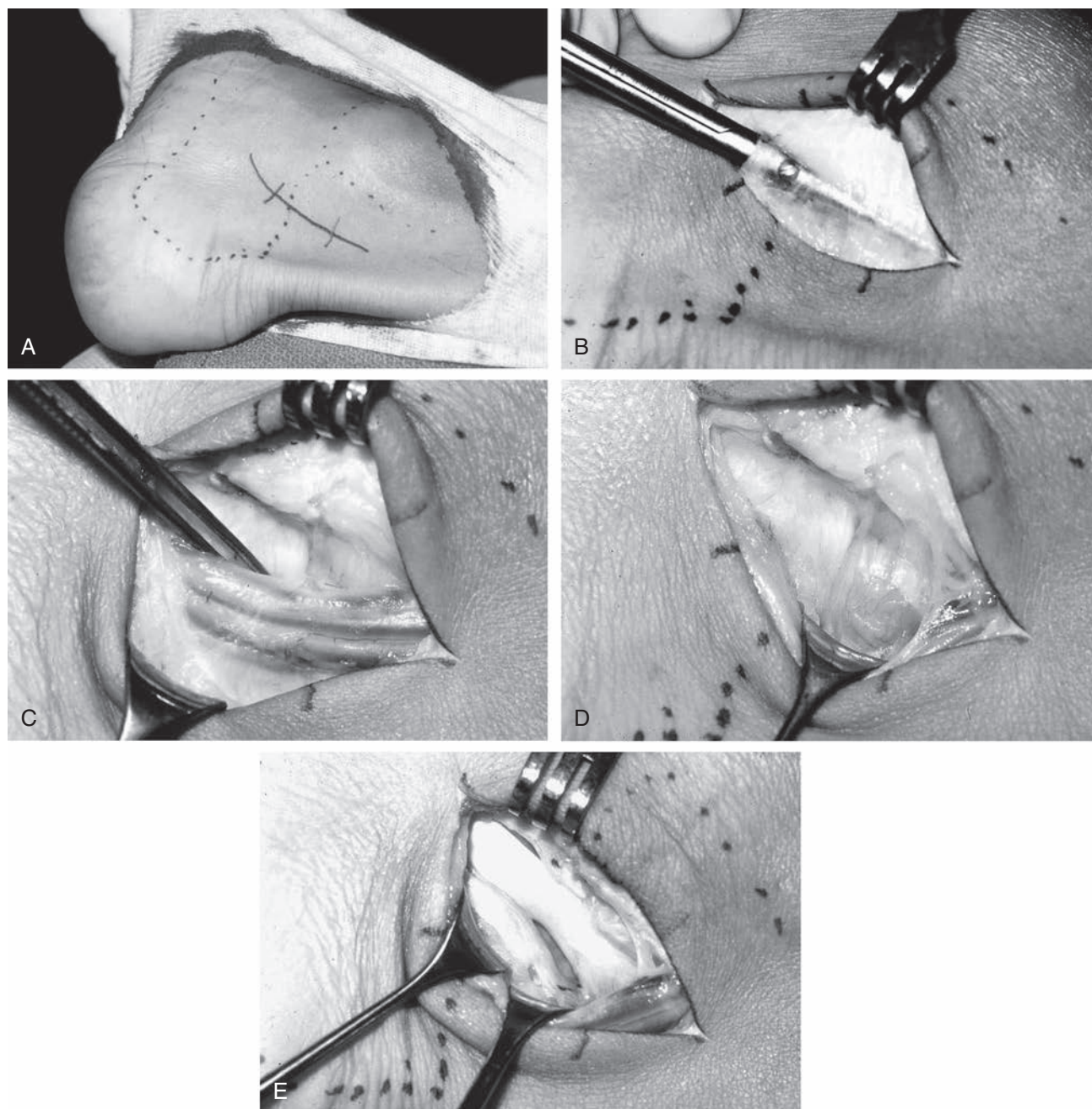


Fig. 2.17 (A) Posteromedial incision. (B) Neurovascular bundle beneath a thin layer of fascia. (C) Neurovascular bundle taken down from the posterior medial malleolus. (D) Posterior tibial nerve protected with a blunt retractor. Underneath lies the flexor hallucis longus (FHL) sheath. (E) FHL sheath opened.

table and the contralateral knee is bent to 90 degrees and secured to a padded post. The foot of the operating table is then lowered allowing access for the mini c-arm. In cases where distraction is not applied, the operative leg is elevated by means of two sterile towel bumps. For intra-articular posterior ankle procedures, the posterolateral portal is first made lateral to the Achilles tendon at the level of the tip of the lateral malleolus (Fig. 2.18). A shallow skin incision is made and blunt dissection is then performed with a straight clamp to avoid injuring the sural nerve. Using fluoroscopic guidance, a blunt trocar is advanced in line with the first web space and toward the posterior process of the talus. Once inside the joint, the trocar is

exchanged for a 4.0 mm or 2.7 mm 30 degree arthroscope. At the same level using a similar technique, the working portal is made just medial to the Achilles tendon. In this case, the blunt trocar is advanced in line with the third web space and aimed medial to the midline.

To gain access to the hindfoot or for extra-articular procedures, blunt dissection is performed through the medial portal in line with the third web space toward the posterior process of the talus. Once the clamp is felt to be resting on bone, it can be exchanged for a 4.0 mm arthroscope, which is directed laterally. A mosquito is then advanced from the lateral portal dissecting toward the tip of the arthroscope. Once



Fig. 2.18 Dissection with the shaver.



Fig. 2.19 Os Trigonum and flexor hallucis longus (FHL) adjacent.

the mosquito can be seen in the arthroscopic field, it can be exchanged for a 3.5-mm shaver. At this point, careful dissection with the shaver is performed to remove adipose tissue and expand the viewing field (Fig 2.18). Careful debridement can be continued from lateral to medial exposing the posterior tibiofibular ligament, the posterior tibiotalar and subtalar joint capsule, the os trigonum, and finally the flexor hallucis longus tendon (Fig 2.19). Once the impinging lesion or OT is identified, the debridement is made as close to bone as possible (Video 2.2 “Os Trigonum”). The position of the FHL should be confirmed throughout the debridement by moving the great toe. Once the OT has been circumferentially debrided, it can be broken down with a 4-mm burr or detached from the posterior process of the talus and retrieved en bloc with arthroscopic grasper. The adequacy of resection can be confirmed by noting the position of the FHL and by direct observation while plantarflexing the ankle. If an FHL

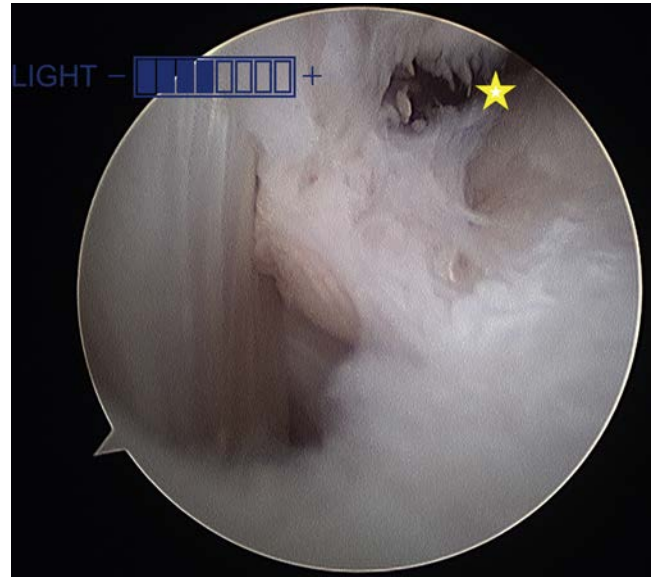


Fig. 2.20 Post ankle scope.

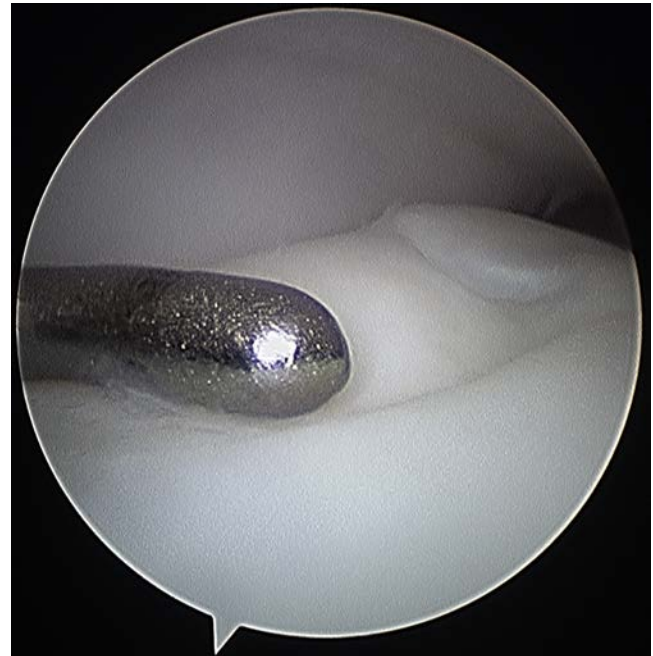


Fig. 2.21 OCD larger posterior approach.

tenolysis is to be performed, the sheath can be released to the level of the sustentaculum staying in the lateral side of the tendon. If any associated osteochondral lesions are present, they can be visualized, debrided (Figs 2.20–2.22), and treated (i.e., microfracture) at this point. Prior to closing, 20 cc of 0.5% Marcaine with epinephrine is injected into each portal and the portals are loosely closed with 3-0 monocryl suture. Postoperatively, patients are placed in a well-padded dressing. Protected weight bearing and range-of-motion exercises are started immediately. At 4–6 weeks, physical therapy should focus on strengthening and proprioceptive exercise, and the patient is permitted to return to sport when full range of motion and strength have returned.

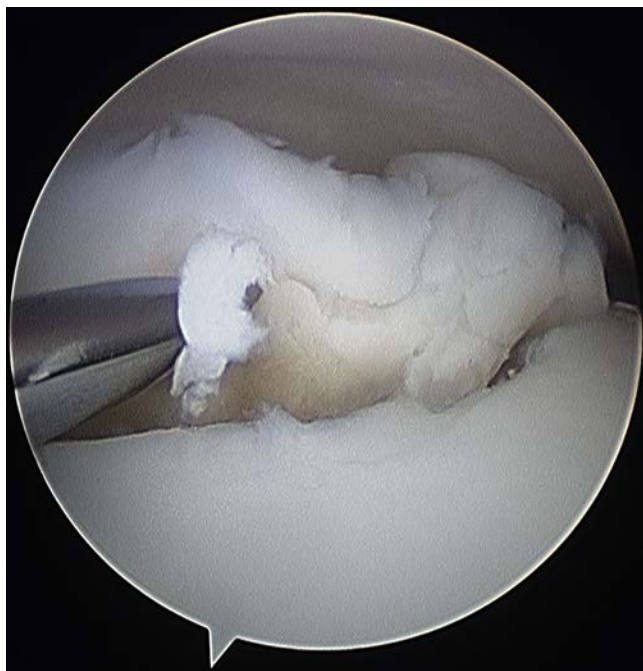


Fig. 2.22 OCD larger.

A retrospective study that looked at the results of posterior ankle and hindfoot arthroscopy in 189 ankles found a low complication rate of 8.5%, which included plantar skin numbness, sural nerve injuries, Achilles tendon contracture, complex regional pain syndrome, infection, and superficial synovial portal cyst.⁴⁰ A separate study found the average time to return to sport was 5.8 months, and 14 of 15 patients were able to return to pre-injury level of play.³⁸ A study by Carreira et al., looking at the results of posterior arthroscopy for impingement, found an increase in American Orthopaedic Foot & Ankle society (AOFAS) score from 75 preoperatively to 94.9 postoperatively, a decrease of visual analog scale (VAS) from 5.4 to 0.85, equal range of motion to the contralateral ankle at 1 year, and all patients were able to return to their pre-injury level of play.⁴²

Medial Ankle Impingement

Medial sprains of the ankle are rare because the medial bony and ligamentous structures are strong and rigid in comparison with the lateral side of the ankle. In the athletic patient who presents with medial ankle pain, posterior tibial tendonitis is the most common cause. When dancers present with medial ankle pain, this is often caused by repetitive pronation of the ankle known as “rolling in,” which causes a chronic strain of the deltoid ligament. Scar tissue forms along the joint line and becomes trapped between the posterior talus and medial malleolus, causing pain. This type of impingement is often associated with lateral ligamentous instability and if unidentified, the patient will continue to be symptomatic following lateral ligamentous reconstruction.^{43,44}

This presentation must also be distinguished from posteromedial ankle pain in dancers, which is usually caused by FHL tendonitis. Medial ankle sprains usually occur from landing off balance with sudden pronation. The part of the ligament that is injured depends on which portion was under tension when the

force was applied. With the foot in equinus, the anterior deltoid will be most affected; with the foot plantigrade, it will be the middle deltoid; and although rare, the posterior portion of the deltoid is most commonly affected when the foot is in a dorsiflexed position. Following a sprain, medial osteophytes can then form and cause bony impingement. Persistent symptoms on the medial side may also be due to an unrecognized fracture of the sustentaculum tali, which can often be seen on a bone scan, or can be due to a fibrous tarsal coalition. An accessory bone, the os subtibiale, may be present in the deep layer of the deltoid and can become symptomatic following a sprain. An x-ray should be taken to rule out bony pathology, including a physeal injury in the appropriate age group. In the acute phase, treatment consists of RICE (rest, ice, compression, and elevation), an aircast stirrup brace, and crutches if necessary, as well as physical therapy. Recovery usually is uneventful. Occasionally, persistent pain over the deltoid ligament is reported, and this is usually from an avulsion fracture or accessory ossicle. These often respond to conservative therapy, and only rarely is surgical excision necessary.

Sinus Tarsi Impingement

Sinus tarsi syndrome is a controversial topic and an uncommon source of ankle impingement, which was first described by Denis O'Connor.⁴⁵ It is an impingement condition affecting the subtalar joint that produces lateral foot and ankle pain deep in the sinus tarsi, which is exacerbated by impact (jumping and running) activities as well as pronation. Patients will present with discrete lateral subtalar pain located within the sinus tarsi, which will often, but not always, be preceded by an ankle sprain. On physical examination, pain is located deep in the sinus tarsi, and can be exacerbated with forceful abduction-pronation of the heel and midfoot. The diagnosis can usually be confirmed with an injection of 0.5 ml of lidocaine into the sinus tarsi. If the pain is relieved by the local anesthetic, a second injection of 0.15 ml of corticosteroid is often effective. Other diagnostic and imaging techniques used to assess for sinus tarsi syndrome have included arthrography, MRI, and more recently SPECT/CT.^{46–48} Although there is currently no clear etiology underlying sinus tarsi syndrome, several theories have been proposed such as soft tissue entrapment or partial tear and scarring of the interosseous talocalcaneal ligament (ITCL), synovial inflammation and impingement, osteophyte impingement (Fig. 2.23), neural entrapment (motor nerve to the extensor digitorum brevis [EDB]), sensory nerve traction injury, arthrofibrosis, and degenerative arthrosis.^{46,47}

Due to their anatomical proximity, it can be difficult to differentiate sinus tarsi syndrome from general subtalar dysfunction. Subtalar osteophytes incidentally found on imaging are not uncommon,⁴⁹ and changes in subtalar motion on physical exam can be an effective method to differentiate the two conditions.

First-line management includes nonoperative treatment consisting of antiinflammatory medication, physical therapy, orthotic support such as a medial heel wedge or arch support that functions to open the sinus tarsi, and local injections. If the diagnosis is clear and symptoms persist despite nonoperative modalities, surgical management is indicated. Arthroscopic



Fig. 2.23 Sinus tarsi syndrome; note osteophytes (arrow).

evaluation and debridement of the subtalar joint and sinus tarsi has shown conflicting results, with some authors showing a high incidence of associated conditions suggesting an alternative diagnosis⁵⁰ while others have shown clear improvements in pain and dysfunction.⁵¹ In patients presenting with sinus tarsi syndrome in conjunction with lateral ankle instability, sinus tarsi exploration and debridement should be considered and, if warranted, performed at the same time as the lateral ligamentous reconstruction.

CONCLUSION

Ankle impingement can be caused by both bony or soft tissue pathologies. In either case, the end result is joint swelling, stiffness, pain, and eventual dysfunction. All quadrants of the ankle joint can be affected by impingement, but the underlying cause will differ due to the anatomy and mechanics of the ankle. Nonoperative treatment consisting of activity modification, physical therapy, retraining, and various shoe wear modifications is often successful. Operative treatment should be reserved for cases that have failed nonoperative management. The surgical goals should be the complete removal of offending osteophytes in order to restore a functional joint space, prevent ongoing impingement, minimize recurrence, and return of the athlete to a pre-injury level of play.

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Video Legends - <https://www.kollaborate.tv/link?id=5c9d1e192b4c3>

▶ **Video 2.1** Title: Posterior ankle arthroscopy for evaluation of Flexor Hallucis Longus tendon and Os trigonum Legend: Posterior ankle arthroscopy video demonstrating tear in FHL tendon and loose Os trigonum.

▶ **Video 2.2** Title: Posterior ankle arthroscopy for resection of posterior process talus Legend: Posterior ankle arthroscopy video depicting resection of posterior process talus and arthroscopic evaluation of posterior ankle and posterior subtalar joint.

Stress Fractures: Their Causes and Principles of Treatment

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INTRODUCTION

Historical Perspective

Stress fractures were first described in 1855 by Briethaupt, a Prussian military physician who observed foot pain and swelling in young military recruits unaccustomed to the rigors of training. He considered it to be an inflammatory reaction in the tendon sheaths resulting from trauma and called the condition *Fussgeschwulst*. It was not until the advent of radiographs that the signs and symptoms were attributed to fractures in the metatarsals.¹ The condition then became known as a march fracture because of the close association between marching and the onset of symptoms. Stress fractures were first noticed in civilians in 1921 by Deutschlander,² who reported six cases in women. However, it was not until 1956, more than a century following their identification in military recruits, that they were recognized in athletes.³

A variety of terms have been used over time to describe stress fractures. These include march fractures, Deutschlander fractures, fatigue fractures, or crack fractures.² Virtually all of these terms have been intended to describe some etiologic attribute of the stress injuries of bone. In recent years the most commonly used term has been stress fracture.

Following the radiographic description of metatarsal stress fractures, many theories were set forth to explain the etiology of this injury. Most of the reports were based on series that were small, and the theories proposed were concerned with either mechanical factors, such as spasm of the interossei, or flat feet, or with inflammatory reactions, such as nonsuppurative osteomyelitis.

ETIOLOGY OF STRESS FRACTURES

It is now recognized that the development of a stress fracture represents the end product of the failure of bone to adapt adequately to the mechanical loads experienced during

physical activity, typically that which is repetitive. Ground reaction forces and muscular contraction result in bone strain. It is these repetitive strains that are thought to cause a stress fracture. Bone normally responds to strain by increasing the rate of remodeling. In this process, lamellar bone is resorbed by osteoclasts, thereby creating resorption cavities that subsequently are replaced with more dense bone by osteoblasts. Because there is a lag between increased activity of the osteoclasts and osteoblasts, bone is weakened during this time. If sufficient recovery time is allowed, bone mass eventually increases. However, if loading continues, microdamage may accumulate at the weakened region. Remodeling is thought to repair normally occurring microdamage. The processes of microdamage accumulation and bone remodeling, both resulting from bone strain, play an important part in the development of a stress fracture. Running, for example, produces ground reaction forces approximately five times greater than walking. The result of excess strain is an accumulation of microdamage leading to fatigue reaction or fatigue failure.

If microdamage accumulates, repetitive loading continues, and remodeling cannot maintain the integrity of the bone; a stress fracture may result. This may occur because the microdamage is too extensive to be repaired by normal remodeling, because depressed remodeling processes cannot adequately repair normally occurring microdamage, or because of some combination of these factors. Another special consideration in the pathophysiology of stress fractures is the influence of skeletal muscle. For example, muscles may protect the tibia during activity by producing shear forces that counteract the joint reaction forces and result in reduced net shear stresses in the lower extremity.^{4,5} It has been hypothesized that reduced lower leg muscle strength increases the risk of stress fracture through this mechanism. There is another theory that brings in the possibility of repeated oxygen deprivation during activity. In this theory, the repeated load of an activity such as running

TABLE 3.1 Distribution of Multiple Stress Fractures According to Anatomic Location in Both Sexes

Location	WOMEN		MEN		TOTAL	
	N	%	N	%	N	%
Metatarsal	15	34.1	13	18.6	28	24.6
Calcaneus	2	4.5	0	0.0	2	1.8
Tarsal	5	11.4	3	4.3	8	7.0
Tibia						
Distal third	7	15.9	13	18.6	20	17.5
Middle third	4	9.1	11	15.7	15	13.2
Proximal third	4	9.1	16	22.9	20	17.5
Fibula						
Distal third	2	4.5	5	7.1	7	6.1
Middle third	1	2.3	3	4.3	4	3.5
Proximal third	0	0.0	1	1.4	1	0.9
Femur						
Distal third	1	2.3	0	0.0	1	0.9
Middle third	0	0.0	0	0.0	0	0.0
Proximal third	1	2.3	3	4.3	4	3.5
Pubic bones	2	4.5	2	2.9	4	3.5
Total	44	100.0	70	100.0	114	100.0

From Korpelainen R, Orava S, Karpakka J et al. Risk factors for recurrent stress fractures in athletes. *J Sports Med.* 2001; 29(3):304-310.

is thought to cause decreased oxygen delivery and brief ischemia in weight-bearing bones. This ischemic environment is thought to stimulate the bone-remodeling process, specifically by increasing osteoclastogenesis.⁶ The end result is a weakened bone that is susceptible to stress fractures.

EPIDEMIOLOGY

Stress fractures have been reported to occur in association with a variety of sports and physical activities. Clinical impression suggests that stress fractures are more common in weight-bearing activities, particularly those with a running or jumping component. Numerous case series have reported that stress fractures comprise between 0.7% and 15.6% of all injuries sustained by athletic populations.⁷

It often is suggested that women sustain a disproportionately higher number of stress fractures than men.⁸ A gender difference in stress fracture rates is, however, not as evident in athletic populations.⁹ Studies either show no difference between male and female athletes or a slightly increased risk for women, up to 3.5 times that of men. Bennell et al. found no significant difference between gender incidence rates even when expressed in terms of exposure.¹⁰ Women sustained 0.86 stress fractures per 1000 training hours, compared with 0.54 in men. Reported increased incidence of stress fractures in women has been attributed to decreased bone density/size, hormonal aberrations, and biomechanical/anatomical differences.¹¹⁻¹⁴ Korpelainen and colleagues looked into risk factors for recurrent stress fractures in

athletes and found runners with a high weekly training mileage are at a high risk of recurrent stress fractures of the foot and shin. Leg-length inequality, a high longitudinal arch of the foot, forefoot varus, and menstrual irregularities may also be etiologic factors for recurrent stress fractures (Tables 3.1 and 3.2).¹⁵

RISK FACTORS FOR STRESS FRACTURES

Repetitive mechanical loading arising from athletic training contributes to stress fracture development. However, the contribution of each training component (volume, intensity, frequency, surface, and footwear) to the risk of stress fracture has not been elucidated. Training also may influence bone indirectly through changes in levels of circulating hormones, through effects on soft tissue composition, and through associations with menstrual disturbances.¹⁶

Training surface has long been hypothesized to contribute to stress fracture development. Anatomic and biomechanical problems can be accentuated by cambered or uneven surfaces, whereas ground reaction forces are increased by less compliant surfaces. Researchers also have implicated training surface or change in surface as a risk factor but do not provide substantial evidence in support.

Biomechanical abnormalities may predispose athletes and active individuals to stress fractures by creating areas of stress concentration in bone or by promoting muscle fatigue. Both excessively supinated and excessively pronated feet can be contributing factors in the development of stress fractures. Pes

TABLE 3.2 Distribution of Multiple Stress Fractures by Sports Event According to Anatomic Location

Bone	SPORTS EVENT							TOTAL	
	Long-Distance Running	Sprinting	Jumping	Orienteering	Skiing	Power Events	Ball Games	N	%
Metatarsal	20	3	2	0	0	1	2	28	24.6
Calcaneus	2	0	0	0	0	0	0	2	1.8
Tarsal	5	2	1	0	0	0	0	8	7.0
Tibia									
Distal third	11	1	0	0	3	2	3	20	17.5
Middle third	7	1	4	0	0	0	3	15	13.2
Proximal third	12	0	2	6	0	0	0	20	17.5
Fibula									
Distal third	5	1	0	1	0	0	0	7	6.1
Middle third	2	0	1	0	0	0	1	4	3.5
Proximal third	1	0	0	0	0	0	0	1	0.9
Femur									
Distal third	1	0	0	0	0	0	0	1	0.9
Middle third	0	0	0	0	0	0	0	0	0.0
Proximal third	4	0	0	0	0	0	0	4	3.5
Pubic bones	3	0	0	0	0	0	1	4	3.5
Total	73	8	10	7	3	3	10	114	100.0

From Korpelain R, Orava S, Karpakka J et al. Risk factors for recurrent stress fractures in athletes. *J Sports Med.* 2001, 29(3):304-310.

TABLE 3.3 Grouping of Intrinsic and Extrinsic Factors

Intrinsic Factors	Extrinsic Factors
Cavus feet	Type of activity
Leg length discrepancies	Excessive/new training regimen
Excessive forefoot varus	Poor equipment/footwear
Tarsal coalitions	Improper technique
Prominent posterior calcaneal process	Type of training surface
Tight heel cords	Sleep deprivation
Osteopenia/osteoporosis	
Poor vascular supply	
Abnormal hormonal levels	

From Parekh et al. Stress fractures of the foot and ankle in athletes. *FAJ.* 2013, 6(6):481-491.

planus (pronated) was initially implicated as a common foot type in athletes who presented to sports clinics with stress fractures; however, athletes with pes planus that did not sustain an injury were not assessed.^{17,18} Therefore, this cannot be solely implicated as a risk. Parekh and colleagues developed a chart representing intrinsic and extrinsic factors that can be used to rule out other causes (Table 3.3).¹⁹ A prospective study that examined a number of clinical biomechanical measurements in athletes, including range of hip rotation and ankle dorsiflexion, calf and hamstring flexibility, lower limb alignment, and static foot posture, did not find any to be useful predictors of stress fracture occurrence.⁹

Dietary behaviors and eating patterns may differ in those with stress fractures. Low calcium intake may contribute to stress fracture development by directly influencing the processes of bone remodeling and bone mineralization or by indirectly affecting soft tissue composition and ovarian function. Other dietary factors, such as fiber, protein, and caffeine intake, may play a role. Scores on a validated test relating to dieting, bulimia and food preoccupation, and oral control (EAT-26) did not differ between ballet dancers or track and field athletes with and without stress fracture.⁹

Theoretically, low bone mineral density (BMD) could contribute to the development of a stress fracture by decreasing the fatigue resistance of bone to loading and by increasing the accumulation of microdamage. The findings of a 12-month prospective study using dual energy x-ray absorptiometry (DEXA) to measure bone mass indicate that low bone density is a risk factor for stress fractures in women and possibly in men.⁹ Female athletes who sustained tibial stress fractures had 8.1% lower tibia/fibula BMD than athletes without stress fractures ($p < 0.01$). In the men, the tibial stress fracture group had 4.0% less tibia/fibula BMD than the non-stress fracture group, although this was not significant ($p = .17$). However, it is important to note that in this study the athletes with stress fractures still had bone density levels that were similar to or greater than less active control subjects. This implies that the level of bone density required by athletes for short-term bone health is greater than that required by the general population.⁹

EVALUATION

A detailed history and physical exam of the patient with a stress fracture is essential for an accurate and timely diagnosis. In addition to obtaining a history of the patient's pain and its relation to exercise, it is important to determine the presence of predisposing factors. In particular, note should be taken of recent changes in activity level, such as increased quantity of training, increased intensity of training, and changes in surface, footwear, and technique. A full dietary history should be taken; particular attention should be paid to the possible presence of eating disorders. In females, a menstrual history should be taken as well.

Typically, the pain is one of insidious onset of activity-related pain. Usually the pain will be described initially as a mild ache occurring after a specific amount of exercise. If the patient continues to exercise, the pain may well become more severe or occur at an earlier stage of exercise. The pain eventually may increase to the point that it limits the quality or quantity of the exercise performed or, occasionally, forces cessation of all activity. In the early stages, pain usually will cease soon after exercise is terminated. However, with continued exercise and increased severity of symptoms, the pain may persist after exercise cessation. Night pain occasionally may occur.

On physical examination the most obvious feature is localized bony tenderness. Stress fractures typically have a more point-specific tenderness, and in comparison, stress reactions will have a more generalized region of tenderness. The physical examination also must take into account the potential predisposing factors; and in all stress fractures involving the lower limb, a full biomechanical examination must be performed. Any evidence of leg-length discrepancy, malalignment (especially excessive subtalar pronation), muscle imbalance, weakness, or lack of flexibility should be noted.

DIAGNOSTIC STUDIES

Radiography has poor sensitivity but high specificity in the diagnosis of stress fractures. The classic radiographic abnormalities seen in a stress fracture are new periosteal bone formation, a visible area of sclerosis, the presence of callus, or a visible fracture line. Unfortunately, in the majority of stress fractures there is no obvious radiographic abnormality. The abnormalities on radiography are unlikely to be seen unless symptoms have been present for at least 2 to 3 weeks. In certain cases, they may not become evident for up to 3 months, and in a percentage of cases never become abnormal.

If plain radiography demonstrates the presence of a stress fracture, then there seldom is any need to perform further investigations. However, in cases in which there is a high index of suspicion of stress fracture, magnetic resonance imaging (MRI) is the recommended advanced imaging study to be used. MRI visualizes marrow hemorrhage and edema well, a characteristically difficult finding with the other advanced imaging computerized tomography (CT).^{20,21} Although CT scan visualizes bone detail, MR imaging does better at distinguishing stress fractures

from a suspected bone tumor or infectious process.²² CT scanning will enable the clinician to differentiate between a stress fracture, which will be visible on CT scan, and a stress reaction. Particularly in the elite athlete, this may help in the rehabilitation program and forthcoming competition program.²³

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of stress fracture can be divided into nonbony or bony causes. Nonbony causes, in particular, relate to muscle or tendon injury; either muscle strain, hematoma or delayed-onset muscle soreness, or tendon inflammation or degenerative change.

Bony pathologies that can mimic stress fracture include tumor and infection. Osteoid osteoma commonly is mistaken for a stress fracture because it presents with pain and a discrete focal area of increased uptake on isotope bone scan. Two distinguishing features of osteoid osteoma are the presence of night pain and the relief of pain with the use of aspirin.²⁴ In addition, a CT scan or MRI can clearly distinguish the nidus of an osteoid osteoma from the cortical abnormality of a stress fracture.

TREATMENT

The basis of treatment of stress fractures involves rest from the aggravating activity, a concept known as relative rest. The amount of time from a diagnosis of a stress fracture to full return to sport depends on a number of factors, including the site of the fracture, the length of the symptoms, and the stage in the spectrum of bone strain. Most stress fractures with a relatively brief history of symptoms will heal in a straightforward manner, and return to sport should occur within 6 to 8 weeks. However, there is a group of stress fractures that require additional treatment to relative rest, and these are considered later.

The primary aim of initial management of stress fracture is pain relief. This may involve the use of mild analgesics or nonsteroidal antiinflammatory drugs (NSAIDs). In some cases, in which activities of daily living are painful, it may be necessary for the patient with a stress fracture to be non-weight bearing or partial weight bearing on crutches for a period of up to 7 to 10 days.²⁵ In the majority of cases this is not necessary, and mere avoidance of the aggravating activity will be sufficient.

When activities of daily living are pain free and there is no focal tenderness, then resumption of the aggravating activity can occur on a graduated basis. Progress should be monitored clinically by the presence or absence of symptoms and local signs. It usually is not necessary to monitor progress by radiography, scintigraphy, CT, or MRI. Radiologic healing often lags behind clinical healing.

It is important that the athlete with a stress fracture be able to maintain strength and cardiovascular fitness while undergoing the appropriate rehabilitation program. The most common ways are biking, swimming, water running, and using upper body weights. These workouts should mimic the athlete's normal training program as much as possible in both duration and

intensity. Water running is particularly attractive to runners for this reason. Water running involves the use of a buoyancy vest as a flotation device. Stretching should be performed to maintain flexibility during the rehabilitation process. Muscle strengthening also is an important component of the rehabilitation phase. In addition to maintaining these parameters of physiologic fitness, it is possible in most cases for the athlete to maintain specific sports skills. In ball sports these can involve activities either seated or standing still. This active rest approach also greatly assists the athlete psychologically.

As with any overuse injury, it is not sufficient merely to treat the stress fracture itself. An essential component of the management of an athlete with an overuse injury involves identification of the factors that have contributed to the injury and, when possible, correction or modification of some of these factors to reduce the risk of the injury recurring. The fact that stress fractures have a high rate of recurrence is an indication that this part of the management program often is neglected.

Bone Stimulators

If healing potential is diminished secondary to physiologic factors or patient wishes to promote a healing response, bone stimulators are an additional option. Electromagnetic fields and their uses in bone healing have been well studied, with most results showing improvement in healing of both bone and cartilage. There are three different methods for bone stimulation; direct current (DC), capacitive coupling (CC), and pulsed electromagnetic field (PEMF). Most supportive data are found in relation to the spine, femur, and tibia, but there is increasing evidence for its use in the foot and ankle for treatment of nonunions and as an adjunctive device in arthrodesis. Scott and King performed a level I prospective double-blind trial with CC on 21 patients with nonunions of femoral and tibial shaft nonunions.²⁶ They did report a significant benefit with bone stimulation between the two groups. Reports/studies have demonstrated that electric bone stimulation with DC, CC, and PEMF devices maybe a useful adjunct in the treatment of delayed unions and nonunions. For high-level athletes, a return to sport as soon as possible is paramount; therefore the use of a bone stimulator can be added to the treatment plan to enhance the possibility of an earlier return. Exogen (Bioventis, Durham, NC) is another device that can be used to stimulate bone healing. It is a pulsed low-intensity ultrasound device. Gold and Wasserman²⁷ did find benefit while using Exogen in their patients with large tibial segmental defects. Other studies have found similar findings.

SURGICAL INTERVENTION

If conservative treatment fails to heal the stress fracture and create a solid union, then surgical intervention is recommended. It is also important to note that with high-level athletes that surgical intervention is commonly the first line of treatment for many stress fractures of the foot and ankle. The type of surgery required varies depending on the location of the stress fracture and the type of bone that is requiring treatment. Briefly,

it can vary from the placement of screws across the fracture site to the placement of plate constructs to stabilize the fracture. Calcium phosphate cement has also been used in some stress fractures where typical screws and/or plates are difficult to place. Biologic augmentation is also being utilized at this time, bone marrow aspirate concentrate, platelet-rich plasma, and synthetic orthobiologic proteins can also be used to augment fixation and the healing response. Overall, the gold standard with all fractures that have healing concerns is autograft. Typically, this is taken from the calcaneus or the tibia on the ipsilateral side of the lower extremity. Surgical options will be discussed further below.

COMMON STRESS FRACTURES

Certain bones of the foot and ankle are more at risk for stress fractures. There are different reasons for why each bone has an increased risk, but many of these can be treated conservatively. Some examples of activities and professions that are at increased risk for stress fractures are long-distance runners, military recruits, and explosive sports.²⁸⁻³⁰ The following will review some of the more common stress fractures and discuss their onset, diagnosis, and treatment plan.

Medial malleolus stress fractures are not very common but do need to be closely watched to check for displacement. They are also notorious for having limited healing ability. Pain is commonly ill defined in the medial ankle region. There is no significant history of trauma that the patient can recall. This injury is commonly misdiagnosed as posterior tibial tendonitis or deltoid ligament injury. X-ray is initially obtained but if inconclusive, MRI is recommended. There is some controversy with treatment. Most individuals will recommend conservative care with immobilization in cast or cam boot and slow return to activity. If the fracture is easily visible on MRI and is more of a vertical orientation, ORIF is recommended to prevent proximal displacement of the fragment.

Lateral Malleolus fractures occur more often than medial malleolus fractures. There is a stress fracture of the distal fibula called "Runner's Fracture," and is believed to be caused by repetitive eccentric contractions of the plantar and long toe flexors with axial load.³¹ This is because when the foot is plantar flexed, the fibula is anatomically closer in relationship to the tibia and may impart undue stress at the distal end. Either way, the presentation is almost identical to all stress fractures with an insidious onset and increased pain with activity that progresses to pain at rest. Standard weight bearing radiographs are the initial standard, and in medial and lateral malleolus fractures, oblique images of the ankle at 45 degrees can also be considered. In the absence of any pathologic findings an MRI is considered a viable next step. Nonoperative treatment is recommended as initial treatment. Rarely is operative treatment necessary for lateral malleolus fractures. A cam boot or immobilization device is used, and a slow progressive return to non-painful activity is recommended.

Calcaneal stress fractures are the most common in military recruits, and many papers have reviewed this phenomenon.³²



Fig. 3.1 A-G, 27-year-old professional soccer player with left anterior ankle pain. Pain was slow in presentation, then chronically all the time. No obvious fracture on plain films. MRI and CT scan reveal a stress fracture of the navicular. Given the fact the patient is an elite athlete, he underwent operative fixation.

Clinically this type of stress fracture presents with a prodromal period and then worsening swelling and plantar heel pain. Typically, a calcaneal compression test can elicit pain and aid in the diagnosis. Commonly, lateral radiographs of the calcaneus can reveal a fracture line between 10–14 days after onset. Ideally, standard of care is an MRI to verify the diagnosis.³³ Treatment is always initially conservative with immobilization and refraining from high-intensity activity. Symptoms largely direct treatment modalities after an initial period of immobilization.

Another common tarsal bone that sustains stress fractures is the navicular. It is not as common as calcaneal stress fractures in military recruits but can occur in the same type of population, as well as explosive athletics such as sprinters. It commonly presents with an indistinct vague achy pain with activity that improves with rest, and pain at the dorsum of the midfoot or along the medial longitudinal arch with activity. It can easily go undiagnosed for quite some time given the difficulty in visualizing the navicular with plain radiographs. Clinically, it is difficult to make the diagnosis, and therefore advanced imaging is necessary (Fig. 3.1). MRI and CT scan can be used to understand the extent of the injury. In non-displaced stress fractures, conservative nonoperative treatment is the appropriate treatment modality.³⁴ When displacement is noted or there is a delay in diagnosis, then operative treatment is recommended. Operative treatment is also considered in elite athletes.

Metatarsal stress fractures occur via a similar mechanism as stated above. More specifically the second and third metatarsals sustain stress fractures more commonly than the other three metatarsals. Clinically, a prodromal period is noted prior to diagnosis of these injuries. Typically, the individual has increased their activity in some way. Radiographs and possibly MRI are imaging modalities routinely recommended as above.

Intrinsic and extrinsic factors have been implemented in the cause. These injuries can be confused with possible neuromas. It is important to remember that typically neuromas are more painful plantarly and are not associated with swelling/edema. Usually these injuries are diagnosed after callus or bony reaction is noted on initial radiographs (Fig. 3.2). Treatment is commonly nonoperative/conservative with a hard-soled shoe or cam boot walker for these injuries. Soreness can usually continue for upward of 3–4 months while these fractures heal. Treatment duration is tailored to symptom duration and activity.

Fifth metatarsal stress fractures are another stress fracture that usually occurs secondary to lateral overload or avulsion of the peroneus brevis. The fifth metatarsal fracture can have a prevalence to injury in a cavovarus foot. Surgery may be recommended in athletes or a recurrent base of the fifth metatarsal fracture. There are certain distinctions with fifth metatarsal stress fractures regarding location and healing rates that need to be taken into account.^{35,36} Occasionally, the cavovarus deformity will need to be corrected as well to reduce the risk of recurrence or non-union. Surgical fixation consists of a single screw placed in an antegrade fashion. Patients have returned to competitive sports within 6 weeks, but it should be noted that causes of failure were linked to early return. This fracture still has an appropriate healing rate in the layperson using conservative measures with immobilization and time.

These areas are the more common locations in the foot and ankle that sustain stress fractures. As stated throughout this text, initial treatment is conservative with immobilization. Occasionally, nutritional lab values can be obtained to possibly determine a physiologic cause in conjunction with immobilization.



Fig. 3.2 A-D, Stress fracture in a ballerina with chronic changes on CT. Healed with conservative treatment and immobilization.

PEARLS

Stress fractures are fatigue fractures and result from repeated overuse, and they are common in the athlete.

MRI is helpful to diagnose a stress fracture early in its presentation and should be ordered immediately for any patient participating in sport. Stress fractures of the second, third, or fourth metatarsals swell and the pain is dorsal, whereas neuromas of the forefoot do not swell and the pain typically is plantar.

Most stress fractures of the foot and ankle heal with relative rest. Navicular, fifth metatarsal, base of second, and medial malleolus may require more involved care for healing.

CONCLUSION

Stress fractures are a common injury, particularly in runners and in sports that involve a large amount of running. Various risk factors for the development of stress fractures have been proposed; however, the relative importance of these is still uncertain. The diagnosis is primarily on clinical grounds, and imaging can be used to confirm the diagnosis or to assess the extent of the injury. The treatment is straightforward in most cases, but there is a small group of stress fractures that require more specific management.

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Medical and Metabolic Considerations in Athletes With Stress Fractures

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INTRODUCTION

Bone stress injuries (BSI) are common injuries of athletes.

Much of the work in this chapter is based on our work with elite athletes over the last 13 years ([Box 4.1](#)).

For the purpose of this chapter, we will define elite athlete as one who is participating in athletics at the high school, college, or professional level. While some of the information may pertain to recreational athletes, we have made no attempt to collect our information on this group of individuals.

There are numerous articles on stress fractures in military populations and settings, beginning with the original reference in 1855 of “march fractures” by Breithaupt.¹ However, “The findings from military recruits (many of whom are undertrained) may not generalize to athletes (many of whom are well- or overtrained) as they may represent different populations.”² Therefore, we did not complete a full review on the military literature, but selected certain information from that body of work that is appropriate to our work.

DEFINITION OF BSI

BSI represent a “presumed” spectrum of bone damage that occurs as the result of repetitive trauma that is not handled correctly by the bone repair mechanisms. For management purposes, BSI may be considered as a “bone stress–failure continuum” in which “shin splints” (more properly, medial tibial stress syndrome) is a relatively mild expression of the damage at the low end of the spectrum and stress fracture is a severe example of the damage at the other end of the spectrum ([Tables 4.1 and 4.2](#)).

These conditions do not necessarily occur concurrently or in temporal sequence.³ In fact, we are unaware of someone with medial tibial stress syndrome who has progressed to a stress fracture or a stress reaction.

Numerous articles have defined the components of this spectrum (medial tibial stress syndrome, stress reaction, and stress fracture) (see [Chapter 23](#)), but there are no official diagnoses of any of these entities from the American Academy of Orthopaedic Surgeons (AAOS), the American Society for Bone

BOX 4.1 Elite Teams and Number of Athletes Seen in the Last 13 Years as of October 5, 2018

- San Diego Chargers – 3
- Houston Texans – 1
- Oakland Raiders – 5
- NY Football Giants – 2^a
- University of Georgia – 1^a
- UC Davis – 1
- Los Angeles Lakers – 1
- Milwaukee Bucks – 1^b
- Golden State Warriors – 4
- Philadelphia 76ers – 2 & 1^a
- Brooklyn Nets – 1
- New Orleans Pelicans – 1
- Dallas Mavericks – 1
- Houston Rockets – 1^a
- Charlotte Bobcats – 1^a
- OKC Thunder – 2^a
- Washington Wizards – 1
- U of Portland male cross-country runner – 1
- Minnesota Timberwolves – 1^a
- Oakland Athletics – 10
- Colorado Rockies – 2
- Los Angeles Dodgers – 1
- Los Angeles Angels – 1
- Detroit Tigers – 1
- Arizona Diamondbacks – 1
- Cleveland Indians – 1 & 1^a
- US Olympic Skating – 1^b
- Harvard female cross-country runner – 1
- UC Berkeley Basketball – 2
- San Diego State Basketball – 2^a
- Wesleyan softball player – 1
- Sacramento Kings – 1
- Valparaiso basketball player – 1^a
- St. Louis Cardinals – 1^a
- Minnesota Timberwolves – 1^a
- New York Knicks – 1^a
- New York Jets – 3^a

^aConsulted but not seen in office

^bNot seen yet but referred

and Mineral Research (ASBMR), or the American College of Sports Medicine (ACSM).^{4–6}

“Shin splints” is a nonspecific lay term associated with a large number of fundamentally different tibial exercise-induced leg injuries where there is repeated foot-to-ground impact. Such distinct leg injuries as tibial and fibular stress fractures, tibial periostitis, anterior and deep posterior compartment syndromes, popliteal artery entrapment, and tibia-lis posterior and anterior muscle strain, or tendinitis have all been referred to under the rubric of shin splints. As part of the push to make a more specific medical evidence-based diagnosis of these different entities, medial tibial stress syndrome (MTSS) has been separated from the other conditions. MTSS is a condition comprising periostitis or symptomatic periosteal modeling occurring in the vicinity of the junction of the middle and distal thirds of the medial border of the tibia. It is

TABLE 4.1 Low-Risk Stress Fracture Treatment Guide

Symptoms	Goal	Treatment Suggestions
Any level of pain	Heal injury	Titrate activity to a pain-free level for 4–8 weeks depending on the grade of injury Braces/crutches Modify risk factors
Pain with no functional limitations	Continue participation	Titrate activity to a stable or decreasing level of pain Closely follow Modify risk factors
Pain with functional limitation	Continue participation	Decrease activity level to point at which pain level is decreasing and until a functional level of pain has been achieved, then titrate activity to stable or continued decrease level of pain Modify risk factors
Limiting pain intensifies despite functional activity modification (i.e., unable to continue to perform at any reasonable functional level despite activity modification)	Heal injury	Complete rest Immobilization Surgery Modify risk factors

From Diehl JJ, Best TM, Kaeding CC. Classification and return-to-play considerations for stress fractures. *Clin Sports Med* [Internet]. 2006 Jan [cited 2018 Jul 6];25(1):17–28, Table 1.

a more diffuse lesion spreading several centimeters over the bony surface.³

As a specific entity, stress reactions are studied much less than MTSS or lower extremity stress fractures. Although the causation of stress reactions, the complaints by which they present, the history, the physical findings, and the imaging by which they are diagnosed are the same as stress fractures, the term “stress reaction” means there is no fracture line or break in the continuity of the bone. X-ray images are usually negative in an initial stress reaction. A bone scan will be positive, but this cannot help tell whether there is or is not a fracture line. Therefore, the diagnosis is made by either magnetic resonance imaging (MRI) or computed tomography (CT) scan.⁷

Stress fractures result from the repeated application of a stress lower than that required to fracture bone in a single loading situation.⁸

CLASSIFICATION SYSTEMS OF BONE STRESS INJURIES

The classification of stress fractures has become more complex since the initial description by Breithaupt in 1855¹ and Stechow’s subsequent observation that on early radiographs, clinical findings in the feet were due to fractures.⁹ Garbuz et al. reviewed the value and role of orthopedic classification systems used to characterize the nature of a problem to guide treatment

TABLE 4.2 Management of and Return-to-Play Strategies for High-Risk Stress Fractures

Anatomic Site	Complications	Suggested Treatment	Level of Data
Femoral neck	Displacement Nonunion Avascular necrosis	Tension: Strict NWB or bed rest Surgical fixation RTP when healed Compression: NWB until pain-free with radiographic evidence of healing, then slow activity progression RTP after no pain on examination or with any activities Surgical fixation (optional)	Level C (expert opinion) Level D (case series)
Anterior tibia	Nonunion Delayed union Fracture progression	Nonoperative: NWB until pain-free with ADL; pneumatic leg splints RTP with slow progression after nontender and pain-free with ADL (9 mo) Operative: Intramedullary nailing RTP is usually faster (2–4 mo)	Level A (RCT) Level B (nonrandomized) Levels C and D
Medial malleolus	Fracture progression Nonunion	Nonoperative: (No fracture line) 4–6 wk pneumatic casting Avoid impact; rehabilitation RTP when nontender, no pain with ADL Operative: (Fracture line, nonunion, or progression) ORIF with bone graft	Levels C and D
Tarsal navicular	Nonunion Delayed union Displacement	Nonoperative: NWB cast 6–8 wk, then WB cast 6–8 wk RTP is gradual after pain-free with ADL Orthotics and rehabilitation suggested Operative: (Complete, nonunion) RTP only when healed	Levels C and D
Talus	Nonunion Delayed union	Nonoperative: NWB cast 6–8 wk RTP is gradual after pain-free with ADL Orthotics and rehabilitation suggested Operative: Reserved for nonunion	Level C
Patella	Displacement Fracture completion	Nonoperative: (Nondisplaced) Long-leg NWB cast 4–6 wk Rehabilitation following RTP is gradual after pain-free with ADL Operative: Horizontal—ORIF Vertical—lateral fragment excision RTP when healed	Level C
Sesamoids	Nonunion Delayed union Refracture	Nonoperative: NWB 6–8 wk RTO is gradual after pain-free with ADL Operative: Excision if fail nonoperative	Level C
Fifth metatarsal	Nonunion Delayed union Refracture	Nonoperative: (No fracture line) NWB cast 4–6 wk followed by WB cast until healed RTP after nontender and pain-free Operative: (Fracture line, nonunion, or individual at high risk for refracture) Intramedullary screw fixation RTP 6–8 wk, early ROM/rehabilitation	Levels C and D

ADL, Activities of daily living; NWB, nonweight bearing; ORF, open reduction with internal fixation; RCT, randomized controlled trial; ROM, range of motion; RTP, return to play; WB, weight bearing; wk, week.

From Diehl JJ, Best TM, Kaeding CC. Classification and return-to-play considerations for stress fractures. *Clin Sports Med* [Internet]. 2006 Jan [cited 2018 Jul 6];25(1):17–28. Table 2.

decision-making and establish an expected outcome for the natural history of a condition. This formed a basis for uniform reporting of results for surgical and nonsurgical treatments and for comparison of results from different centers.¹⁰

Various classification systems have been proposed on the basis of clinical findings (e.g., client history and physical examination)¹¹; radiographic results,¹² including scintigraphy,^{13,14} ultrasound,¹⁵ CT,¹⁶ MRI^{17,18} and dynamic contrast-enhanced MRI¹⁹; fatigue versus insufficiency (pathogenesis)^{20–22}; high- versus low-risk fractures^{23,24}; and on practices that involve multiple components of

these aspects of fractures. Arendt and Griffiths developed a classification based on x-ray, scintigraphy, and MRI findings.²⁵

The most intensive and thorough review of this issue comes from Kaeding and his group in a series of articles starting in 2005.^{26–29} In their initial paper,²⁶ they reviewed the pathophysiology, diagnosis, and classification of stress fractures on the basis of the separation of low-risk stress fractures and high-risk stress fractures.^{23,24} High-risk stress fractures (see also [Chapters 3 and 5](#)) occur at the femoral neck, the patella, the anterior tibial diaphysis, the medial malleolus, the talus,

TABLE 4.3 Stress Fracture Classification

High-Risk Fractures	Low-Risk Fractures
Occur on tension side	Occur on compression side
Natural history poor	Natural history favorable
Often require aggressive treatment, including surgery or strict nonweightbearing	Often require nonsurgical treatment with rest and gradual return to weight-bearing

From Kaeding CC, Najarian RG. Stress fractures: classification and management. *Phys Sportsmed* [Internet]. 2010 Oct 13 [cited 2018 Jul 6];38(3):45–54. Table 2 Available from: <http://www.tandfonline.com/doi/full/10.3810/psm.2010.10.1807>.

the tarsal navicular, the proximal fifth metatarsal and the first metatarsal phalangeal sesamoids. Low-risk stress fractures include the femoral shaft, medial tibia, ribs, ulna shaft, and first through fourth metatarsals. Previous studies showed that high-grade injuries (grade 3 and 4) took longer to heal than low-grade injuries (grade 1 and 2).

The management of each fracture should be individualized. “The key difference between a low-grade stress fracture at a high-risk versus low-risk location is that an individual who has a low-grade fracture at a low-risk site can be allowed to continue to compete but an individual who has a low-grade fracture at a high-risk site needs to heal before full return to activity”²⁷ (Table 4.3). High-risk stress fractures have more frequent complications like delayed union, nonunion, and refracture.

Kaeding and Najarian²⁷ continued the development of their classification system in 2010, stressing the important distinction between a high-risk and a low-risk fracture.

High-risk fractures occur on the tension side, have a poor natural history, and require aggressive treatment, whereas low-risk fractures occur on the compression side, have a favorable natural history, and frequently can be handled by nonsurgical treatment. A complete classification system of a stress injury (reaction or fracture) requires knowledge of the anatomic location and the grade of injury. Arendt and Griffiths²⁵ and Fredericson¹⁸ have developed grades of increasing severity of these stress fractures, from 1 to 4, with the latter representing a complete fracture. At that time, they felt that the management of stress injuries should be determined by the location and grade of the injury.

Subsequently, Miller, Kaeding, and Flanigan conducted a systematic review of the literature of Classification Systems of Stress Fractures.²⁸ They wanted to determine if there was a system that was “reproducible, inexpensive, safe, broadly applicable, widely accessible, and clinically relevant to prognosis and treatment considerations.” According to their review, 27 previous systems were found and analyzed for strengths and weaknesses. Of the most commonly cited systems in their review, none included a clinical parameter or parameters. None of the classification systems tested for inter- or intra-observer agreement; therefore, their reproducibility of use by single or multiple observers is unknown. Of the 27 systems evaluated, 16 were applicable to the entire skeleton, whereas 11 were applicable only to a specific bone or location. The more modern classification systems included MRI as an imaging technique. Arendt and Griffiths’ system is most often cited since 1990, and provides a system of radiologic grading of

TABLE 4.4 Proposed Stress Fracture Classification System

Grade	Pain	Radiographic Findings (CT, MRI, Bone Scan, or Radiograph)		Description
I	No	Imaging evidence of stress fracture, no fracture line		Asymptomatic stress reaction
II	Yes	Imaging evidence of stress fracture, no fracture line		Symptomatic stress reaction
III	Yes	Nondisplaced fracture line		Nondisplaced fracture
IV	Yes	Displaced fracture (≥2 mm)		Displaced fracture
V	Yes	Nonunion		Nonunion

From Kaeding CC, Miller T. The comprehensive description of stress fractures: a new classification system. *J Bone Jt Surg - Ser A* [Internet]. 2013 Jul 3 [cited 2018 Jul 6];95(13):1214–20. Table 1. Available from: <https://insights.ovid.com/crossref?an=00004623-201307030-00010>.

stress fractures incorporating x-ray, bone scan, and MRI findings graded from 1 to 4. Five other systems used multiple imaging modalities. Four of the less frequently cited systems did use clinical parameters, and pain was the most common symptom mentioned. None of the studies incorporated an assessment of the healing capacity of the fracture combined with a notation of the extent of structural damage. The authors were able to determine from their systematic review of the literature that the ideal stress fracture classification system they hoped to find did not exist.

Subsequent to the that analysis, Kaeding and Miller sought to develop a system that incorporated their belief that the classification describe not only the extent of the structural damage but also the healing potential of the lesion.²⁹ This is complicated by the tremendous variability of the stress fracture lesion. Their classification system employs three descriptors: 1) fracture grade; 2) fracture location; and 3) imaging modality used (Table 4.4).

The system they developed had the reproducibility they desired, was simple, easy to use, and formed the basis for treatment. When reporting the stress fracture, “a CT scan revealing a nondisplaced fracture line in a tarsal navicular in a healthy collegiate basketball player would be reported as a Grade-III tarsal navicular stress fracture on CT scan.”²⁹ We have adopted this system for use at our center.

EPIDEMIOLOGY OF BONE STRESS INJURIES

For further details, see Chapters 3 and 5.

PATHOPHYSIOLOGY

The understanding of the pathogenesis of stress fractures has advanced since Breithaupt’s original description,¹ advancing beyond the concept that they are due to performing repetitive tasks resulting in overuse with accumulation of microdamage. Recent advances in our understanding of bone biology enable us to have a deeper insight into the actual events²⁹ (Fig. 4.1).

There are numerous beautiful descriptions of the pathogenesis of stress fractures on a macroscopic level but very few on a microscopic or nano-structural level because most of these cellular and

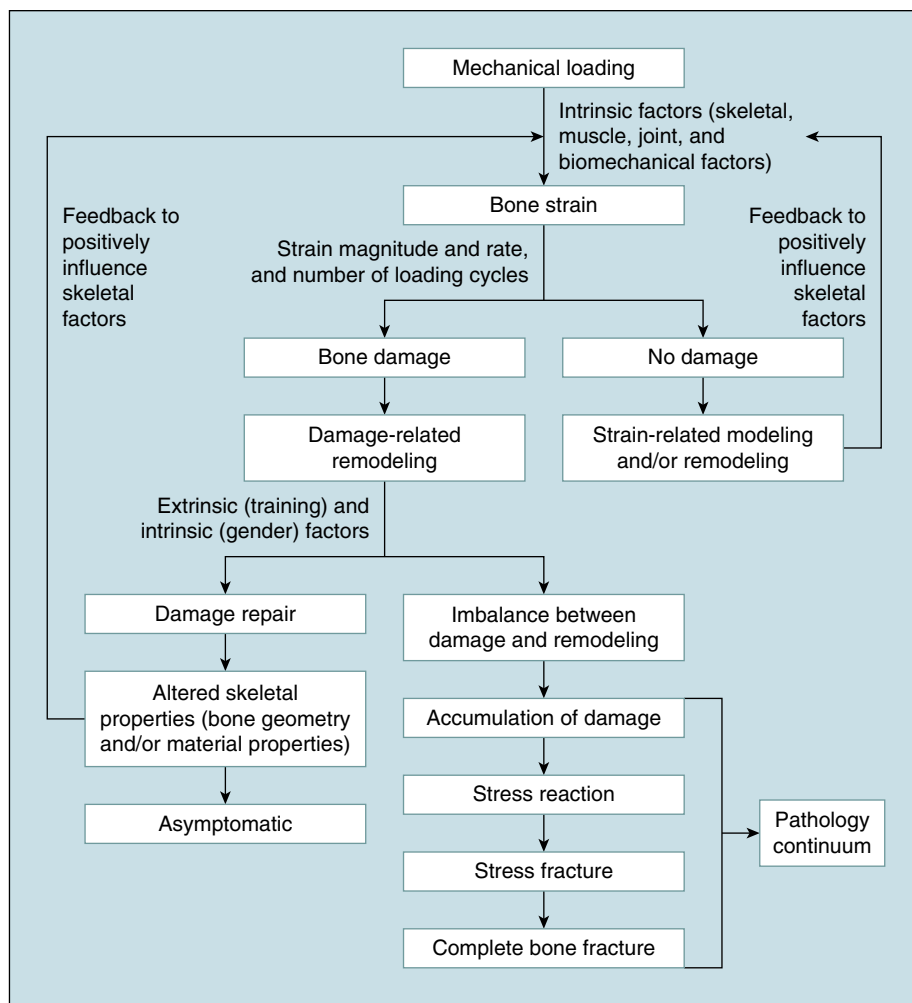


Fig. 4.1 Proposed pathophysiology of stress fractures. (From Warden SJ, Burr DB, Brukner PD. Stress fractures: pathophysiology, epidemiology, and risk factors. *Curr Osteoporos Rep.* 2006 Sep;4(3):103–109. Fig 1.)

subcellular evaluations are new and the findings are just being incorporated into the overall picture as our knowledge develops.

In 1998, Harold M. Frost, one of the clearest thinkers about bone physiology, histomorphometry, and bone pathology, stated, “Bone is a fatigue-prone material.”³⁰

In 2001, Boden et al. stated that the “exact mechanical phenomenon responsible for initiating stress fractures remains unclear.”²⁴ But it is clear that an increase in the duration, intensity, or frequency of physical activity, either military basic training or athletics, without sufficient rest intervals may lead to increased osteoclast activation and bone resorption. Muscle fatigue may also result in excessive forces being transmitted to the bone. In 2002, Romani et al. added that “stress fractures are not the result of one specific insult. Instead, they arise as the result of repetitive applications of stresses that are lower than the stress required to fracture the bone in a single loading.”³¹ Whenever a low level of force is directed on to the bone, whether due to contact with the ground or muscle activity, it causes the bone to deform, which is known as a strain. The bone’s stress–strain response depends on the load’s direction; the bone’s geometry, microarchitecture, and density; and the role of attached muscle and its contractions. “In most activities of daily living (ADLs), when the force is removed, the bone elastically

rebounds to its original position. The force that a bone can endure and still rebound back to its original state without damage is within the elastic range. Forces that exceed a critical level above the elastic range are in the plastic range. Once forces reach the plastic range, a lower load causes greater deformation; it is at this level that forces summate to permanently damage the bone.”³¹

Warden and his colleagues stated in 2006 that the “precise pathophysiology of stress fractures is unknown, and current models are based on theory.”³² Although the pathogenesis of stress fractures in these models is usually discussed at the macroscopic level, damage really initiates at the level of the collagen fiber or below.³³ Fatigue is the loss of strength and stiffness that occurs in materials subjected to repeated cyclic loads.³⁴ Bone fatigue fractures (now known as stress fractures) are a complex in vivo phenomena in which mechanical damage and biological repair have major roles.³⁵ If microdamage from bone fatigue activity accumulates at a slow rate, normal biological remodeling may be able to repair the damage and retain the structural integrity of the bone. However, the creation of microcracks initiates osteoclastic bone resorption and the microdamage removal in a bone that continues to be excessively loaded with high cyclic stresses may accelerate the accumulation of fatigue damage.

With every bone movement, and especially repetitive episodes of mechanical loading, bone strain occurs, producing microdamage. Strain is defined as the change in length per unit length of a bone and is frequently expressed as microstrain ($\mu\epsilon$). Usual strains (400–1500 $\mu\epsilon$) are far below the single load-failure threshold (10,000 $\mu\epsilon$). But strains below the single load-failure level can have a cumulative effect on the bone structure. Carter et al. studied the fatigue behavior of adult cortical bone. The bone fatigue microdamage accumulates at a slow but unknown rate, and how much is too much is also unknown. Cortical bone fails in fatigue within 10^3 to 10^5 loading cycles when strains are between 5000 and 10,000 $\mu\epsilon$.³⁵ According to Schaffler and colleagues, strains in the physiologic range of 1000 to 1500 $\mu\epsilon$ in ex vivo studies have been shown to cause fatigue and microdamage but not to result in complete fracture of cortical bone even after 37 million loading cycles.³⁶

The repetition of sub-maximal strains produces microdamage in the bone. Much effort has been undertaken to decide how much strain can produce microdamage and how much microdamage can produce fatigue failure of bone. Schaffler et al. summarized some other work by noting that “strains in the range of 1200–1500 microstrain and strain rate of 0.03 s^{-1} are typical of the strain environment measured on tensile surfaces of long bone diaphysis during running. If one assumes (conservatively) a stride length of three feet for a runner. Each [sic] limb would be loaded every six feet, and each million cycles would correspond to about 1136 miles of running. Ten million load cycles correspond to more than 11,000 miles of continuous loading without the advantages of remodeling or repair.”³⁵ This implies a much greater fatigue resistance for compact bone at physiological strains than would be calculated from earlier studies.³⁴ The military recruit experience suggests that stress fractures occur within 6 weeks after the start of basic training, or at about 100 to 1000 miles of vigorous exercise, or an estimated 100,000 to 1,000,000 loading cycles by the previous calculations. This “suggests that other mechanisms may be involved in fatigue failure.”³⁶ They hypothesized that “brief stress or strain loading would lead to complete fracture.”³⁶

The excessive strains produce microcracks in the bone and microdamage resulting in collagen fiber-matrix debonding, disruption of the mineral-collagen aggregate, and collagen fiber failure.³³

There are three distinct types of microdamage based on differences in staining properties in bone. These different staining characteristics also allow for the demonstration of microcracks in the bone. “Fatigue loading and the extent of microdamage are associated.”³⁷ Microdamage is difficult to demonstrate in bone specimens and takes special staining procedures, developed initially by Frost. It is considered impossible to apply these techniques to in vivo situations. The specific interaction between mineral and collagen is poorly understood. To understand microdamage in vivo is going to take the development and use of new imaging tools, like Trabecular Bone Score (TBS) and Texture Research Investigation Platform (TRIP) (Medimaps Group SA, Geneva, Switzerland), μCT , finite element analysis (FEA), and high-resolution peripheral CT (HRpQCT).³⁸

Hughes et al. reviewed the role of adaptive bone formation in the etiology and prevention of stress fractures. The typical model of stress fracture development does not account for the actual clinical occurrence of a stress fracture. In the previous calculation by Schaffler,³⁶ the basic training recruits are exposed to less than 1/400th the duration of loading required to develop a fatigue-induced stress fracture under in vitro loading conditions. This may be due to differences between the experimental loading conditions, where bones may be repetitively strained in one plane, and the live loading conditions, in which the soldier or the athlete is subjected to multidirectional loading. This may result in more abnormal loading and strain, producing more microdamage than the experimental model does. Additionally, in the experimental model, a small portion of bone is utilized, and the human bone is larger and is more likely to have weaker regions than the laboratory specimen, and in vivo bones are undergoing remodeling in response to increased mechanical loading, which can transiently weaken the bone.³⁹ Remodeling directed at removing microdamage is referred to as *targeted remodeling*. This repair activity causes a temporary porosity that may contribute to stress fracture risk, although the authors say, “the link between increased porosity and stress fracture risk remains to be demonstrated experimentally. In principle, the process of bone remodeling in response to physical training is paradoxical in that it may *promote* stress fracture development by introducing an acute increase in porosity, but may also *prevent* stress fracture development by replacing fatigue-damaged bone.”³⁹ The porosity that develops represents a temporary negative bone balance that exists until the resorption cavity is filled with new bone that becomes fully mineralized. However, deposition of osteoid, newly formed unmineralized bone, by osteoblasts does not immediately restore normal bone stiffness and other characteristics. What is needed is mineralization, which occurs in two phases: primary mineralization, which occurs during the first few weeks and results in 65%–70% of the final mineralization; and secondary mineralization, which occurs slowly over the next 8–12 months.⁴⁰ “As stress fractures may occur within weeks of onset of physical training, newly activated remodeling cycles remain in early stages when a negative bone balance can theoretically contribute to a cycle of increased strain and accumulation of microdamage upon continued loading until stress fracture ensues.”⁴⁰ Targeted remodeling is a key process for replacing fatigue damage. Bone remodeling is a process characterized by four phases: the activation phase, when the osteoclasts are recruited; the resorption phase, when the osteoclasts resorb bone; the reversal phase, when the osteoclasts undergo apoptosis and the osteoblasts are recruited; and the formation phase, where the osteoblasts lay down new organic bone matrix that subsequently mineralizes. By definition, remodeling is a process where osteoclasts and osteoblasts work sequentially. Bone modeling is the process in which bones are shaped or reshaped by the independent (noncoupled anatomically or temporally) action of osteoblasts and osteoclasts. Skeletal development and growth take place by the process of bone modeling.⁴¹

Adaptive bone formation deposits bone, via bone modeling (as opposed to remodeling), on the periosteal, endocortical, or trabecular surfaces in response to mechanical loading.

Bone modeling is the process of bone growth that takes place in infants, children, and adolescents where bone is forming and involves the independent action of osteoblasts without prior osteoclastic bone resorption. Modeling involves osteocyte activation where the osteocytes act as mechanotransducers. Osteocytes are important regulators of bone function (and will be discussed more thoroughly below). The mechanosensing and mechanotransducing osteocytes transform an induced deformation of the bone matrix from some external force into biochemical and flow signals that lead to new bone formation.³⁹ The mechanism for stimulation of osteocytes is thought to be electric streaming potential created by ionic fluid movement through the lacuna-canalicular system and cellular shear stress generated by fluid flow along the osteocyte cell body and dendritic processes. Apparently, the cell body, the primary cilia, and the dendritic processes are responsible for mechanosensation. Changes in the osteocytes and their dendrites lead to increased intra-osteocyte calcium signaling, and formation of pro-osteoblastic molecules such as prostaglandin E₂ (PGE₂), insulin-like growth factor (IGF-I), nitric oxide, and adenosine triphosphate (ATP) that positively affect bone formation and suppress osteocyte production of negative regulators of the Wnt/ β -catenin pathway, such as sclerostin and dickkopf-1 (DKK1).⁴²

Deposition of bone along the diaphysis of long bones on the periosteal surface provides great mechanical advantage.³⁹ Long bones with mass distributed furthest from the neutral axis, i.e., wide bones, are stronger in relation to bones with similar masses that are narrower.⁴³ Stress fracture risk is affected directly by the properties of the skeleton, like wider bones or denser bones, and thus it is thought that modification of these properties via the adaptive ability of bone may be used as a way of reducing an individual's risk. Warden et al. looked at bone adaptation to a site-specific mechanical loading program using a rat ulna axial loading model that compared the loaded right forearm (ulna) to the control, an unloaded left forearm (ulna). The mechanical loading induced bone changes that resulted in a significant increase in ulna fatigue resistance. The authors found that by improving the structural properties of a bone through a mechanical loading program, the bone's fatigue resistance could be significantly improved. They suggested that an exercise program directed at changing the structural properties of the skeleton can be employed as a possible prevention strategy for stress fractures. When the fatigue life of the trained and untrained limbs was compared, the untrained limb fractured after 15,000 cycles whereas the trained limb failed after an average of 1.5 million cycles. This 100-fold increase in fatigue resistance after a 5-week loading regimen shows the potential impact of adaptive bone formation with physical training.^{39,44}

Milgrom and his group performed three prospective studies of military recruits in different basic training classes to evaluate bone's adaptation ability to lower the incidence of stress fractures. Different groups of 452, 433, and 404 elite infantry recruits had their physical fitness assessed by a timed 2-km run, the maximum number of chinups they could perform, and the number of situps they could perform in 1 minute. The pre-induction participation in sports activity was assessed. In the third study,

the questionnaire was revised to subdivide the type of ball sports into soccer, basketball, volleyball, tennis, and handball. Of the 1118 soldiers who completed basic training, stress fractures in the group that did not play ball ranged between 28.9%, 27%, and 18.8% in the individuals in each group, respectively, who did not participate in ball sports and 13.2%, 16.7%, and 16.3% in the individuals in each group who did play ball sports. They also inserted strain gauges into the tibias of three volunteers and found that the tension, compression, and shear strain rates during rebounding were higher than those during running and were 2.16 to 4.60 times higher during rebounding and running than during walking.⁴⁵ In Scandinavian⁴⁶ and Israeli⁴⁷ studies, a history of long-distance running or jogging did not affect the incidence of stress fractures in military recruits. Previous activities such as weightlifting, swimming, and martial arts did not lower the incidence of stress fractures, and a history of swimming increased the risk for stress fractures. In the paper presenting three separate studies,⁴⁵ those recruits who played ball sports for more than two years before their military training, in the first two of the three studies, had only 50% of the stress fractures; in the third study, where the recruits were specifically asked what sport they played and what ball sport they played, 90% of the recruits played basketball, and in those who played ball sports, the stress fracture rate was 20% (80% decrease) compared to those who did not play ball sports. To explain this phenomenon, *in vivo* human tibial bone strain measurements were obtained in a number of different studies including the above-cited one.⁴⁵ The principal compression strain was 48% higher, the principal tension strain 15% higher, and the shear strain 64% higher during basketball, rebounding than during running. The compression strain rate was 20% higher, the tension strain rate 6% higher, and the shear strain rate 28% higher during basketball rebounding than during running. The amount of strain and strain rate change are major determinants of adaptive bone formation to loading. The authors felt that the high strains and strain rates that occur during playing basketball can cause maximum adaptive bone formation. This resulted in stiffer bone in the basketball players who played for 2 years before entering the military and, thus, less bone strain during basic training than the recruits who did not play basketball, and therefore fewer stress fractures in the basketball players. Milgrom et al. concluded that, "On the basis of this study, a logical strategy for lowering the incidence of stress fractures in military recruits and athletes would be to adapt their bones before they begin formal training. This would involve a pretraining program, over a course of at least 2 years, of properly applied high-strain- and high-strain-rate-generating exercises that mimic the strain and strain rates that occur during basketball. Such a program would ideally stiffen the bone and not lead to stress fractures during this adaptation period."⁴⁵ In our opinion, a modern understanding of the pathophysiology of stress fractures requires an understanding of the role of osteocytes in bone physiology and pathophysiology.

Osteocytes were first described by Carl Gegenbaur (also Gegenbauer), a German physician, anatomist, zoologist, and physiologist,⁴⁸ in 1864,⁴⁹ only 9 years after Breithaupt¹ described "march" fractures. It would take over 150 years for stress fractures and osteocytes to come together.

In the last few years, there have been multiple reviews of the etiology, evolution, and function of osteocytes. It is clear from these studies that the matrix-producing osteoblasts can become an osteocyte, a lining cell, or can undergo programmed cell death.^{50,51}

Hazenbergh and colleagues state that human bone contains between 13,900 and 19,400 osteocytes per mm³.⁵² Buenzli and Sims give slightly different numbers of 20,000 to 30,000 osteocytes per mm³, but since it is estimated that 5% of osteocyte lacunae are empty, this suggests an average osteocyte density of 19,000 to 30,000 osteocytes per mm³. This results in an estimate of 42 billion osteocytes in the human skeleton.⁵³ The osteocytes form an interconnected network through their dendritic processes, creating communication between individual osteocytes and the surface bone lining cells. The number of osteocytic dendritic processes varies per species. Osteocytes contain between 40 and 60 cell processes per osteocyte with a cell-to-cell distance of 20–30 µm per Hazenbergh, and 18–106 processes per Buenzli, making a total of 3.7 trillion projecting from the osteocyte cell bodies. This results in a cumulative length of all osteocytic dendritic processes in the human skeleton to be 175,000 km (108,740 miles). One cell process may form up to 12.7 termini on average, so that a single osteocyte may possess up to 1128 termini connecting with other cells. Extrapolated to the whole skeleton, this calculates to 23.4 trillion osteocytic connections.⁵³ Transmission of mechanical signals to the osteocytes can occur directly via cell surface receptors through the solid matrix of the tissue due to load-induced fluid flow or indirectly via fluid pressure and shear stresses.⁵²

Osteocytes have numerous functions in bone. They can serve as orchestrators of bone remodeling including formation and resorption; inducers of osteoclast activation; modulators of mechanical loading via mechanosensation and transduction; sources of factors and regulators of mineral metabolism; remodelers of the perilacunar matrix; and other functions, such as regulators of mineralization. Osteocyte cell death leads to skeletal fragility via the recruitment of osteoclasts to the site. Viable osteocytes secrete an as yet unknown factor or factors that inhibit osteoclast activity, and when they die, osteoclasts are released from inhibition to start the process of bone resorption.⁵⁴ They perform these functions via the release of signaling molecules such as nitric oxide, prostaglandin E₂, and ATP from the osteocytes in response to external stimuli, such as mechanical strain. Fluid fluxes in the canaliculi and, perhaps, electromechanical signals induced by the mechanical loading also participate. The osteocytes are multifunctional cells, and they undertake some of these functions via an endocrine role. Osteocytes are known to produce fibroblast growth factor 23 (FGF 23), one of the most important osteocyte-secreted endocrine factors, which plays a role in phosphate metabolism, is a marker of early kidney failure, and can down-regulate 1- α hydroxylase, which is required for the conversion of 25-hydroxyvitamin D to the active 1,25-dihydroxyvitamin D. They also produce sclerostin, which is an inhibitor of bone formation in the Wnt- β catenin system. In addition, other factors involved in phosphate metabolism, such as DMP1, PHEX, and MEPE, are expressed by the osteocyte. There is crosstalk

between osteocytes and muscle cells, which also play a role in response to mechanical stimuli.

Microcracks develop as a result of daily cyclic loading, which is repaired by a balanced process between resorbing and forming cells.⁵² Microcracks can damage the osteocyte and its processes, inducing the osteocyte to send signals to initiate bone resorption and formation. Microdamage and bone fatigue are both associated with loss of osteocyte integrity.⁵⁵ The role of osteocytes is being increasingly recognized in a wide variety of metabolic bone diseases. Lower osteocyte density has been shown to play a role in some patients who are destined to sustain a vertebral compression fracture.⁵⁶ Apoptosis of osteocytes has been recognized as a factor in glucocorticoid-induced osteonecrosis of the hip.⁵⁷ Parathyroid hormone (PTH) and the PTH Type 1 Receptor (PTH1R) play a role in osteocyte survival as well as in the mechanosensory process.⁵⁸

Although no real research has been performed on stress fracture patients, the increasing understanding of the role of fatigue and microdamage in disrupting canalicular flow and creating apoptosis of osteocytes, which initiates bone resorption and remodeling, are all steps in the pathogenesis of stress fractures. In 2016, we formulated the hypothesis that stress fractures may be due to disordered, dysfunctional, or diseased osteocytes where fluid mechanics, shear/strain forces, mechanosensory forces, mechanotransducer forces, and production and release of bone growth inhibitors (Dkk1, sclerostin) and bone growth stimulation (activation of the Wnt/ β -catenin pathway) may result in abnormal bone remodeling, including the necessary bone resorption and, perhaps, the lesser or delayed bone formation that allows bone failure to occur with subsequent development of a stress fracture. It is increasingly clear that the effect of many anti-osteoporotic drugs, like PTH, may be mediated by their direct or indirect effect on osteocytes.^{59–61} (The role of PTH and other anabolic drugs for bone is discussed under the section on Treatment)

GENETIC PREDISPOSITION

The ease with which it is possible to study the human genome has improved tremendously in the last several years, and the cost of doing so has markedly decreased, putting these individual analyses within reach of the general public. We now can both perform genome-wide association studies (GWAS) and study single nucleotide polymorphisms (SNPs).

Many studies have looked at the genetics of osteoporosis,⁶² the genetics of low bone mineral density (BMD),^{63,64} and the genetics of fragility fracture.^{65,66}

It has been increasingly shown that many sports injuries have a genetic basis. Some of this pioneering work has been done by Stuart K. Kim and colleagues at Stanford. Among other injuries, a genetic predisposition has been shown for medical collateral ligament (MCL) rupture,⁶⁷ shoulder dislocation,⁶⁸ rotator cuff injury,⁶⁹ ankle sprains and strains,⁷⁰ De Quervain's tenosynovitis,⁷¹ and plantar fasciitis.⁷² There is increasing evidence that, to some degree, there is a genetic predisposition to stress fractures.

One of the early interesting occurrences in this regard was reported by Singer et al. in 1990. Two 18-year-old identical twin brothers, who were in the same basic training program in the Israel Defense Force (IDF), were seen with pain in the proximal part of the left thigh starting 4 weeks before examination in the sixth week of their training class. Both brothers were in good physical health and exercised regularly prior to their induction. They both underwent nuclear medicine bone scans with Technesium-99m diphosphonate, which showed significant uptake in both the left and right proximal femurs along with some uptake in the tarsal bones of their right feet. Although the authors listed numerous potential clinical risk factors, they stated that genetic factors had never been considered to play a role in predisposition to stress fractures, but the finding in this monozygotic twin set suggested that genetic factors might need to be considered in the future.⁷³

In 1997, Burnstock summarized work being performed to elucidate the role of purine nucleotides as signaling molecules.⁷⁴ Subsequent experimental studies showed that osteoclasts and, perhaps, a subpopulation of osteoblasts contain cell surface nucleotide receptors and established a role for the P2X nucleotide receptor in bone formation and resorption. P2X7 receptor-deficient mice have smaller bone diameter and lower cortical mass and a reduction in periosteal bone formation. Deletion of the P2X7 receptor resulted in decrease in periosteal mineralizing surface, mineral apposition rate, and bone formation rate consistent with reduced periosteal osteoblast number and activity. Nucleotides released from many cell types in response to mechanical stimulation are felt to mediate mechanotransduction in bone.⁷⁵ This has now been confirmed in work by Li and Turner, where it was shown that the P2X7 nucleotide receptor mediates skeletal mechanotransduction.⁷⁶ Subsequently, the occurrence of mutations in the cytoplasmic domain of the P2X7 receptor has been reported.⁷⁵

Advancing from these murine studies to human studies, numerous (and increasing) functional SNPs have been identified, which result in either gain or loss of function of the P2X7 receptor protein (P2X7R) and have been associated with important clinical bone alterations. Jørgenson and colleagues conducted a 10-year genetic analysis of SNPs of the P2X7R gene in the Danish Osteoporosis Prevention Study (DOPS) population. They were able to show that several of the uncommon loss-of-function variants induced a predisposition to accelerated loss of BMD in postmenopausal women similar to the loss produced in knockout mice in the previously cited studies. The small number of individuals in each of three different risk groups prevented them from showing a relationship to osteoporotic fracture.⁷⁷ Gartland and colleagues, using the Aberdeen Prospective Osteoporosis Screening Study, showed that polymorphisms in the P2X7R gene were also associated with low lumbar spine BMD in addition to confirming accelerated bone loss in their postmenopausal women.⁷⁸ Wesselius and his group, which included some of the aforementioned researchers, studied men and women ≥ 50 years of age who had presented to the osteoporosis clinic at the Maastricht University Medical Centre (MUMC), the Netherlands, following a traumatic or fragility fracture. The subjects were genotyped for 15 nonsynonymous

SNPs within the P2X7R gene. Some SNPs seemed to be gain-of-function polymorphisms and were associated with higher BMD, whereas others were loss-of-function polymorphisms and were associated with lower BMD.⁷⁹ Husted et al. studied SNPs of the P2X7R gene in a population of 462 osteoporotic women and men with a T-score less than -2.5 or one low trauma vertebral compression fracture referred to the Department of Endocrinology at Aarhus University Hospital. The effect of various genotypes on fracture risk was examined and factors associated with fracture risk and BMD and/or body weight were found. Again, these findings were in accord with the phenotype of the knockout mouse described by Ke.^{75,80}

The vitamin D system includes 25 vitamin D, its active form 1,25 dihydroxyvitamin D, a variety of enzymes involved in its formation, and a specific receptor, the Vitamin D Receptor (VDR), which mediates its actions.⁸¹ Mutations in the VDR gene are known to cause disorders such as 1,25 dihydroxyvitamin D-resistant rickets, a rare monogenetic disease. Apparently, polymorphisms (more subtle sequence variations) in the VDR gene happen more frequently in the population than the severe deleterious mutations.⁸² In a study of 32 young (age range 19–30 years) stress fracture patients and 32 healthy volunteers, Chazipapas and colleagues genotyped the study subjects for four different polymorphisms of the VDR: FokI in exon 2, BsmI and Apal in intron 8, and TaqI in exon 9. For example, the FokI polymorphism contained FF, Ff, and ff genotypes; stress fractures were found to be eight times more likely in subjects with the ff and Ff genotypes compared to the FF genotype. Similar data were produced for the other polymorphisms. FokI and BsmI polymorphisms were found to be independent risk factors for stress fractures.⁸¹

Korvala et al. looked at the genetic predisposition for femoral neck stress fractures in a group of Finnish soldiers. All military conscripts who had suffered a femoral neck stress fracture between 1970 and 1995 were invited to a follow-up study in 2002 to 2003; 72 subjects participated. The diagnosis of stress fracture had been made based on standard X-ray, nuclear medicine, or MRI criteria. A group of 120 soldiers without stress fractures served as a control population. Clinically, the cases were shorter and had lower body weight and BMI than the controls. A total of 15 SNPs in six genes (COL1A1, COL1A2, CTR, IL-6, VDR, and LRP5) were genotyped. The COL1A1 RS2586488 and COL1A2 rs3216902 SNPs were associated with stress fractures in a recessive model, and the risk was increased in carriers of the LRP5 rs2277268 SNP minor allele in comparison with noncarriers. The authors felt that genetic factors might play a role in the development of stress fractures in individuals subjected to heavy exercise and mechanical loading who were lighter weight, and thus the heavy loads they were subjected to were responsible for the relatively higher numbers of neck stress fracture than in larger individuals.⁸³

Yanovich et al. studied candidate genes in Israeli soldiers with stress fractures. The study population consisted of 203 soldiers (162 males and 41 females) with no findings of stress fractures and 182 soldiers (165 males and 17 females) with known stress fractures. Of interest, 10 participants from the stress fracture group had a family history of bone disorders or stress fractures; 5 reported that their fathers had suffered stress fractures during

their military service. A total of 268 candidate SNPs from 17 genes spanning 12 chromosomes were selected for study. Sequence variants in a total of eight genes were associated with an increased risk for the development of stress fractures. Of note, variants in six genes were associated with decreased risk of stress fractures. One of the limitations of this study was that it may have been underpowered to detect significant differences, because correcting for multiple comparisons resulted in the fact that none of the comparisons remained significant.⁸⁴

Varley and coworkers undertook an evaluation of the role of the RANK/RANKL/OPG pathway, which is important in osteoclastogenesis controlling osteoclast activation, formation, and differentiation, working with a convenience sample of 518 elite athletes (449 males and 69 females) to form the Stress Fracture in Elite Athletes (SFEA) cohort who participated in a variety of sports including soccer, cricket, track and field, running events, rowing, boxing, tennis, hockey, and gymnastics. Each sport produced both stress fracture and nonstress fracture subjects. Genomic DNA came from saliva samples. A SNP of RANKL rs1021188 was shown to be associated with stress fractures in the whole group, the male group, and the multiple stress fracture group. A SNP of RANK rs3018362 was also shown to be associated with stress fracture occurrence in different groups. A rare allele of rs4355801 had a greater association with stress fractures in the OPG group. They concluded that SNPs of the RANK/RANKL/OPG signaling pathway were associated with stress fractures. Although the specific function of the genotyped SNPs was not known, this pathway is associated with osteoclast differentiation and activation that could decrease bone resorption, thus, perhaps, affecting the ability to respond to or repair microdamage.⁸⁵

Additionally, in another study, Varley and colleagues postulated that the P2X7 receptor gene, now recognized as a key regulator of bone remodeling, might play a role in the development of stress fractures both in military recruits and elite athletes. They studied a group of 210 Israeli Defense Force (IDF) military recruits and 518 elite athletes. The elite athletes (449 men and 69 women) formed the SFEA cohort. This cohort was recruited from the United Kingdom and North America and were predominantly white Caucasian (83.2% in the stress fracture cases and 79.9% in the nonstress fracture controls). Both groups had had stress fractures diagnosed in standard ways by complaints, physical examination, and appropriate imaging. From the military participants, DNA was extracted from peripheral blood leukocytes, and from the SFEA cohort, genomic DNA was derived from saliva. Analyses of five P2X7R SNPs in the SFEA cohort showed that specific SNP (designated rs1718119) was associated with multiple stress fractures. Thus, the findings in these two distinct populations (military cohort data not presented) are the first to demonstrate an independent association between stress fracture incidence and specific SNPs (rs1718119 and rs3751143). As described previously, in multiple Scandinavian studies, these SNPs have been shown to affect various bone parameters, e.g., bone loss rate, vertebral compression fracture, etc. The mechanisms by which these SNPs in the P2X7R gene are involved in the production of stress fractures is unknown. However, since it is hypothesized that stress fractures are related to repetitive loading causing microdamage and bone

fragility, the sensitivity to mechanical loading and the expression of mechanotransduction by osteocytes may be at the basis of the pathophysiology.⁸⁶

In 2017, further studies by Varley et al. offered newer data from the previously discussed SFEA cohort. They investigated 11 SNPs in the vicinity of Wnt signaling pathway, especially the SOST gene, which have a role in bone formation and mechanotransduction. By this point in time, 125 stress fractures were reported in the SFEA cohort. Three SNPs in the SOST gene and the VDR gene studied were reported as being associated with increased incidence of stress fracture. Again, at this point in time, the mechanisms by which these SNPs increase the stress fracture risk is not known, but since the SOST gene is involved with regulating bone formation, it is possible that the rare allele of rs1877632 down-regulates sclerostin expression, via its role in inhibiting Wnt signaling, which could result in a reduction in bone formation, thus impairing the response to accumulation of stress fracture microdamage.⁸⁷

RISK FACTORS

In perhaps the largest study of stress fractures, Bulathsinhala et al. looked at racial and ethnic differences in 1.3 million US Army soldiers using the Total Army Injury and Health Outcomes Database (TAIHOD), a large repository of administrative (medical and demographic) data on the entire Active Duty Army (ADA) population. Race origin was Non-Hispanic black, Non-Hispanic white, Hispanic, American Indian/Alaskan Native, Native Hawaiian/Pacific Islander, and mixed races. Race was categorized as Black, White, American Indian, Asian, and More than one race. Ethnicity was also expressed, e.g., Asian consisted of Chinese, Japanese, Korean, Vietnamese, Filipino, Indian, and other Asian. They identified 21,549 incident stress fractures among 1,299,332 soldiers during 5,228,525 person-years. The overall incidence of stress fractures was 4.12 per 1000 person-years from 2001 to 2011. Female soldiers had a 3.6-fold higher incidence of stress fractures than did male soldiers. Non-Hispanic white and Hispanic groups had a higher risk of stress fractures than non-Hispanic blacks. Non-Hispanic white men and women had the highest risk of stress fracture. There was further breakdown of the racial and ethnic groups. The youngest soldiers (<20 years) were more susceptible to stress fractures than older groups, and those with lower weight were at higher risk than those of normal weight. The reasons for these race and ethnic-related risks for stress fractures are unknown, but probably they are related to issues of bone mineral density and bone quality including issues like bone size, bone architecture, and microdamage handling—all issues we now know are probably related to underlying genetics.⁸⁸

For further details of issues about Risk Factors, see Chapters 3, 5 and 28 as well as below.

HISTORY AND PHYSICAL EXAM

The clinical history should make the health care professional (physician, nurse practitioner, and/or physician's assistant) suspect the presence of a stress fracture. The most important diagnostic tool

TABLE 4.5 Intrinsic and Extrinsic Factors in the Causation of Stress Fractures

Intrinsic Risk Factors	Extrinsic Risk Factors
<ul style="list-style-type: none"> • Gender • Age • Ethnicity • Body Mass Index • Bone characteristics • Muscle strength • Pretraining fitness level • Lower extremity morphology • Nutrition factors • Genetics • Menstrual dysfunctions • Muscle fatigue • Flexibility • Previous injury and inadequate rehabilitation 	<ul style="list-style-type: none"> • Training errors • Training surfaces • Worn-out/inappropriate footwear • Excessive training intensity • Environment

Adapted from Rosenthal and McMilan, *Recruit Medicine*, Chapter 11, 2006, ed, Bernard L. DeKoning, Office of the Surgeon General, pp 175–202.

is a detailed clinical history supported by a complete (for the non-surgeon) and/or a focused physical examination (for the orthopedist). Most athletes relate an insidious onset of pain over 2–4 weeks. This is usually associated with the initiation of a training program (e.g., I thought I would run a 5K, a half-marathon, or a marathon), an increase in training regimen (e.g., getting in shape for the start of a season), or a change in equipment (e.g., new running shoes). Utilization of a list or a preset questionnaire (Table 4.5) will help the history-taker be complete and cover more of the important issues. The pain is focal and local as opposed to medial tibial stress syndrome where the pain is more generalized along the anterior medial surface of the tibia.

Initially, the pain occurs only during the offending activity, such as running. At this point, suspicion must be high to make the diagnosis. Usually, the athlete notices pain at the end of an event but typically the pain subsides quickly with cessation of the activity; then, over the next several days to weeks, the pain progresses to occur earlier in the event and becomes more severe, although frequently the athlete is trying to play through the pain. The pain then increases to the point where it persists for a prolonged period of time after the event and, eventually, starts to occur between events, then extends to occur between events without an obvious precipitating activity, and ultimately to pain at rest. Throughout this progression, there is a decrease in mileage or in time spent playing the activity, like basketball. At this point, when the athlete is finally unable to perform, he or she may complain to the trainer, other staff members, or another health care professional.¹¹

Physical examination, at this point, is usually focused on the site of pain. Often the patient can point to the site of the pain, especially in the lower extremity, and one can find local tenderness to palpation or possibly slight nodular swelling. In Matheson et al.'s series of 320 athletes, localized tenderness was found in 65.9% of cases and swelling in 24.6%.⁸⁹ Milgrom and their group conducted a clinical assessment of femoral stress

fractures in a prospective study of 372 male infantry recruits. If the response to the stress fracture history was positive, a complete physical examination was performed; each bone in the lower extremity was examined by palpation to determine if tenderness was present. “The femurs, because they lie within a large cuff of muscles were examined for tenderness by a ‘Fist Test.’ That is, pressure was applied simultaneously to the anterior aspect of both thighs, directly over the femurs, beginning distally and progressing stepwise proximally. This was done with the clenched fists of the examiner applying the weight of his upper body. An area of specific tenderness difference in sensitivity between femurs using the Fist Test was considered suggestive of a femoral stress fracture.” By using this expanded stress fracture clinical assessment (SFCA) and employing the full upper-body weight of the examiner, they uncovered more previously asymptomatic femoral stress fractures to more appropriately classify them as symptomatic.⁹⁰ Giladi and their group also looked at external rotation of the hip as a predictor for stress fractures. Each of the group of 295 new male infantry recruits between 18 and 20 years of age who were evaluated in this study underwent a pre-basic training screening that included an extensive orthopedic examination with measurements of joint motion including the range of internal and external rotation of the hip with the hip flexed to 90°, among other measurements and assessments for ligamentous laxity. External rotation of the hip was found to have a significant relationship to all types of stress fractures ($p = 0.0163$), and specifically tibial stress fractures ($p = 0.0345$), but not for femoral stress fractures. They divided the external rotation into two categories—external rotation $\geq 65^\circ$ and $< 65^\circ$ —and found that the recruits with an external rotation $\geq 65^\circ$ had a 1.8 times higher incidence of stress fractures than the $< 65^\circ$ group. They hypothesized that the $\geq 65^\circ$ group might represent those with retroverted hips, increased joint laxity, a different gait pattern, or different collagen characteristics of their bone.⁹¹

Matheson and colleagues found alignment and biomechanics of the lower extremities are significant factors in the causation of stress fractures. The frequency of varus alignment was reviewed: genu varum 29%, tibial varum 18.9%, subtalar varus 71.9%, and forefoot varus 72.6%. Pronated feet were most common in tibial and tarsal stress fractures and least common in metatarsal stress fractures. Cavus feet were found most commonly in metatarsal and femoral stress fractures.⁸⁹ All of these alignment and biomechanical abnormalities create gait difficulties, and some are quite subtle. Therefore, sometimes an individual with a stress fracture or, more likely, multiple stress fractures or stress reactions or a combination of the two may be well served to have an evaluation at a human performance laboratory, including gait analysis.

Three-dimensional instrumented gait analysis (3D-GA) results in information on normal and pathological gait to provide comprehensive data about joint motions (kinematics), time-distance variables (spatio-temporal data), and joint movements and powers (kinetics). 3D-GA can be helpful for obtaining objective information for analysis of functional limitation or for follow-up over time. A number of indices have been developed including: normalcy index (NI), hip flexor index (HFI), gait deviation index (GDI), gait profile score

(GPS), and movement analysis profile (MAP). The NI is the most extensively validated and used measurement in clinical gait research and practice.⁹² Napier and colleagues conducted a systematic review of gait modifications that have been undertaken to change lower extremity gait biomechanics in runners. Several measures including rearfoot eversion, vertical loading rate, and foot strike index have shown an association with running-related injuries. Some of the biomechanical issues, such as cadence and foot strike, may be modifiable. They found 27 articles that investigated different gait-retraining interventions. Foot strike manipulation was the most common intervention; step frequency and step length were also common interventions. Some studies looked at other manipulations. They covered changes in hip kinematics, changes in knee kinematics, changes in ankle kinematics, vertical, and leg/lower extremity stiffness, spatiotemporal variables, step frequency, step length, and ground contact time. Impact loading (the sudden force applied to the skeleton at initial contact) has demonstrated the greatest relationship with lower extremity overuse injuries from any of the biomechanical variables.⁹³

Zadpoor and Nikoyan⁹⁴ conducted a metaanalysis of 13 articles of the relationship between lower extremity stress fractures and the ground reaction force (GRF). The GRF is an approximate measure of the loading of the lower extremity musculoskeletal system, is fairly easy to measure, and is an important feature to measure in the study of the kinetics of the lower extremity during running. The vertical loading rate (VLR) is defined as the slope of the initial part of the vertical-GFR time curve (between the foot strike and the vertical impact peak). According to the authors' analysis of the included literature, the studies do not agree on whether or not the vertical GFR and/or loading rate are significantly different between the stress fracture groups and the control groups. However, the average VLR and the instantaneous VLR are significantly higher in the stress fracture group ($p < 0.05$). One of the limitations the authors state of the cited studies was that they were only short-term studies, and many individuals with lower extremity injuries are running for a long time, and consequently, muscle fatigue may play a role in their injuries. When muscles fatigue, the amount of energy transmitted to the surrounding bones increases. Grimston and colleagues from the Human Performance Laboratory at the University of Calgary showed, in a study of a 45-minute run in subjects with a history of a tibial stress fracture ($n = 5$) and no stress fracture history ($n = 5$), maximum lateral forces were significantly greater for the stress fracture subjects during both early and late stages of the run compared to nonstress fracture subjects. "This finding of increased loads during the course of a 45 min run in SF, and constant or decreased loads in NSF, may be indicative of differences in fatigue adaptation and warrants further study."⁹⁵

Of the different measurements used to test impact loading, average vertical loading rate (AVLR) is the most serious running-related injury risk factor. There is a link between step frequency, step length, and ground contact time. Typically, a greater step length and ground contact time has been associated with a higher incidence of stress fracture. Edwards and his group created a probabilistic stress fracture model based on the effects of stride length and running mileage. They investigated two stride

lengths (preferred and -10% preferred) and three running regimens (3, 5, and 7 miles) in 10 experienced male runners free of any lower extremity injuries. A 10% reduction in stride length resulted in a corresponding reduction in peak resultant contact force. Increasing running mileage from 3 to 5 miles resulted in an increase in stress fracture probability of 4% to 5% . Increasing running mileage from 3 to 7 miles increased stress fracture probability from 7% to 10% . Their results suggested that a 10% reduction in preferred stride length reduces the risk for a tibial stress fracture, and that if this were done, it would allow runners to run an additional 2 miles per day and maintain the same low risk of fracture. They also felt that the benefits of reduced stride length are noticed more at higher weekly running mileages. The authors stated that the "difficulty for the clinician is in identifying those runners 'at risk' for stress fracture that would benefit from a 10% stride length reduction. Presumably, these would be inexperienced runners beginning a weekly running routine or runners with a history of stress fracture. Poor physical fitness and low physical activity before physical training and a previous history of stress fracture are both associated with a higher risk of stress fracture development."⁹⁶

Crowell and Davis studied gait retraining to reduce lower extremity loading in runners. They performed a pretraining instrumental gait analysis. They then began a retraining program, which included eight sessions over a 2-week period in which an accelerometer was taped to the distal tibia and subjects ran on a treadmill, during which time they were instructed to "run softer": make their footfalls quieter and to keep their acceleration peaks below a given line. The monitor depicting the peak line was placed in front of the treadmill for the runners to view. Run time was gradually increased from 15 to 30 minutes over the eight sessions. Feedback was provided continuously for the first four sessions and then removed. A comparison of the pretraining and posttraining results revealed significant reductions in peak positive acceleration (PPA), vertical instantaneous (VILR), and vertical average loading rates (VALR), and a vertical impact peak (VIP) of about $20\text{--}30\%$. These reductions were maintained at the 1-month follow-up. The reductions in PPA, VILR, VALR, and VIP achieved in the current study were at least two times greater than those achieved through the use of cushioning shoes, foot orthoses, or shock-attenuating insoles, indicating that an individual's ability to alter their own running mechanics is greater than the ability of any of these external devices to assist them. So, lower extremity impact loading can be reduced with a gait retraining program that uses real-time visual feedback.⁹⁷ But most of the gait retraining studies have taken place in the laboratory and not in the natural setting, like a track or cross-country course, a marathon course, or a basketball court.⁹⁸

Napier and his colleagues concluded, from their meta-analysis, that gait retraining works in the short term to produce small to large effects on kinetic, kinematic, and spatiotemporal results during running. Foot strike changes had the greatest effect on kinematic measures, and real-time feedback also had its largest change on kinetic measures, whereas combined training protocols had the biggest alteration on spatiotemporal measures. Further research on these and other interventions is still needed.⁹³

BONE DENSITOMETRY

Bone density testing, in its present form, has been available for about 30 years.⁹⁹ Initially developed in 1961 by Cameron, and improved upon by Cameron and Sorensen,¹⁰⁰ this early device measured cortical bone in the forearm by single photon absorptiometry (SPA). The first commercial devices were manufactured by Norland Corporation (now Norland at Swissray, Fort Atkinson, WI, USA). Subsequently, Mazess and others developed dual photon absorptiometry (DPA) in the early 1970s. The modern era of bone densitometry was ushered in by the development of dual-energy X-ray absorptiometry (initially DEXA; now, DXA) in 1986 by Hologic, Inc., (Waltham, MA, USA) and subsequently again by Mazess at Lunar Radiation Corporation (now GE Healthcare, Madison, WI, USA). Over the last 30 years, DXA has emerged as the primary clinical device for fracture risk assessment, for monitoring changes in bone health or for diagnosing osteoporosis before or after a fracture has occurred. The International Society for Clinical Densitometry (ISCD), through its Consensus Development Conferences, has expanded on the criteria for the diagnosis of osteoporosis and developed positions of official ROIs for diagnosis and monitoring and principles of clinical monitoring changes in bone density over time.^{101–104}

Although the vast majority of clinical DXA centers perform a minimum study of AP Spine (L1–L4), and one hip (measuring regions of interest [ROIs] that are known as the Total Hip and the Femoral Neck), the densitometers have developed into sophisticated tools that can measure multiple parameters of musculoskeletal health.⁹⁹ Many of the early papers on bone densitometry in the area of stress fractures dealt with female athletes, including those with the Female Athlete Triad.

Perhaps one of the first uses of bone densitometry in athletes was the report by Drinkwater et al. in 1984 on the bone mineral content (BMC) of amenorrheic (age 24.9 ± 1.3 years) and eumenorrheic athletes (25.5 ± 1.4 years).¹⁰⁵ Twenty-eight women athletes, 14 of them amenorrheic, were studied, and the 14 eumenorrheic controls were selected from a larger pool of potential subjects. SPA and DPA were used to measure forearm bone mineral density (BMD) at two sites at the one-tenth and one-fifth forearm sites and lumbar spine BMD of L1–L4, respectively. Although the forearm bone density did not differ at either site between the two groups, the BMD of the lumbar vertebrae was significantly lower in the amenorrheic group of athletes. Before the World Health Organization classification was developed,¹⁰⁶ the BMD of the amenorrheic athletes was said to be equivalent to that of women 51.2 years of age; two of these athletes had a vertebral mineral density below the “fracture threshold” as defined by Riggs et al., which is 0.965 g/cm^2 .¹⁰⁷

In a follow-up study, Drinkwater and her group reported on seven of the original amenorrheic women who regained their menses within 1–10 months after 40.4 months (range 11–86 months) of amenorrhea. Even though the miles run per week by the amenorrheic group was higher than the eumenorrheic group, their mileage had decreased by 10%, their weight had increased by 1.9 kg in the women who regained their menses, and they had an increase in calcium intake. The bone density increase was 6.2% in 14.4 months.¹⁰⁸

In 1990, Myburgh and colleagues showed that low bone density was an etiologic factor for stress fractures in athletes. In their study, 25 athletes with stress fractures were recruited from the University of Cape Town Sports Injury Clinic during a 1-year time interval. They were compared with a group of control subjects who matched the injured athletes closely. Bone mineral density was measured by DXA (Hologic QDR 1000) at the lumbar spine (L2–L4) and the left hip (including the left total femur, Ward [sic] triangle, femoral neck, greater trochanter, and the intertrochanteric space), although now, per ISCD Official Positions the Ward’s triangle, greater trochanter and intertrochanteric space are not “official” ISCD ROIs. In subjects with a history of a left femoral neck or shaft stress fracture, the contralateral side was measured, and vice versa. All measurements were made at least 6 months after the diagnosis of stress fracture was made and when all previously injured subjects were exercising regularly. Of the 25 injured athletes and 25 control athletes, 32 participated in road running, 2 in track, 4 in aerobics, and 12 in both aerobics and running. Of the 50 subjects, 38 were women. They were matched for age (32 ± 8 years), body mass, and height. Injured runners and their controls were matched for average training distance (53 ± 27 and $45 \pm 17 \text{ km/wk}$). Nonrunners and their controls had similar training times (6 ± 4 and $5 \pm 2 \text{ h/wk}$). In the 25 athletes with stress fractures, 7 fractures occurred in the foot, 6 in the femoral neck, 3 in the pubic rami, 3 on other areas of the femur, 4 in the tibia, and 2 in the fibula. Of the 25 injured subjects, 7 had previous shin splints and 5 had a history of one or more previous stress fractures. Focusing on the bone density results only, for the purposes of this chapter, bone mineral density was lower in injured subjects than in control subjects in the lumbar and the proximal femur ($p < 0.02$, each, respectively). The six subjects with femoral neck stress fractures had significantly lower femoral neck bone density than the matched controls. Other clinical risk factors were also studied but are not reported here. The authors felt that the most important finding was that athletes with stress fractures had lower bone mineral densities than did well-matched control athletes. They also showed that the lower bone mineral density occurred in both the axial and appendicular skeleton. The authors concluded that “evidence suggests the etiology of stress fractures in athletes is more complex than traditionally believed.”¹⁰⁹

Giladi, Milgrom, and their colleagues reviewed identifiable risk factors for stress fractures in the Israeli Defense Force. They studied a group of 312 male military recruits during 14 weeks of basic training; after dropouts, 289 soldiers were studied. As part of the pretraining evaluation, foot and tibial radiographs were obtained to measure tibial bone width, BMC was measured by SPA at 8 cm above the ankle mortise, and BMD was measured by a Compton bone densitometer, which was a precursor of quantitative computed tomography (QCT), measuring the bone density (in grams per cubic centimeter) for a cancellous window in the center of the tibia. The main bone-related risk factor found was: soldiers with wider tibia sustained less tibial, femoral, and total stress fractures than those with narrow tibia.¹¹⁰

Lauder et al. performed an early study on the relationship between stress fractures and bone mineral density in active-duty US Army women at Fort Lewis, WA, with a total of 423 subjects of which 190 women were available for the BMD evaluation study; 30 of these women qualified by having one or more stress fractures in the last 2 years. Five women were excluded for a variety of invalid entries on some data items, producing a study population of 185 women. Of the 185 women, 27 had stress fractures, and 158 women without stress fractures were used as controls. An extensive evaluation of demographics and risk factors for stress was undertaken. BMD of the PA lumbar spine (L2–L4) and femoral neck was measured on all subjects by DXA using a Lunar DPX (now GE Healthcare) by a trained technician. Their multivariate analysis revealed a strong inverse relationship between femoral neck BMD and the probability of a stress fracture as their most significant finding, indicating that lower levels of femoral neck BMD were associated with an increased likelihood of stress fracture. BMD of the lumbar spine was not found to be a significant predictor of stress fractures.¹¹¹

Marx and colleagues from the Hospital for Special Surgery looked at stress fracture sites as they are related to underlying bone health in athletic females.¹¹² They noted that the most commonly described sites for stress fractures are cortical ones, including the tibia, the metatarsals, and the femoral shaft, whereas fractures are less common at sites of trabecular (or cancellous) bone, such as the femoral neck, pelvis, and sacrum. They felt that patients who had stress fractures of trabecular bone sites had lower bone mineral density than those who had cortical bone stress fractures at their Women's Sports Medicine Center. They conducted a retrospective chart review of 65 patients diagnosed with stress fractures over a 4-year period. Patients underwent DXA scanning. They did not describe the type of DXA machine or software versions used in their study. They did state that 15 of the DXA scans were performed at their institution and 5 at other locations, so presumably they were different machines from different manufacturers. (At that time, some of the individual machine differences were not understood as clearly as they are today).^{101–104} They utilized the World Health Organization definition of osteopenia, which had been recently formulated.¹⁰⁹ This definition has also been refined.^{101,102} Because of study patients eliminated for a variety of reasons, they had DXA scans of 9 patients with stress fractures of trabecular bone sites and 11 with stress fractures of cortical bone sites. Using this small population, they found that stress fractures of trabecular bone sites were associated with “early onset osteopenia ($p = 0.01$).” Eight of the nine patients with stress fractures at trabecular sites had DXA scans that indicated osteopenia, while only three of the patients with cortical bone site stress fractures had osteopenia. They concluded a trabecular bone site stress fracture in a young female might be a warning sign of “early onset osteopenia.” They recommended that females under age 40 who have documented stress fractures of either trabecular or cortical bone sites (with risk factors for osteopenia) undergo bone density evaluation. (Current ISCD Official Positions would refer to these DXA measurements as “below the expected range for age” if the Z-score is ≤ -2.0 and “within the expected range for age” if the Z-score is > -2.0).¹¹³)

With further technologic development of DXA and sophistication of our understanding of the use of the tool, additional ROIs are available for study, like the forearm (usually, the nondominant forearm 1/3 radius site, which is an Official ISCD site)¹⁰⁴, vertebral fracture assessment (VFA), and whole body bone mass (also known as total body bone mass [TBBM])¹¹⁴ from which body composition measurements can be determined with percent body fat and lean and fat mass.^{101,102}

This newer understanding of DXA and the ISCD definitions of an abnormal DXA for children, adolescents, and premenopausal women has been incorporated into the latest 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad.^{113,115} That panel utilized the definitions published by the ISCD as well as the ACSM criteria for female athletes involved in regular weight-bearing sports¹¹⁶ (Box 4.2).

Looking at another role for bone densitometry in the athletic population, Gustavsson and her colleagues studied rapid bone loss of the femoral neck after cessation of ice hockey training over 6 years in young males. They assessed the effects of training and detraining on the BMD of the total body, spine, and femoral neck in a cohort of adolescent male hockey players in Sweden. The study group initially consisted of 65 ice hockey players and 30 controls with a mean age of 16.7 ± 0.6 years and 16.8 ± 0.3 years, respectively. After a mean period of 2.5 years, 59 hockey players and 30 controls agreed to participate in a first follow-up session; 12 of the ice hockey players had stopped training and were excluded; one of the controls was excluded for a variety of extraneous medical reasons. After a mean period of 5 years and 10 months, 22 active and 21 retired hockey players and 25 controls participated in a second follow-up examination. At baseline, the average training per week for the hockey players was 9.4 ± 2.6 hours. The training consisted of ice hockey training or games, with additional weight and aerobic training. The control group's physical activity was playing soccer and football, distance running, and some weight training. At the start of the study, all boys participated in 2 hours of physical education in school each week. The subjects were divided into different pubertal Tanner stages; all were judged to be at least Tanner stage 4. Using a Lunar DPX-L (now GE Healthcare) bone densitometer, they measured the total body and spine BMD and BMC and area of the right femoral neck at baseline and the two follow-up examinations. The authors felt their most important finding was the effect of detraining on the femoral neck BMC in the retired players. These retired players lost significantly more bone at the femoral neck ROI between 19 and 22 years of age than the ice hockey players who continued their training. Thus, the gain in BMD from training effect is not sustained after cessation of training.¹¹⁷

Studies by Davey et al. carried the differences in axial and appendicular bone density in stress-fractured and uninjured royal marine (RM) recruits even further. According to the authors, RM training is widely acknowledged as one of the most arduous and longest (32 weeks) military training programs in the world. In their study, they measured BMD by DXA, by ultrasound, and by peripheral quantitative

BOX 4.2 Low Bone Mineral Density (BMD) Diagnosis

How is Low BMD Diagnosed?

The Panel has utilized the definitions published by the International Society of Clinical Densitometry (ISCD) for low BMD and osteoporosis in children and adolescents and for premenopausal women and adolescents and for premenopausal women, as well as ACSM-suggested criteria for female athletes involved in regular weight-bearing sports. Criteria are described below for who and what site should be considered for a DXA scan and how often DXA should be performed.

Who Should Get DXA Scans for BMD Testing?

The Panel agreed that indications for obtaining a DXA scan for BMD testing in an athlete should follow Triad risk stratification (see Clearance and Return to Play section) and include the following:

(1) ≥ 1 "High risk" Triad Risk Factors:

- History of a DSM-V-diagnosed eating disorder
- BMI ≤ 17.5 kg/m², $< 85\%$ estimated weight, OR recent weight loss of $\geq 10\%$ in 1 month
- Menarche ≥ 16 years of age
- Current or history of < 6 menses over 12 months
- Two prior stress fractures, 1 high risk stress fracture, or a low-energy nontraumatic fracture

Prior Z-score of < -2.0 (after at least 1 year from baseline DXA)

OR

(2) ≥ 1 "Moderate risk" Triad Risk Factors:

- Current or history of DE for 6 months or greater
- BMI between 17.5 and 18.5, $< 90\%$ estimated weight, OR recent weight loss of 5% to 10% in 1 month
- Menarche between 15 and 16 years of age
- Current or history of 6 to 8 menses over 12 months
- One prior stress reaction/fracture
- Prior Z-score between -1.0 and -2.0 (after at least 1-year interval from baseline DXA)

- (3) In addition, an athlete with a history of ≥ 1 nonperipheral or ≥ 2 peripheral long bone traumatic fractures (nonstress), should be considered for DXA testing if there are 1 or more moderate- or high-risk Triad risk factors. This will depend on the likelihood of fracture given the magnitude of the trauma (low or high impact) and age at which the fracture occurred. Athletes on medications for 6 months or greater that may impact bone (such as depot medroxyprogesterone acetate, oral prednisone, and others) should also be considered for DXA testing.

How Often Should Athletes Get DXA Testing?

The Panel agreed that the frequency of BMD assessment by DXA will depend on the initial BMD and ongoing clinical status of the athlete. We agree with the ISCD 2013 guidelines that repeat DXA screening should be obtained when the expected change in BMD Z-scores equals or exceeds the least significant change. Those with definitive indications for DXA testing may require BMD testing every 1 to 2 years to determine if there is ongoing bone loss, and to evaluate treatment.

Which Sites Should be Screened with a DXA Scan?

Bone mineral density Z-scores (and not T-scores) should be reported for all children, adolescents, and premenopausal women.

(1) Adult women ≥ 20 years

Weight-bearing sites (posteroanterior spine, total hip, femoral neck)

Nonweight-bearing sites, namely the radius (33%) if weight-bearing sites cannot be assessed for any reason.

(2) Children, adolescents, and young women < 20 years

Posteroanterior lumbar spine bone mineral content (BMC) and areal BMD

Whole body less head if possible (otherwise whole body) BMC and areal BMD

Adjust for growth delay (with height or height age) or maturational delay (with bone age)

Use pediatric reference data, and when possible, report height-adjusted Z-scores.

Data from De Souza MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. *British Journal of Sports Medicine* 2014;48:289.

computed tomography (pQCT). In their cohort of 1090 recruits, 78 (7%) developed one or more stress fractures; 62 pairs of stress-fractured recruits and controls were assessed with DXA; the 62 fractured recruits had 79 stress fractures; 7 recruits had 2 fractures, 3 had 3 fractures, and 1 had 4 fractures. The most common sites of fracture were the metatarsals ($n = 41$) and tibia ($n = 26$). Areal BMD assessed with DXA (two-dimensional projection) was lower at all sites in the stress fracture group compared with the control group ($p < 0.01$). Although they used T-scores (the group of males were aged 16–32 and Z-scores should have been utilized), they did state that 28 of the 62 had T-scores "below the normal range (T-score < -1.0)" but, if Z-scores had been appropriately used, per ISCD, these would have been classified as "normal for age" since we do not know if any had Z-scores < -2.0 , which would have been classified as "below normal for age." There was no difference in ultrasound measured by broadband ultrasound attenuation (BUA) of the dominant or nondominant foot at baseline (week 2) between stress fractures and control recruits. In the recruits measured by pQCT, there were 51 pairs for the first three slices of the tibia (4%, 14%, and 33%) and 43 pairs for the 66% slice of the tibia.

There were structural differences between stress fractured recruits and controls at all slices of the tibia, with the 38% slice the most marked difference between the groups; there was also a strong negative correlation between cross-sectional area of the tibia and BMD at this site. There were no differences in the serum C-Telopeptide (s-CTX), a marker of bone resorption, between the stress fracture cases and controls. They concluded that stress-fractured young RM male recruits undergoing specialized prolonged military training had a lower BMD of the spine and hip, narrower tibiae, and reduced tibial strength indices compared with the study controls.¹¹⁸

Edmondson and Schwartz reviewed non-BMD DXA measurements of the hip.¹¹⁹ These included hip axis length (HAL), hip structural analysis (HSA), and finite element analysis (FEA), among other techniques. Beck and colleagues utilized an additional DXA technique that involved scanning at both the mid thigh (midfemur length) and distal third of the lower leg (one-third tibial length from the medial malleolus). With this DXA technique, they prospectively followed 626 US Marine Corps recruits for 12 weeks of basic training to study cross-sectional geometric properties of the midshaft femur and middistal

tibia. Previous studies had shown that the most important geometric properties of a long bone are the cross-sectional area (CSA) and, for bending and torsion, the cross-sectional moment of inertia (CSMI). Within a bone under a given load, the stress forces are determined by the bone structural geometry, while the bone's ability to resist these forces is defined by the bone material properties. The CSA is an index of axial strength and is related to shear strength, while the CSMI is an index of bending rigidity. The section modulus (Z), an index of bending strength, was also calculated as the CSMI divided by half bone width. Twenty-three of the 626 recruits (3.7%) presented with 27 lower extremity stress fractures. The most common site was the tibia (n = 11), metatarsals (n = 7), femur (n = 5), and tarsals (n = 4); two recruits had fractures at two sites and one had fractures at three sites. Most anthropometric dimensions were significantly smaller in the stress-fractured recruits than in normal individuals. Small body size and narrow long bone diaphysis relative to body size were risk factors for the development of lower limb stress fractures during the 12-week basic training program. CSA, CSMI, Z, and pelvic and knee width were significantly smaller than normal in the tibia and femur. After correcting for body weight differences, CSA, CSMI, Z, and pelvic and knee width as well as BMD were all significantly smaller in the fractured recruits. The authors stated that "bone structural data derived from DXA provides important new information that may be useful in the identification of subjects at higher risk for stress fractures under intense physical training conditions."¹²⁰

Nattiv and colleagues studied the correlation of MRI grading of bone stress injuries with clinical risk factors and return to play in a 5-year prospective study in collegiate track and field athletes. In their study, DXA exams were performed at baseline and annually. Athletes with a higher MRI grade injury exhibited a lower BMD at the total hip ($p < 0.050$) and radius ($p < 0.047$). Those athletes with bone stress injuries at trabecular sites had significantly lower bone mass at the lumbar spine, femoral neck, and total hip ($p < 0.001$).¹⁷

Other imaging modalities, such as ultrasound measurements of the calcaneus, have also been employed in studying the bone of athletes.¹²¹

We utilize bone densitometry on every patient we see at our center, not to make the diagnosis of stress fracture, but to help us differentiate between those individuals with fatigue fractures and those with insufficiency fractures. Thus, at the Northern California Institute for Bone Health, Inc., which has both Lunar Prodigy Advance (GE Healthcare, Madison WI, USA) and Hologic Discovery A (Hologic, Inc., Waltham, MA, USA) bone densitometers, we perform a comprehensive DXA study including the following ROIs: PA Spine (L1–L4), both hips (for total hip and femoral neck), nondominant forearm (1/3 forearm [radius]), VFA, a screening of the PA and lateral spine from T4 to L4, TBBM, which allows analysis of lean body mass, fat mass, and percent body fat. We particularly use TBBM in athletes less than 30 years old because they may not have achieved peak bone mass and in female athlete triad and individuals with eating disorders, even if they are older than 30 years of age. There is increasing interest in the use of TBBM in National Football League (NFL, USA) players^{122,123} and in other athletic settings.¹¹⁴

BONE QUALITY

Since the NIH Consensus Development Panel on Osteoporosis Prevention, Diagnosis and Therapy was formed in 2000,¹²⁴ the concept that osteoporosis is a result of compromised bone strength has evolved. Both low BMD and micro-architectural deterioration of bone tissue (bone quality) combine to lead to increased bone fragility and consequent increase in fracture risk. It seems the same principles must apply to the pathophysiology of stress fractures and increased fracture risk in other metabolic bone diseases, that is, they result from a combination of bone mass and bone quality issues. Sorting out the role of these issues in the individual patient is increasingly possible.

Interest in bone quality has grown significantly in the last several decades as investigators have realized some of the limitations of DXA: 1) many fractures occur among patients with normal bone density; 2) fluoride treatment did not reduce fractures despite large increases in spinal BMD; 3) small changes in areal BMD in patients treated with antiresorptive therapy result in greater than expected decreases in future fracture risk; 4) the measured changes in BMD in patients treated with antiresorptive drugs explain only a small part of the variance in the reduction of future fracture risk; 5) reductions in future fracture risk are evident long before early maximal changes in BMD are expected or can occur; and 6) patients receiving glucocorticoid therapy for a variety of illnesses have more fractures than individuals of the same BMD who have not received glucocorticoid therapy. Contributors to bone quality include, among others, trabecular architecture, the rate and extent of bone turnover, the organic and inorganic composition of the bone matrix, the type and amount of collagen crosslinks, the degree of matrix mineralization, microdamage accumulation, and cell viability.^{125,126} Issues of bone quality have been well studied in the bone fragility of osteoporosis^{127,128} (Fig. 4.2). Tommasini et al. looked at the relationship between bone morphology and bone quality in male tibias and their implications for stress fracture risk. Having a narrow (i.e., slenderer) tibia relative to body mass, an aspect of bone geometry, is a predictor of stress fracture risk and bone fragility in male military recruits and male athletes. This was assessed by testing the biomechanical properties of tibias

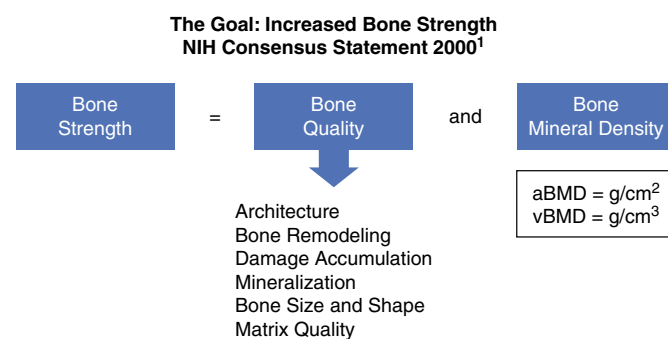


Fig. 4.2 Increased Bone Strength (From NIH Consensus Development Panel on Osteoporosis Prevention, Diagnosis, and Therapy. Osteoporosis prevention, diagnosis, and therapy. *JAMA*. 2001;285(6):785-795. <https://doi.org/10.1001/jama.285.6.785>)

from young adult males. Tibias of 17 male donors (15 white, 1 Hispanic, 1 black, 32.9 ± 10.4 years of age; range 17–46 years) were acquired from the Musculoskeletal Transplant Foundation (Edison, NJ, USA). Extensive whole bone morphology was studied; CSMI, CSA, and Z were assessed. A slenderness index (S) was calculated; an inverse ratio was created so that a tibia with a large S was thinner for weight and height of the individual; and a small S reflected a heavier or larger tibia. Cortical bone samples were prepared from the diaphysis of each tibia for biomechanical testing. Tissue-level mechanical properties and damageability were assessed. There were significant correlations between tibia morphology and mechanical properties in tissue brittleness and damageability. Narrower bone was made up of tissue that failed in a more brittle way and accumulated more damage. Positive correlations were observed between measures of bone size and measures of tissue ductility, and negative correlations were observed between bone size (CSMI and Z) and tissue modulus. “The correlation between tissue ductility and bone size may help explain why male military recruits and male athletes with narrow bones show a higher incidence of stress fractures compared with individuals with wide bones.” “The data provide a new paradigm that may explain how variation in bone slenderness contributes to stress fracture risk.” Narrower tibias were composed of tissue that was more brittle and prone to accumulate more damage compared with tissue from wider tibias. “Having tissue that is more or less damageable may be inconsequential during day-to-day activities. However, tissue-level mechanical properties like total energy and ductility become particularly important in defining the response of bone to an extreme loading condition, such as that expected during military training...” From this study, it is now clearer as to why bone size is a risk factor for stress fractures.¹²⁹

The search for how to measure bone quality clinically has been like the search for the Holy Grail (or the Holy Chalice).^{130–132} But in the last several years new tools (e.g., Trabecular Bone Score [TBS] [Medimaps Group, Geneva, Switzerland] and Osteoprobe [ActiveLife Scientific, Santa Barbara, CA, USA]) have become available to enable insights into bone quality.

Trabecular Bone Score (TBS)

TBS is a software program that is an add-on to the DXA software in the densitometer database. It measures the texture parameter that evaluates the pixel gray-scale variations in the DXA images of the lumbar spine. The TBS variations may reflect bone microarchitecture, and thus TBS has become a surrogate for aspects of bone quality. It uses the experimental variogram of 2D projection images. The TBS is calculated from unprocessed raw computer data from the DXA acquisition. TBS is based on X-ray absorption by tissues, similar to BMD computation. Calculation of TBS and BMD is done separately and by different methods. TBS is derived after the BMD measurement is made and at the same region of interest (PA spine). TBS is a unitless measurement. A high TBS value indicates “good” microarchitecture associated with “good” mechanical strength and a reduced fracture risk; a low TBS value indicates poor-quality microarchitecture and, therefore, increased fracture risk.¹³³

TABLE 4.6 Trabecular Bone Score (TBS) Interpretation

HOW IS THE NUMBER INTERPRETED?	
BMD	TBS
Normal T-score > -1	Normal TBS > 1.350
Low bone mass -1 < T-score < -2.5	Partially degraded 1.200 < TBS < 1.350
Osteoporosis T-score < -2.5	Degraded TBS < 1.200

BMD, bone mineral density; TBS, trabecular bone score.

From <http://www.medimaps.fr/tbs-insight>

Analogous to the World Health Organization (WHO) classification of osteoporosis of normal, low bone mass (osteopenia), and osteoporosis,¹⁰⁶ the TBS values for postmenopausal Caucasian women was initially established with the advent of the software: TBS ≥ 1.350 is “normal;” TBS between 1.200 and 1.350 is considered to be associated with “partially degraded” microarchitecture; and TBS ≤ 1.200 is classified as “degraded” microarchitecture (Table 4.6).

These cutoff thresholds were established by a working group of TBS users from different countries.¹³⁴ McCloskey et al. conducted a meta-analysis of TBS in fracture risk prediction. This study resulted in a change of the classification thresholds so that a TBS value < 1.23 is “degraded;” between 1.23 and 1.31 is “partially degraded;” and > 1.31 is “normal.” There was no difference between sexes.¹³⁵ At this point in time, the FDA has approved only a postmenopausal Caucasian database, although other gender- and race-specific databases are available: female and male, White; male and female, Black; and male and female Mexican American (personal communication). We use these additional databases in our research.

Most of the studies reported have dealt with older populations, as the primary use of TBS has been in establishing fracture risk in the osteoporotic population.^{136,137}

In 2015, Silva et al. reviewed the literature on fracture risk prediction by TBS for the International Society for Clinical Densitometry (ISCD) and established its official positions: 1) TBS is associated with vertebral, hip, and major osteoporotic fracture risk in postmenopausal women; 2) TBS is associated with hip fracture risk in men greater than the age of 50 years; and 3) TBS is associated with major osteoporotic fracture risk in men older than the age of 50 years. Thus, the official positions applied to an older population.¹³⁸

Over the last several years, there have been reports of the responses of TBS to various osteoporosis antiresorptive and anabolic medications and other disease states. Sean et al. reported on the results of teriparatide (Forteo) and ibandronate (Boniva) on spine BMD and TBS in 210 postmenopausal women with osteoporosis (70 treated with teriparatide 20 μ g self-injected subcutaneously daily versus 140 treated with intravenous ibandronate 3 mg every 3 months) who were 68.9 ± 9.0 years of age versus 67.4 ± 6.5 years of age, respectively. Only women with evaluable DXA scans for both LS BMD and TBS at baseline and after 2 years were included in the analysis; this made the final

numbers 65 (93%) and 122 (87%) in the teriparatide and ibandronate groups evaluable, respectively. Both groups started with TBS of 1.206 ± 0.100 versus 1.209 ± 0.100 , respectively, both in the “degraded” class. After 24 months of therapy, lumbar spine BMD and TBS increased significantly more with teriparatide compared with ibandronate ($+7.6 \pm 6.3\%$ versus $+2.9 \pm 3.3\%$ and $+4.3 \pm 6.6\%$ versus $+0.3 \pm 4.1\%$, respectively; $p < 0.0001$ for both). Compared to baseline, increases in BMD were significant for both teriparatide and ibandronate, while increases in TBS were significant only for those treated with teriparatide ($p < 0.0001$), suggesting a stronger positive effect on bone microarchitecture with teriparatide.¹³⁹ Saag et al. reviewed the results for TBS obtained from archived DXA reports from a randomized clinical trial of patients with chronic glucocorticoid therapy-induced (median 7.5 mg/d prednisone for ≥ 90 days) osteoporosis treated with alendronate or teriparatide. In patients treated with teriparatide, TBS was significantly increased from baseline at 18 and 24 months and increased by 3.7% at 36 months; in patients treated with alendronate (Fosamax), TBS did not change significantly at any time from baseline to 36 months.¹⁴⁰ Bilezikian et al. analyzed the effects of subcutaneous abaloparatide on TBS in a post hoc retrospective analysis of 138 subjects from a phase II 24-week double-blind randomized clinical trial. In the 80 μ g abaloparatide subcutaneous self-injection group (which would prove to be the clinically approved dosage), the TBS measured 1.181 ± 0.078 versus 1.201 ± 0.068 in the 20 μ g teriparatide group (which is the approved dose); both these measurements are in the “degraded” group. In the 80 μ g abaloparatide subcutaneous self-injection group, TBS increased by 2.37% in the abaloparatide group ($n = 24$) versus 1.16% in the teriparatide group ($n = 31$) at 12 weeks and TBS increased by 5.23% in the abaloparatide group and by 3.27% in the teriparatide group at 24 weeks. Therefore, the effect of abaloparatide on TBS was greater than in the placebo group and the teriparatide group. An increase in TBS greater than the least significant change (LSC) was attained by 52.2% of subjects treated by abaloparatide versus 30.0% of the teriparatide group. The authors conclude that the increase in TBS values in the context of anabolic treatment is associated with a reduction in fracture risk, over and above what an increase in BMD would indicate (although this remains to be demonstrated), and the results help to differentiate abaloparatide from teriparatide in terms of potential effects on bone microarchitecture as determined by its surrogate measurement, TBS.¹⁴¹ The exact implications of these differences and their significance in the treatment of stress fractures and fracture healing, if any, remains to be determined.

The effects of denosumab on TBS,¹⁴² the effects of other antiresorptive agents,¹⁴³ and the effects of various disease states, such as Crohn’s disease¹⁴⁴ and end-stage renal disease in patients on hemodialysis,¹⁴⁵ primary aldosteronism,¹⁴⁶ ankylosing spondylitis,¹⁴⁷ and in so-called causes of secondary osteoporosis¹⁴⁸ have also been studied, but are beyond the scope of this text.

The normative database extends down to 20 years of age (and up to 80 years of age). However, only a few studies have looked at younger individuals^{149–151} and at young women with anorexia nervosa. In this later study, Donaldson and colleagues reported

on 57 adolescent girls age 11–18 years with anorexia nervosa recruited from an urban eating disorders clinic where they had undergone DXA examinations and peripheral QCT studies. The TBS of 6 (11%) of the participants were degraded and of 19 (33%) were partially degraded, according to adult normative values.¹⁵²

Heiniö et al. looked at the association between long-term exercise loading and TBS in different exercise loading groups. Eighty-eight Finnish female athletes competing at a national or international level and 19 habitually physically active non-athletes with a mean age of 24.3 years (range 17–40 years) who were all postpubertal and premenopausal were analyzed. The athletes represented seven different sports and five different loading regimens based on their sport-specific training history. Triple jumpers and high jumpers comprised the high-impact group, soccer and squash players made up an odd-impact loading group, power lifters made up the high-magnitude group, endurance runners a repetitive-impact group, and swimmers a repetitive nonimpact group. Several parameters of training and fitness status were employed, including maximal isometric force and dynamic performance of the lower extremities. Endurance runners’ mean TBS was about 6% lower than controls. Power lifters had about 3% higher TBS compared with the reference group. In the high-impact group, the correlation between maximal isometric leg press force, peak jumping force, and TBS was significantly positive. The authors “found that athletes experiencing a large number of monotonous impacts (repetitive, moderate impact loading represented by endurance runners) in their training and competition had significantly lower TBS compared with all other groups including the reference group, whereas the athletes experiencing extreme axial loading (high-magnitude loading represented by power lifters had somewhat higher crude TBS values compared with the reference group).” These TBS values were independent of lumbar spine BMD.¹⁵³

Thus, with increasing interest in the TBS of relatively young elite athletes, we undertook a study of our athlete population, most of whom had been referred for recurrent bone stress injuries, including traumatic fractures, stress fractures, stress reactions, and various degrees of delayed healing or nonunions. Prospectively, studying the athletes referred to us, we noticed some athletes with degraded or partially degraded TBS values. Therefore, we undertook a retrospective study of the TBS values in our athletic population in whom DXA studies had been performed. We evaluated 10 Major League Baseball (MLB) players, 5 minor league players, 7 NBA players, 2 collegiate basketball players, 5 active and 1 retired NFL players, and 4 amenorrheic intercollegiate female runners ($n = 34$). All TBS databases employed were gender and ethnic specific. One MLB player (Black) and one minor league player (White) had degraded TBS; one minor league player (White) had partially degraded TBS; two NBA players (Black) had degraded and one NBA player (White) had partially degraded TBS; one NFL player (Black) had partially degraded TBA ($n = 7$), indicating 20% of the population we deal with had an abnormally low TBS (Fig. 4.3).

All of the individuals had normal or “high” BMD based on their Z-scores, and therefore, BMD and TBS were discordant in these individuals.¹⁵⁴ Although the role that an abnormally low TBS plays in the pathogenesis of bone stress injuries or fracture

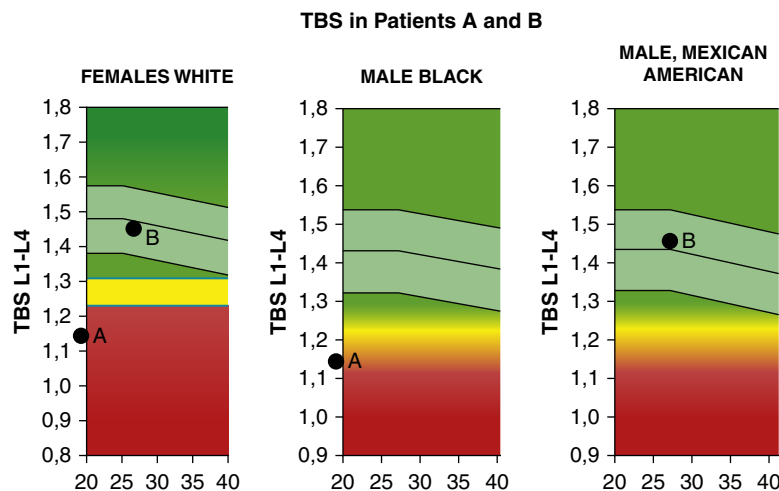


Fig. 4.3 Trabecular bone score (TBS) in 2 NBA centers: A = “degraded;” B = “normal;” 1st panel utilizing FDA-approved normal female Caucasian database; utilizing gender and ethnic specific-databases: 2nd panel utilizing male Black database; 3rd panel utilizing male Mexican American database; for gender- and ethnic-specific databases.

healing is not certain at this time, it probably indicates some underlying microstructural abnormality for which it is a surrogate, and therefore might be able to serve as a screening tool for athletes at greater risk of bone problems during their careers. What to do about this structural abnormality remains to be seen.

New approaches are constantly being developed for further evaluation of bone. A new software product, Texture Research Investigation Platform (TRIP) (Medimaps Group SA, Geneva, Switzerland), for the analysis of individual bone TBS is now available and will be utilized going forward. This new product will enable us to take the image from various modalities including x-ray, DXA, CT, pQCT, HRpQCT, and micro CT or MRI and analyze it for bone quality and microarchitecture. It displays images with several formats (e.g., Dicom, JPG, PNG, TIFF, BMP) and various sizes (personal communication). We hope that TRIP will be able to further help evaluation of the patient with a stress fracture because it can be localized to the bone in question and not applied to a bone that may not be involved in the local process as is lumbar spine TBS.

Osteoprobe

In 2006, Hansma and colleagues discussed the development of a prototype bone diagnostic instrument that took advantage of work being done on indentation to assess deteriorating biomaterials and was applied to alterations in bone microarchitecture.¹⁵⁵ Zysset subsequently reviewed the development of depth-sensing technologies allowing for better measurement of the tip displacement during the indentation process.¹⁵⁶

Osteoprobe is now a handheld medical device that creates an indentation in the anterior surface of the tibia to measure the material properties of the bone in vivo. The device uses reference point indentation (RPI) technology to perform precise micro-indentations in the bone and quantify a patient's bone score or bone material strength index (BMSI), a measure that may be related to bone quality.¹⁵⁷ In the last several years, significant literature has appeared utilizing Osteoprobe. Farr and colleagues from Mayo Clinic discussed their in vivo assessment of bone quality in postmenopausal women with type 2 diabetes. Because it now seems

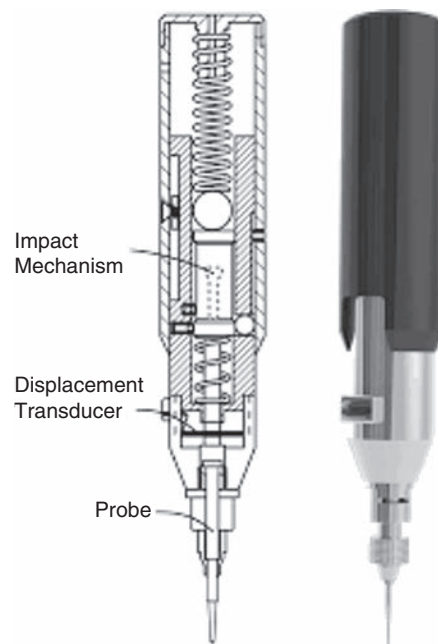


Fig. 4.4 Osteoprobe (Courtesy of Davis Brimer, ActiveLife Scientific, 2014).

that both BMD and FRAX* underestimate fracture risk in type 2 diabetes, other factors, such as bone quality, have been evaluated. Therefore, Farr et al. conducted a study of indentation in the nondominant anterior tibia site. Type 2 diabetes patients had significantly lower BMSI by 10.5% ($p < 0.001$), implying that fragility fractures in these patients may be due to reduced BMSI¹⁵⁸ (Fig. 4.4).

*FRAX (Fracture Risk Assessment Score), a tool to assess personal fracture risk, in use since 2008. FRAX uses demographic information (age, sex, weight, height, and ethnicity) and clinical risk factors (previous fracture after 50 years of age, parental hip fracture, current smoking, current glucocorticoid use ≥ 5 mg/d for >3 months, rheumatoid arthritis, secondary osteoporosis, three or more alcoholic drinks/day, femoral neck [FN], BMD, and, after 2015, TBS).

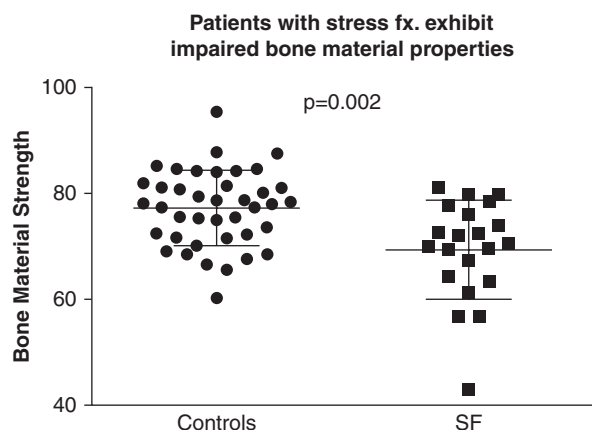


Fig. 4.5 Patients with stress fractures exhibit impaired bone material properties. fx, fractures; SF, stress fracture. (From Sosa and Eriksen, ASBMR, Abstract MO 0028, Sept, 2014. S350) Davsi Duarte Sosa and Erik Fink Eriksen, Patients with stress fractures exhibit impaired bone mineral properties by microindentation. *Journal of Bone and Mineral Research (JBMR)*, 2014, 29(S1):S359. Abs #MO0028.

Over the last several years, the utilization of micro-indentation has increased in an effort to further understand the role of BMSI in various settings.^{159–164} Sosa and Eriksen utilized the Osteoprobe device to study micro-indentation measurements in a retrospective case-control population of 30 pre- and postmenopausal women with a history of previous stress fracture. A control population of 30 subjects free of bone disease was also recruited. BMD was measured at the hip and lumbar spine, and body composition was also obtained. Bone turnover markers (BTOM), procollagen type 1 N-terminal propeptide (P1NP), a product of the osteoblast, and serum C-telopeptide (s-CTX), a product of osteoclast function, were also measured. Bone micro-indentation was performed by a described protocol where the average of eight measurements was taken and converted to BMSI. All micro-indentation measurements were made a median time of 4 (2–6) months after fracture (Fig. 4.5).

Compared with the controls, the stress fracture patients had significantly lower BMSI values—unadjusted (–6.7%; $p = 0.01$), age-adjusted (–5.2%; $p = 0.02$), and following adjustment (–6.3%; $p = 0.01$). The stress fracture patients had a mean BMSI value of 70.5 ± 8.7 versus control subjects' BMSI of 77.1 ± 7.2 . Mean BMSI was similar in the 6 postmenopausal women and in the 24 postmenopausal women with stress fractures (69.1 versus 71.8; $p = 0.9$). Femoral neck and lumbar spine BMD were both statistically lower in the stress fracture group than in the control group. Mean s-CTX levels were similar in the two groups; however, subjects with stress fractures had higher serum P1NP levels than controls. The authors concluded that “the lower values of BMSI in patients with stress fracture combined with a lower BMD may contribute to the increased fracture propensity in these patients.” They added that “the technique might possibly help identify subjects who are at increased risk of developing stress fractures in the future—after further validation in a prospective setting.”¹⁶⁵

Contraindications to this procedure might include a significant skin disorder, bruising, local edema or infection, history of lower extremity clotting disorders, undergoing treatment for a clotting disorder, or severe coagulation defects.¹⁵⁸ At the present time, the Osteoprobe has received approval for clinical use in Europe and is awaiting approval by the FDA in the US.^{159,166}

LABORATORY WORKUP AND BTOM IN STRESS FRACTURES

In the evaluation of patients with osteoporosis, the initial evaluation of the patient must include history and physical examination, and a workup for so-called causes of secondary osteoporosis¹⁶⁷ (Table 4.7, Box 4.3). The tabular list of diseases described may vary^{167–169} depending on the time when the list was originally compiled, as new diseases are constantly being added, e.g., aromatase inhibitor-induced bone loss, androgen deprivation therapy, and chronic use of proton pump inhibitors. In addition, the laboratory workup recommended varies in different references and guidelines.^{167,170} The workup may also change depending on whether the workup is undertaken by a primary care physician or by a specialty center looking for unusual occult causes of secondary osteoporosis.^{171,172} Most of the laboratory tests have been established as independent risk factors in older adults, and some of their value in the stress fracture population remains to be determined. Over the years, we have refined our laboratory workup to consist of the evaluation as shown in Box 4.4.

Bone metabolism consists of two processes: bone formation and bone resorption, which together result in bone modeling in children and adolescents and bone remodeling in adults after bone growth has stopped. There are two groups of bone turnover markers, assessed using blood or urine tests, that measure the function of the osteoclasts and osteoblasts. For bone formation, we utilize three blood tests to look for: bone-specific alkaline phosphatase (BSAP), osteocalcin (OC), and P1NP. For bone resorption, we utilize one blood test, the C-terminal cross-linking telopeptide of type I collagen (s-CTX), and one urine test, the N-terminal cross-linking telopeptide of type I collagen (u-NTx).^{173,174} Other markers of importance that we don't utilize are for formation, C-terminal propeptide of type I procollagen (P1CP) and for resorption, isoform 5b tartrate resistant acid phosphatase (TRACP5b). There is a great deal of individual and systematic variability in all these tests. They are affected by food intake, by menses, by seasonal variation, by exercise and physical activity, by various diseases, and by time of day. It has been shown that there is a strong circadian variation in the serum concentration of CTx. Qvist and colleagues studied 100 individuals with blood samples drawn every 3 hours over 27 hours and showed that the highest level of CTx occurred at 5:00 AM, with a low level at 2:00 PM with a variation of $\pm 40\%$ and was similar in premenopausal and postmenopausal women with normal and low bone mass; the circadian cycling was not affected by 5 days of bed rest.¹⁷⁵

Ben-Sasson et al. looked at whether sleep disturbances might increase the risk of stress fracture and showed that there was evidence of increased urinary calcium and hydroxyproline in the two groups that did not sleep horizontally (63 hours of sleeplessness [Group A] and vertical sleep in a seated position [Group B]); fasting urinary calcium increased by 170% and fasting urinary hydroxyproline (a marker of bone resorption) increased by about 100%. About 40% of individuals in Groups A and B were classified as “responders” and the remainder as “nonresponders.” Following this study, a mandatory sleep of 6 hours a day was instituted. “Preliminary results” from two cycles of basic training indicated a drop of more than 50% in the incidence of stress fractures among major combat units.¹⁷⁶

TABLE 4.7 Conditions, Diseases, and Medications That Cause or Contribute to Osteoporosis and Fractures

Lifestyle Factors		
Alcohol abuse	Excessive thinness	Excess vitamin A
Frequent falling	High salt intake	Immobilization
Inadequate physical activity	Low calcium intake	Smoking (active or passive)
Vitamin D insufficiency		
Genetic Diseases		
Cystic fibrosis	Ehlers-Danlos	Gaucher disease
Glycogen storage diseases	Hemochromatosis	Homocystinuria
Hypophosphatasia	Marfan syndrome	Menkes steely hair syndrome
Osteogenesis imperfecta	Parental history of hip fracture	Porphyria
Riley-Day syndrome		
Hypogonadal States		
Androgen insensitivity	Anorexia nervosa	Athletic amenorrhea
Hyperprolactinemia	Panhypopituitarism	Premature menopause (<40 years)
Turner and Klinefelter syndromes		
Endocrine Disorders		
Central obesity	Cushing syndrome	Diabetes mellitus (types 1 and 2)
Hyperparathyroidism	Thyrotoxicosis	
Gastrointestinal Disorders		
Celiac disease	Gastric bypass	Gastrointestinal surgery
Inflammatory bowel disease	Malabsorption	Pancreatic disease
Primary biliary cirrhosis		
Hematologic Disorders		
Hemophilia	Leukemia and lymphomas	Monoclonal gammopathies
Multiple myeloma	Sickle cell disease	Systemic mastocytosis
Thalassemia		
Rheumatologic and Autoimmune Diseases		
Ankylosing spondylitis	Other rheumatic and autoimmune diseases	
Rheumatoid arthritis	Systemic lupus	
Neurological and Autoimmune Diseases		
Epilepsy	Multiple sclerosis	Muscular dystrophy
Parkinson disease	Spinal cord injury	Stroke
Miscellaneous Conditions and Diseases		
AIDS/HIV	Amyloidosis	Chronic metabolic acidosis
Chronic obstructive lung disease	Congestive heart failure	Depression
End-stage renal disease	Hypercalciuria	Idiopathic scoliosis
Posttransplant bone disease	Sarcoidosis	Weight loss
Medications		
Aluminum (in antacids)	Anticoagulants (heparin)	Anticonvulsants
Aromatase inhibitors	Barbiturates	Cancer chemotherapeutic drugs
Depo-medroxyprogesterone (premenopausal contraception)	Glucocorticoids (≥ 5 mg/day prednisone or equivalent for ≥ 3 months)	GnRH (gonadotropin-releasing hormone) agonists
Lithium cyclosporine A and tacrolimus	Methotrexate	Parental nutrition
Proton pump inhibitors	Selective serotonin reuptake inhibitors	
Tamoxifen (premenopausal use)	Thiazolidinediones (such as Actos and Avandia)	Thyroid hormones (in excess)

From: The Surgeon General's Report [1], with modification

From Cosman F, de Beur SJ, LeBoff MS, Lewiecki EM, Tanner B, Randall S, et al. Clinician's guide to prevention and treatment of osteoporosis. *Osteoporos Int* [Internet]. 2014 Oct 15 [cited 2018 Jul 11];25(10):2359–81. Table 1 Available from: <http://www.ncbi.nlm.nih.gov/pubmed/25182228>

BOX 4.3 Exclusion of Secondary Causes of Osteoporosis

Consider the following diagnostic studies for secondary causes of osteoporosis

Blood or serum

- Complete blood count (CBC)
- Chemistry levels (calcium, renal function, phosphorus, and magnesium)
- Liver function tests
- Thyroid-stimulating hormone (TSH) +/- free T₄
- 25(OH)D
- Parathyroid hormone (PTH)
- Total testosterone and gonadotropin in younger men
- Bone turnover markers

Consider in selected patients

- Serum protein electrophoresis (SPEP), serum immunofixation, serum-free light chains
- Tissue transglutaminase antibodies (IgA and IgG)
- Iron and ferritin levels
- Homocysteine
- Prolactin
- Tryptase

Urine

- 24-h urinary calcium

Consider in selected patients

- Protein electrophoresis (UPEP)
- Urinary free cortisol level
- Urinary histamine

From Cosman F, de Beur SJ, LeBoff MS, Lewiecki EM, Tanner B, Randall S, et al. Clinician's guide to prevention and treatment of osteoporosis. *Osteoporos Int* [Internet]. 2014 Oct 15 [cited 2018 Jul 11];25(10):2359-81. Table 4 Available from: <http://www.ncbi.nlm.nih.gov/pubmed/25182228>

BOX 4.4 Biochemical Risk Factors for Fracture^a

- Homocysteine-Van Meurs, *N Eng J Med*, 2004;350:2033-41
 - Ferritin-Lipovetzki, *Immunol Res*, 2016;Aug 9 [Epub ahead of print]
 - Vitamin B 12-Clemens, *N Eng J Med*, 2014;371:963-4
 - Carotene-Accessed at <http://mayomedicallaboratories.com/test-catalog>
 - Hemoglobin A1c-Schwartz, AV, *Curr Osteoporos Rep*, 2007;5:105-11
 - Hs CRP-Eriksson, *J Bone Miner Res*, 2014;29:418-423
 - Magnesium-Orchard, *Am J Clin Nutr*, 2014;99:926-33
 - Phosphorus (Phosphate)-Wigner, *J Bone Miner Res*, 2010;25:724-33
 - Total and free testosterone-Eberling, *N Eng J Med*, 2008;358:1474-82
 - Serum and urine calcium-Bauer, *N Eng J Med*, 2013;369:1537-43
 - 25 vitamin D level-Miller, JR, *J Foot ankle Surg*, 2016;55:117-120
 - Estradiol-Reginster, *Calcif Tissue Int*, 2000;346:180-8
 - TSH, free T4-Bauer, *Ann Int Med*, 2001;134:561-8
 - SPEP, MGUS-Melton, *J Bone Miner Res*, 2004;19:25-30
 - Vitamin A-Feskanich, *JAMA*, 2002;287:47-54
 - Vitamin K-Pearson, *Nutr Clin Pract*, 2007;22:517-44
- Collagen Diagnostic Lab (Peter Byers, M.D., Univ of Washington) - When appropriate.

^aMostly in older men and postmenopausal women

There are an extensive number of studies that review changes in the bone turnover markers due to exercise, i.e., low-trauma and high-trauma fractures, and stress fractures. Research on bone turnover markers in the response to fractures has been going on

for almost 30 years. Oni and colleagues measured serum OC and total alkaline phosphatase levels as prognostic indicators in tibial shaft fractures. Serum was obtained from 50 adults with closed unilateral fractures of the tibia treated nonoperatively in a larger study of delayed union (100 consecutive patients) of these fractures. Total alkaline phosphatase levels were obtained immediately after fracture and every two weeks thereafter for 20 weeks, and OC levels were measured at 0, 4, 8, 12, and 16 weeks after fracture. The interesting findings were in the OC tests. OC was lower in high-trauma fractures as compared with low-trauma fractures throughout the study. The differences were statistically significant at 12 weeks. Additionally, the OC levels were higher from the fracture occurrence in the normal union group, compared with the delayed union group. The authors suggested that this “indicates that patients who develop delayed union may have a depressed osteoblastic function very early on...Thus, [OC] activity following a fracture may prove a useful indicator of the ability of a fracture to heal...”^{177,178}

Ivaska and coauthors updated the earlier information on bone turnover markers before and after fracture. Most of the previous studies took samples after the fractures had already occurred.¹⁷⁷ In order to avoid that problem, Ivaska's group utilized data from the Malmo Osteoporosis Prospective Risk Assessment (the Malmo OPRA) study, a population-based prospective, longitudinal study of 1044 women 75 years of age at inclusion selected at random from the city files of Malmo, Sweden. Specimens for blood and urine were obtained at entry and stored. Eventually, 113 women had low-trauma fractures and were available for the study after various exclusions. They had both serum and urine samples available at baseline, immediately after injury, at 4 months after injury and at about 12 months after injury. For bone resorption, they measured: s-TRACP5b, s-CTX, urinary total deoxypyridinoline (u-DPD); for bone formation, they measured serum total OC (s-totalOC), serum intact OC (s-OC), urinary osteocalcin (u-OC) and urinary midOC (u-midOC [referring to the middle of the OC molecule]), and s-P1NP. There were 30 hip fractures, 27 wrist fractures, 4 clinical symptomatic vertebral fractures, and 24 other fractures. Within 1 day after the occurrence of the fracture, there was no significant change in any of the markers. At 4 months after fracture (18/30 hip fracture patients and 22/27 wrist fracture patients sampled), the bone turnover markers were significantly elevated: 67% for s-OC, 55% for s-totalOC, 51% for s-P1NP, and 60% for u-midOC. The levels of all the bone turnover (except u-totalDPD) remained above prefracture level at 1 year. At the 4-month-after-fracture point, all bone formation and bone resorption markers were significantly elevated; formation more than resorption, especially after hip fracture. Analyzing the changes in the markers over time allowed researchers to monitor the different stages, processes, and patterns of osteoblast and osteoclast activity in the bone remodeling of fracture healing. There was an early increase in resorption markers and a latter increase in formation markers. Although all the markers were above baseline at the 1-year point, most of the increase occurred within 6 months of the fracture. Thus, although the fracture may be clinically healed or biomechanically healed, there is still some remodeling going on at the fracture site for at least 1 year. Because the bone turnover markers “sampled within a few hours after a

fracture are not significantly altered from preinjury levels, immediate postfracture sampling may...provide information on the baseline state of bone turnover of a fractured patient [and] this information will be a prerequisite if, in the future, we would by means of BTMs, be able to monitor the effects of pharmacological interventions promoting fracture healing.”¹⁷⁹

Coulibaly et al. studied the use of bone formation markers to monitor callus development and fracture healing. They reviewed a great deal of the literature (some of it cited already) and concluded “that PINP is the best candidate for use as a serological marker of bone healing.”¹⁸⁰

It has been thought that monitoring of bone metabolism by bone turnover markers in athletes would be instructive on the state of the responses and their bone health. Banfi et al. reviewed this topic in depth in 2010. There was tremendous variability in the results of individual studies depending on the gender of the athlete, the age of the athlete, the type and intensity of the exercise (high-impact versus low-impact), the markers studied (formation versus resorption), and whether or not the exercise was acute or chronic (training). The authors drew a number of conclusions: short exercise is insufficient for modifying serum concentrations of bone metabolism markers; variations in serum concentrations of bone metabolism markers are more evident during various hours or days after the exercise; bone metabolism markers show variable patterns depending on the type of exercise and the study design; acute changes may be due to changes in plasma volume and renal function; stimulation of osteoblast and osteoclast functions are exercise-dependent but immediate, and delayed effects need to be distinguished; and changes in osteocalcin may be partly due to changes in energy metabolism and increased osteoclast activity.¹⁸¹

For example, Eliakim and colleagues performed a study with two components: 1) a cross-sectional investigation of the correlation between fitness and bone turnover markers and 2) a prospective, controlled, endurance exercise training intervention. Forty-four high school males, 71% Asian, 20% Caucasian, and 9% Hispanic, 15–17 years old, at different Tanner stages, who were enrolled in a summer school program of two classes per day, were recruited and randomized to a control ($n = 22$) and training group ($n = 22$). All subjects took part in the 2-hour teaching program. The training group undertook an endurance-type training consisting of running, aerobic dance, competitive sports (e.g., basketball), and weightlifting. Bone formation was measured by osteocalcin, bone-specific alkaline phosphatase, and the C-terminal procollagen peptide (PICP), and bone resorption was measured by urinary free deoxypyridinoline cross-links (dPYR), urine C-Telopeptide (u-CTX), and urine N-terminal peptide (u-NTx). Blood and urine specimens were collected early in the morning during the week before and the week after the exercise program was concluded. All three bone formation markers showed a significant increase in the training group but not in the control group. In the training group, two of the three urinary bone resorption markers did not change but u-NTx decreased significantly. None of the urinary markers changed in the control group. The positive changes in bone formation were in the range of 15–30%. The effect of the

training intervention on bone resorption was less consistent than the effects on bone formation.¹⁸²

Banfi et al. concluded the following: bone formation markers change in sedentary subjects engaged in a physical activity program; professional athletes show changes in bone formation depending on program intensity whereas bone resorption appears to be stable; during prolonged training, the characteristics of exercise (e.g., weight-bearing, impact) are crucial; and different training baseline levels due to prestudy training history may partly explain different results between athletes and sedentary controls. The type of sport is more important than gender and whether the sports are high impact and weightbearing or nonweightbearing, e.g., male cyclists usually have lower BMD than male runners. They concluded that, given the lack of homogeneous behavior of bone markers in athletes, specific studies are needed that take into account the different effects a certain sport will have on bone metabolism.¹⁷⁷

Other studies have also looked at the response of bone turnover markers (and cytokines) to a single episode of high-intensity exercise in order to define the earliest changes due to exercise. Earlier studies have shown that single episodes of 60-minute strenuous exercises such as treadmill running and cycling stimulate a bone turnover response, with increased bone resorption over bone formation. Mezil et al. performed research on short bouts of high-intensity interval exercise (HIE) (or training–HIIT), which has become popular to produce results similar to more conventional long-term exercise. They designed a trial of 23 male university students who undertook a course of exercise that involved 12 minutes of intermittent cycling of six 1-minute high-intensity cycling intervals at 90% of maximum workload separated by six 1-minute active rest periods. Markers studied included bone-specific alkaline phosphatase (BSAP), the receptor activator of NF- κ B (RANK), the ligand of RANK (RANKL), osteoprotegerin (OPG), which acts as a decoy receptor for RANKL, and NTx. Blood was drawn at baseline, at 5 minutes after exercise, 1 hour after exercise, and 24 hours after exercise. Also, selected cytokines were studied. There was a significant time effect for all markers: at 5 minutes after exercise, BSAP, OPG, and RANKL increased from baseline by 10.9%, 13.5%, and 34.2%, respectively; at 1 hour after exercise, only BSAP was significantly higher than baseline by 9.5%; at 24 hours after baseline BSAP was still 10.9% higher than baseline, whereas NTx was 14.6% lower than baseline. There were significant correlations between the percent exercise-induced changes in bone turnover markers and cytokines. Since this study had a high level of exercise, as opposed to lower levels of exercise in some other studies, it may be more like the level of exercise in elite athletes, and if there is a decrease over time in bone resorption, then, maybe after HIIT, bone resorption cannot keep up with the degree of microdamage developed by the exercise program and eventually, failure of bone occurs.¹⁸³

Starting in 1988, the question of whether bone turnover markers could be used to select a population of at-risk individuals in either the military or athlete populations has been actively researched. Murguia and coauthors measured basal plasma hydroxyproline, a product of collagen degradation in 104 male Navy Seal candidates 1 week into their basic underwater demolition (BUD/S) training program to see if it correlated to the incidence of connective tissue injuries, that occurred later in the training program. Eleven

subjects (10.6%) were diagnosed as having connective tissue injuries, and they had a significantly higher mean hydroxyproline level (4.02 $\mu\text{g/mL}$) than trainees without injury (3.10 mcg/mL). They concluded that there was a relationship between baseline elevated hydroxyproline levels and subsequent connective tissue injuries, which may indicate an increased risk of connective tissue injuries including stress fractures in individuals with the highest level of hydroxyproline at baseline.¹⁸⁴

Bennell and colleagues, in a three-part series of studies on track and field athletes, looked at the relationship between stress fractures and bone turnover in these individuals. Eventually, after exclusions, 95 athletes (46 women, 49 men) were available for analysis. Of these athletes, 5 women and 11 men competed in sprints, 17 women and 18 men in middle-distance running, 9 women and 10 men in distance running, 6 women and 5 men in hurdles, 5 women and 5 men in jumps, and 4 women and 0 men in multi-events. During the 12-month study period, 10 women (21.7%) and 10 men (20.4%) had at least one stress fracture. One man and one woman had multiple stress fractures. Of the 26 fractures in the 20 athletes, 45% occurred in the tibia, 15% in the navicular, 12% in the fibula, and 8% in the metatarsal bones. OC was measured as a marker of bone formation, and urinary pyridinium cross-links (u-Pyr) and deoxypyridinoline (D-Pyr) and u-NTx were collected appropriately. Samples were collected at baseline and monthly over the course of the study. There was no difference between stress fracture and nonstress fracture groups. After adjusting for age and sex, integrated monthly values of u-Pyr, D-Pyr, and OC were 5%, 4%, and 35% higher, respectively, in those athletes who developed a stress fracture, although these were not statistically significant. In the hydroxyproline study, where there was elevation of that value in the sailors with connective tissue injuries, these authors point out the differences in the markers, including the main fact that hydroxyproline is not specific to bone, so some of the elevation may have been from nonskeletal sources, whereas the markers in this study were more specific for osteoblast function and collagen breakdown. Several issues, including the high variability of their markers and the size of the study group, may have contributed to these differences between the Bennell and Murguia studies. They concluded that single and serial measurements of bone turnover are not useful in predicting the tendency to stress fractures in individual athletes.¹⁸⁵

Etherington et al. studied the effects of 10 weeks of military training on heel ultrasound and bone turnover in 40 male military recruits entering their basic training in the United Kingdom; 10 (25%) of the recruits sustained an injury. Subjects were assessed at the start and end of training. They measured bone formation via OC and BSAP, and bone resorption by TRAP. There were two clinically diagnosed stress fractures: one in the tibia and one in a metatarsal. OC declined 11.6% significantly, and there was a nonsignificant decrease in BSAP 13.3% and TRAP of 9.5%. Baseline concentrations of all bone markers were not significantly higher in those recruits subsequently injured compared with those not injured. These changes suggest there is a decrease in bone turnover in response to the level of strenuous exercise to which these recruits were subjected.¹⁸⁶ Evans and colleagues looked at the effects of a 4-month recruit training program on markers of bone metabolism. In this study, 257 healthy men ($n = 58$) and women (n

$= 199$) entered a gender-integrated basic recruit training program in the IDF and blood for bone turnover markers was collected before training (baseline), at the midpoint of training (about 2 months), and the day before graduation from training (about 4 months). Bone formation was measured by BSAP and PINP, and bone resorption was measured by TRAP5b and s-CTX. All markers of bone turnover were significantly higher in men than women at baseline and remained higher at all three time points. The bone formation markers increased significantly over time ($p < 0.001$) in both genders. For BSAP, the increase was evident from baseline to 2 months ($p < 0.001$) with no change from 2 to 4 months. Women had a nonsignificant 20.6% increase in PINP, while men had an increase of 5.2% from baseline to 2 months, which declined significantly from 2 months to 4 months ($p < 0.01$). Bone resorption markers changed with the same trend in both genders: CTx increased in both men and women between 0 and 2 months ($p < 0.001$) and declined to baseline at 4 months ($p = 0.003$), and TRAP5b increased from 0 to 2 months only ($p < 0.001$). Thus, a strenuous 4-month period of military recruit training resulted in similar increases in serum markers of bone formation and resorption in both men and women primarily during the first 2 months of training, implying that the initial response to exercise is an acceleration of bone turnover that does not differ between men and women.¹⁸⁷

Hetland et al. studied bone turnover in 120 male medium- and long-distance runners selected randomly from a population of 1520 men who were respondents to a questionnaire sent to 2467 participants in various running events in Denmark. They measured bone density by DXA of the lumbar spine and hips and SPA of the forearm. u-Pyr and u-Dpd, serum total alkaline phosphatase, and osteocalcin were also studied along with testosterone, progesterone, estradiol, leuteinizing hormone, follicle-stimulating hormone, and sex-hormone binding globulin. All the bone turnover markers were significantly and positively correlated to the weekly distance run. The bone turnover markers were within the normal laboratory range in all of the elite runners, but the markers of the elite runners were 20–30% higher than the controls. Of interest, their “principal finding” was a significant negative correlation between running distance and BMD. The male long-distance runners had a significantly lower bone mineral content (BMC) at the lumbar spine, proximal femur, distal forearm, and total body than the controls. The difference between runners and nonrunners increased as the weekly distance run increased. For those who ran more than 100 km a week, the lumbar spine BMC was, on the average, 19% lower than controls. Thus, the long-distance runners had lower BMC and higher bone turnover. There were no significant changes in the hormonal studies. Thus, the BMC and bone turnover studies suggest long-distance running causes bone loss.¹⁸⁸

Ruohola and colleagues studied randomly selected healthy individuals (796 men and 24 women) with a mean age of 19.8 (range 18–28) years entering military service. Baseline blood samples were obtained for TRACP5b, a marker of bone resorption. Subsequent TRACP5b samples were drawn on the day the stress fracture was diagnosed or suspected (Sample I), and then drawn every 3 to 4 days for three more samples (Samples II, III, IV). Previous studies had shown 85% of stress fractures occurred

in the first 8 weeks of basic military training for all conscripts in Finland. Twenty of the 820 (2.4%) individuals developed stress fractures; 6 were lost to follow-up. Therefore, 21 stress fractures were diagnosed in 14 individuals; 3 conscripts had bilateral stress fractures; 1 had two unilateral tibial fractures; 1 had fractures in the first and second metatarsals; 12 (57%) were in the tibia, 6 (29%) were in the metatarsals, and 3 (14%) were in the calcaneus. A comparison of the ratio of Sample IV results and baseline values between the fracture and control groups indicated that the odds of a stress fracture were eight-fold greater in the soldiers with greater serum TRACP5b results, but this value was not significant ($p = 0.17$). The difference in TRACP5b activity levels between baseline and Sample III was statistically significant ($p = 0.039$), but although the TRACP5b was increased and increased over time from the diagnosis of a stress fracture, the overall increase remained statistically nonsignificant when the point of significance was set at $p < 0.05$. Therefore, “the usefulness of serum TRACP5b measurement for early diagnosis of bone stress injuries could not be confirmed,” although the elevated level probably indicates a high bone turnover rate in these individuals.¹⁸⁹

Yanovich and coauthors recruited 85 male members of a combat unit of the IDF; 69 of them completed an 18-week training course, 22 of whom developed a stress fracture. They measured BSAP, PINP, TRAP5b, and s-CTX. At baseline, none of the bone turnover markers differed between soldiers who did and did not have stress fractures. During basic training, starting at baseline, none of the bone turnover markers tested differently between subjects who subsequently did and did not have stress fractures, although all marker levels decreased by week 18: in the stress fracture group, BSAP decreased by 13.3%, PINP decreased by 22.3%, TRAP5b decreased by 2.6%, and CTx decreased by 19.4%; in the nonstress fracture group, BSAP decreased by 19.7%, PINP by 40.5%, TRAP did not change, and s-CTX decreased by 21.5%. There were multiple limitations to this study, most importantly the small sample size. So, neither the bone formation markers or the bone resorption markers could identify soldiers who had stress fractures and those who did not, so they could not be used as diagnostic or predictive tools for stress fracture evaluation in soldiers during basic training.¹⁹⁰

A series of recent articles from Japanese sports medicine programs looked at bone turnover markers, various athletic programs, and stress fractures in athletes as opposed to military recruits. Wakamatsu et al. studied 84 elite university lacrosse players (male = 35 with control group = 30; female = 49 with control group = 42). There were 5 male athletes and 7 female athletes with stress fractures. They measured blood BSAP, NTx, and TRAP5b as well as homocysteine and pentosidine as bone quality markers. There were no significant differences in the levels of BSAP, NTx, and TRAP5b as observed between the stress fracture and control groups for all subjects. In male players, no significant differences in BSAP, NTx, and TRAP5b were observed between the stress fracture and control groups. Similarly, in female players, no significant differences were seen in the levels of BSAP or NTx; however,

TRAP5b levels were 409 ± 209.3 and 318.6 ± 81.6 mU/dL ($p < 0.05$) in the stress fracture and control groups, respectively. They concluded “that TRAP5b may be a useful bone metabolism marker for monitoring bone status in female lacrosse players.”¹⁹¹ Harada and colleagues looked at bone metabolism markers in 21 male college artistic gymnasts who were at an Olympic level of competition. They measured BSAP, PINP, and PICP (cleaved C-terminal propeptide), s-NTx, u-NTx, and TRACP5b. The study was conducted to clarify bone turnover in periods of different training intensity based on the annual schedule. Measurements were performed in three periods: the preseason, the competition period, and the training period. BSAP was significantly higher during the competition period than during the preseason or training periods; no significant difference was found between the preseason and competition periods. PINP was significantly higher during the competition period than during the training period; no significant difference was found between the preseason and the competition periods or between the preseason period and the training period. s-NTx was significantly higher during the preseason period and the training period than the competition period. No significant difference was found between any period for u-NTx. TRACP5b was significantly higher during the preseason and training period than the competition period and significantly higher during the training period than the preseason period. They decided that PINP and TRACP5b sensitively reflect bone turnover. Bone formation–dominant bone turnover occurred when training intensity was high and significant mechanical stress was applied to the gymnasts’ bones, and training intensity bone resorption–dominant bone turnover occurred when the training intensity was lower and less mechanical stress was applied to the bones.¹⁹²

Beyond the effect of exercise on bone turnover markers (and cytokines), there has been an interest in the effect of stress fractures on these tests. Fujita and colleagues looked at the evolution of urine NTx bone resorption marker prospectively over a 3-year time interval to attempt to capture any changes in bone turnover markers if a stress fracture developed. To undertake this study, they recruited 25 female long-distance runners, ages 19–34 years (avg 23.99 ± 4.11). In order to have baseline measurements, they took u-NTx 11 times during a period from 2011 to 2014. Six participants ended up with less than three specimens and were excluded, so the study ended up with 19 participants. The onset date was when the participants experienced pain at the injury site. The mean u-NTx before a stress fracture in the whole group was 40.16 ± 9.10 nmol BCE/mmol creatinine; the mean u-NTx after a stress fracture was 64.08 ± 16.07 nmol BCE/mmol creatinine ($p < 0.01$). This indicated increased bone resorption, which they ascribed to the accumulation of excessive microdamage.¹⁹³

In order to more precisely define the role of bone turnover markers in the prediction of stress fracture risk, the pathophysiology of stress fractures, or the healing of stress fractures, additional larger series that are adequately powered to present the results in statistically significant numbers need to be conducted.

THERAPY-CALCIUM, VITAMIN D, ANABOLICS, BISPHOSPHONATES, AND BONE GROWTH STIMULATORS

Calcium

It goes without saying that calcium is important for bone stability and building. We have criteria from the Institute of Medicine (IoM) in 2011 to set guidelines for calcium intake. The Committee concluded that available scientific evidence supports a key role of calcium and vitamin D in skeletal health^{194,195} in community-living, ambulatory, noninstitutionalized individuals. The question is whether elite athletes have different requirements (Table 4.8).

Increased activity in and of itself does not increase the need for calcium.¹⁹⁶ There may be some excess calcium loss in the sweat, but this has not been shown to be irreplaceable with a normal to high-calcium diet,¹⁹⁷ but this is not true for all studies.¹⁹⁸ So if an athlete is in hot, humid climate or profusely sweating, a diet adequate in calcium should be consumed or some additional calcium intake and/or supplementation may be necessary. Certainly, athletes restricting calories to reduce or control weight may be reducing their intake of many minerals and may require supplementation.¹⁹⁶

TABLE 4.8 New Recommended Daily Amounts of Calcium and Vitamin D

Life Stage Group	Calcium Recommended Dietary Allowance (mg/day)	Vitamin D Recommended Dietary Allowance (IU/day)
Infants 0 to 6 months	*	**
Infants 6 to 12 months	*	**
1–3 years old	700	**
4–8 years old	1000	600
9–13 years old	1300	600
14–18 years old	1300	600
19–30 years old	1000	600
31–50 years old	1000	600
51–70 years old	1000	600
51–70 years old females	1200	600
71+ years old	1200	600
14–18 years old, pregnant/lactating	1300	600
19–50 years old, pregnant/lactating	1000	600

*For infants, adequate intake is 200 mg/day for 0 to 6 months of age and 260 mg/day for 6 to 12 months of age.

**For infants, adequate intake is 400 IU/day for 0 to 6 months of age and 400 IU/day for 6 to 12 months of age.

New recommended Daily Amounts of Calcium and Vitamin D NIH MedlinePlus, 2018

From *MedlinePlus The Magazine*, Winter 2011, Page 12

https://magazine.medlineplus.gov/pdf/MLP_Winter_2011.pdf

How We Use Calcium

Most of our patients have 24-hour urine collections for creatinine clearance and total urinary calcium. If the calcium is low, we give calcium by adding dairy sources to the diet (each dairy source is about 300 mg). If they are not able to take dairy or additional dairy, we add CitraCal with vitamin D, one to two tablets per day in divided doses.

Vitamin D

A complete review of vitamin D synthesis, action, and metabolism is beyond the scope of this chapter. However, vitamin D deficiency or insufficiency can cause or exacerbate low bone mass (osteopenia) or osteoporosis, can cause osteomalacia and muscle weakness, and can increase the risk of fracture.¹⁹⁹ Although there is no consensus on optimal levels of 25-hydroxyvitamin D (25 [OH]D) as measured in the serum, and there are methodological differences and lack of standardization in the measurement of 25 hydroxyvitamin D levels that result in significant interlaboratory variability,²⁰⁰ the 25 hydroxyvitamin D deficiency level, as defined by most experts, is below 20 ng/mL (50 nmol/L).¹⁹⁹ Data exist that suggest that a 25 hydroxyvitamin D level of 21–29 ng/mL (50–80 nmol/L) indicates an insufficiency state of 25 hydroxyvitamin D, and a level of 25 hydroxyvitamin D equal to or above 30 ng/mL indicates a sufficient level of 25 hydroxyvitamin D,¹⁹⁹ but the IoM report indicated that a level of 20 ng/mL (50 nmol/L) meets the vitamin D needs of 97.5% of the population.¹⁹⁴ The IoM report also set guidelines for vitamin D intake, but did state that individuals with bone disease might need more intake or higher levels.

Without vitamin D, only 10%–15% of dietary calcium and 60% of phosphorus is absorbed. When 1, 25-dihydroxyvitamin D interacts with the vitamin D receptor (VDR) at the intestinal lumen, it increases the efficiency of intestinal calcium absorption to 30%–40% and phosphorus absorption to approximately 80%. Bischoff-Ferrari et al. reviewed numerous studies in an attempt to estimate optimal levels of 25 hydroxyvitamin D for multiple different health outcomes.²⁰¹ One cited study showed that maximal bone density was obtained in both sexes and multiple ethnicities when a 25 hydroxyvitamin D level of 40 ng/mL was reached.²⁰² In addition, it was shown that optimal fracture prevention occurred at a 25 hydroxyvitamin D concentration of 40 ng/mL (100 nmol/L).²⁰³

There is an extensive body of literature that documents an apparent worldwide deficiency or insufficiency in vitamin D; by some estimates, this number may be as high as 1 billion people.¹⁹⁹ The athletic population is not immune to this phenomenon. Numerous studies have looked at convenience samples of athletes. Villacis et al. evaluated 223 (40%) of 559 NCAA Division I athletes at the University of Southern California in 2012. One resting venous blood sample was taken from each study participant between June and August 2012. Measurement of serum 25(OH)D was done using liquid chromatography tandem mass spectrometry (LC-MS/MS) that had an analytical sensitivity of 4 ng/mL for 25(OH)D2 and 25(OH)D3. Vitamin D “sufficiency” was defined as a serum 25(OH)D level greater than 32 ng/mL; a level between 20 and 32 ng/mL was defined as “insufficient,” and a level less than 20 ng/mL was defined as “deficient.” Results showed

148 (66.4%) of the athletes had sufficient levels and 75 (33.6%) had abnormal levels, of which 68 (30.5%) were insufficient and 7 (3.1%) were deficient. Male athletes had a 2.8-fold higher chance of having an abnormal vitamin D level than female athletes did. Dark-skinned athletes had a 15.2-fold chance of having an abnormal vitamin D level than did light-skinned athletes. Winter sports athletes had a greater chance of having a low vitamin D level than did spring sports athletes. Indoor sports athletes had lower levels of vitamin D than did outdoor sports levels. Athletes with muscle injuries have significantly lower vitamin D levels compared with uninjured patients. Those with higher 25(OH)D levels have statistically significant higher jump velocities, jump heights, and fitness indices. The authors concluded that vitamin D insufficiency was common in elite athletes, and dark skin tone was the only statistically significant risk factor.²⁰⁴ A recent study examined the vitamin D levels of 89 players on a single NFL team and found that 30% of the players were deficient while 51% had insufficient levels.²⁰⁵ Vitamin D levels of an NBA team, performed at pre-season physicals in 2007, 2008, and 2009, showed that between 9 and 12 out of 16 individuals per year were deficient or insufficient, and of an MLB team in 2011 found 90% of players were low, defined as less than 30 ng/mL. In these two studies, 25(OH)D levels were measured by the LC-MS/MS methodology.²⁰⁶

Ruohola and colleagues looked at the association between serum 25(OH)D levels and stress fractures in young Finnish men. In this prospective study, 800 young Finnish men undergoing military training were randomly selected. Blood samples for serum 25(OH)D levels were drawn from these 800 recruits; because of failed samples, incomplete follow-up data, and other issues, the total final study population consisted of 756 subjects. During the 3-month follow-up of the 756 recruits, 30 stress fractures were identified in 22 recruits. The median 25(OH)D level was lower in the stress fractured group (25.7 ng/mL) than the nonfractured group (30.5 ng/mL), and 81.8% of the fracture patients were below the median. Thus, this study showed that a lower level of 25(OH)D was a risk factor for developing stress fractures.²⁰⁷ In a prospective double-blind, placebo-controlled, randomized clinical trial of female US Navy recruits during the 8-week basic training program, Lappe et al. treated 5201 volunteers. The study groups were given treatment of 2000 mg calcium and 800 IU of vitamin D or identical control placebo. Three hundred nine recruits were diagnosed with 496 stress fractures in the tibia, fibula, femur, or pelvis. There was a 21% lower incidence of stress fractures in the supplemented group than in the control group.²⁰⁸

There is a large body of literature on the topic of vitamin D's effect on athletic performance, but that is beyond the scope of this chapter.^{209–211} Magnesium, phosphorus, and other nutrients play a role in athletic performance but do not have a known role in the prevention or causation of stress fractures or in fracture healing.^{212–215}

One issue with regard to vitamin D needs to be touched upon: it has been known for many years that community-dwelling Black Americans have lower levels of total 25(OH)D than whites, and thus they are frequently given a diagnosis of vitamin D deficiency. Because of this, Powe and her colleagues considered whether vitamin D-binding protein (VDP) might play a role in this phenomenon. VDP is the primary vitamin D carrier protein, binding 85%–90% of total circulating 25(OH)D. The

non-VDP fraction (bioavailable 25(OH)D) consists of albumin-bound 25(OH)D (10%–15% of total 25(OH)D), with less than 1% of total 25(OH)D in the free form. Common genetic polymorphisms in VDP gene produce variant VDP that differ in their affinity for vitamin D. The prevalence of these polymorphisms differs between racial groups.

Clinical assays measure the level of 25(OH)D without measuring fractions bound to carrier proteins. Powe et al. studied 3720 participants from the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) and measured levels of total 25(OH)D2 and 25(OH)D3 via LC-MS/MS, levels of VDP using a commercial enzyme-linked immunosorbent assay that uses two monoclonal antibodies in a sandwich format, levels of parathyroid hormone (PTH), and calcium levels corrected for albumin. BMD was also measured. DNA samples of the subjects were genotyped for two common SNPs at the coding region of the VDP gene (rs4588 and rs7041). Bioavailable 25(OH)D was defined as circulating 25(OH)D not bound to VDP. Subjects were divided into quintiles to examine relationships between 25(OH)D and markers of vitamin D status (PTH level, calcium level, and BMD). Racial differences in total 25(OH)D levels persisted after multivariable adjustment (17.3 ± 0.3 ng/mL in Blacks versus 25.5 ± 0.4 ng/mL in Whites ($p < 0.001$)). Race explained 22.7% of the variation in total 25(OH)D. Adjusted mean BMD at the femoral neck, the calcium levels, and the PTH levels were higher in Blacks than Whites. Blacks were more likely than Whites to have the T allele at rs7041, whereas Whites were more likely to have the G allele, and Blacks were less likely to have the A allele. These genetic differences explained 79.4% of the variation in VDP levels and 9.9% of the variation in total 25(OH)D levels. Thus, the concentration of VDP and the genotype of VDP appeared to explain about 31.2% of the variation. Researchers concluded that low vitamin D levels in Blacks did not necessarily indicate vitamin D deficiency; that bioavailable 25(OH)D may be a more appropriate cross-racial marker of vitamin D sufficiency; that levels of total 25(OH)D are, in part, genetically determined; and that to improve the determination of vitamin D status in different populations, the measurement and level of VDP needs to be incorporated into the assessment.²¹⁶ In an accompanying editorial, Holick noted that “vitamin D deficiency may need to be redefined to consider not only total but also bioavailable 25-hydroxyvitamin D levels.”²¹⁷

This potentially exciting new insight into vitamin D levels and metabolism drew a great deal of interest. In a series of letters to the editor of the *New England Journal of Medicine*, numerous issues were raised, including whether or not the vitamin D levels were correctly measured, whether or not the two monoclonal antibodies in the VDP assay had equal affinity for all genotypes, and a variety of other technical issues.²¹⁸ In a subsequent letter to the editor, Nielson et al. stated that it was unclear whether circulating free or bioavailable 25(OH)D is a better test of vitamin D status than is total 25(OH)D, especially in racially diverse populations. They “presented evidence that the use of a monoclonal ELISA to measure vitamin D-binding protein in persons of African ancestry introduces a critical flaw into the calculation of free or bioavailable 25-hydroxyvitamin D, a limitation that influenced the conclusions of Powe et al. (see earlier section) and other investigators.” And “in conclusion,

median levels of free 25(OH)D were significantly lower in black men than in white men in the United States when the levels were measured directly or calculated on the basis of polyclonal antibodies against vitamin D-binding protein.” “These results contradict previous reports of similar levels of free or bioavailable 25-hydroxyvitamin D between races.” Their results emphasize “the importance of the choice of assay for vitamin D-binding protein in the calculation of free 25-hydroxyvitamin D in diverse populations and support the measurement of total 25-hydroxyvitamin D in the general population as a marker of vitamin D status, regardless of race or GC genotype.”²¹⁹ Denberg et al. reported the first study that compared the measurement of VDP by three assays: two widely used commercially available immunoassays, a monoclonal and polyclonal ELISA; and an LC-MS/MS and estimates of free and bioavailable 25(OH)D concentrations. Combinations of two SNPs produce three major VDP isoforms (Gc1f, Gcs, and Gc2). The authors felt their results confirmed the differential and biased performance of the monoclonal ELISA according to genotype and, therefore, race. “Inferences from studies that have used this assay should be made cautiously, especially when interpreting reported race differences in vitamin D status. Future studies of DBP and free or bioavailable vitamin D metabolites should employ DBP [VDP] assays that are not biased by DBP [VDP] genotype.”²²⁰ Bouillon, in a commentary on laboratory implications in the *Journal of Bone and Mineral Research*, stated that “monoclonal R&D [one of the manufacturers of one of the assays] DBP assay displays a bias in the measurement of DBP/Gc1f and cannot be considered a reliable method for genetically mixed populations. Polyclonal antibodies do not display this bias, but the absolute concentration of DBP differs very widely according to the assay technique. Therefore, *assay standardization is needed*....” In a section on clinical implications, he wrote that in the conclusions of the Powe study (see earlier section) there are racial and genetic differences in free 25(OH)D that cannot be revealed by total 25(OH)D, and suggested that there is a need for a new evaluation of measured vitamin D status and health outcomes. This conclusion “was based on a wrong DBP assay resulting in marked overestimation of free 25(OH)D in African Americans.” Thus, the studies of directly measured free 25(OH)D studies are contradictory at this point in time.²²¹ In addition, experience with direct measurements of free levels is limited at the present time, but it is likely that free vitamin D levels will play an important role in the assessment of vitamin D status in the future.²²² However, “the final answer about racial or genetic differences in measured free 25(OH)D is, therefore, still unsettled.”²²¹

Clinical Application

How we use vitamin D. Presuming there is no previous or recent vitamin D level, when we see a light-skinned athlete, we obtain a 25 vitamin D level by LC-MS/MS technique and wait for the results before deciding on the vitamin D prescription that may consist of vitamin D3 at 1000 IU, 2000 IU, or 5000 IU per day or 50,000 IU per week. Depending on the initial value and the prescription, we repeat the 25 vitamin D level test in 1- to 3-month intervals. When we see a dark-skinned athlete, where the likelihood of a low vitamin D level is high, presuming there is no previous or recent vitamin D level, we will determine a 25 vitamin

D level by the LC-MS/MS technique at baseline and then give the athlete five sample tablets of a vitamin D3 50,000 IU preparation to take daily. Then we usually will have the vitamin D level result from the laboratory, and follow up as before, frequently continuing vitamin D3 50,000 IU weekly in this population. There are two phases of vitamin D treatment: restoration and maintenance. Each of these has its own issues, techniques, and tricks for correction and preservation. It is important to become familiar with these steps in practice.

Anabolic Agents for Bone

In early 2002, the clinical use of teriparatide (PTH 1-34) began the era of anabolic drugs for the treatment of osteoporosis. This required that attention be paid to further defining the nature and role of medications that are anabolic for bone. All previously available medications (etidronate, calcitonin, estrogen, alendronate, risedronate, and subsequently ibandronate) had antiresorptive mechanisms of action. Anabolic drugs can increase bone mass and reduce fracture risk.²²³ These medications act by increasing bone remodeling and formation more than resorption, whereas the other type of bone-active medications, classified as antiresorptive or anticatabolic, act by reducing bone remodeling. In the adult skeleton, virtually all the bone-forming units, called basic multicellular units (BMUs), participate in bone remodeling. During the growth phase of the skeleton, many BMUs are committed to a different process called bone modeling, in which bone formation occurs without a preceding resorption phase and can produce large increases in bone mass (growth from fetus to adolescence). Teriparatide affects both remodeling and modeling, and newer anabolic medications also stimulate both bone remodeling and modeling; however, the effectiveness of the anabolic agent, especially with regard to the off-label use of fracture healing, may well depend on the proportions of remodeling or modeling that a specific drug produces. Intermittent PTH does this by increasing the number of osteoblasts and their function and life expectancy.^{224,225}

Experimental studies looking at the effects of various PTH extracts on the bones of growing rats and the histological changes that occurred from those extracts date back to the 1930s.^{226,227} By the 1990s, it was clear that the intermittent administration of PTH had a greater anabolic effect on bone than the continuous infusion of PTH. In these early studies, intermittent PTH increased bone mass by approximately 30%, whereas continuous PTH had a smaller and less consistent effect on bone mass. In a prelude to a potential future use, researchers found that both continuous and intermittent PTH reversed the decrease in bone formation by diphosphonates (now bisphosphonates).²²⁸

Teriparatide (Forteo) rh PTH (1-34)

In 2001, Neer and colleagues reported on the effect of parathyroid hormone (1-34) on fractures and BMD in postmenopausal women with osteoporosis. Native PTH is a 1-84 amino-acid peptide. Its biologic effect is concentrated in the first 1-34 amino acids. They screened almost 10,000 women more than 5 years postmenopause and selected 1637 who were randomized to placebo (544 women) or to parathyroid hormone (1-34) at a dose of 20 µg per day (541) or 40 µg per day (552). PTH (1-34) at the 20 µg or 40 µg per day dose reduced the risk of one or more new

vertebral fractures by 65% or 69%, respectively; the risk of two or more fractures was reduced by 77% and 86%, respectively; and the risk of at least one moderate or severe vertebral fracture was reduced by 90% and 78%, respectively. Nonvertebral fractures were reduced by 35% and 40%, respectively. BMD of the spine increased 9.7% and 13.7%, respectively; BMD of the femoral neck increased 2.8% and 5.1%, respectively; BMD of the total hip increased 2.6% and 3.6%, respectively; shaft of the radius (a cortical bone site) declined 2.1% and 3.2%, respectively. These BMD changes may have relevance to the issue of fracture healing (see below). Side effects included nausea, headache, dizziness, and leg cramps, with less severity in the 20 μ g group. Injections of PTH (1-34) raised the serum calcium 4–6 hours postinjection, and levels came down to normal at 16–24 hours after the injection. The urinary calcium increased by about 30 mg per day but the incidence of hypercalciuria did not. Serum 25-hydroxyvitamin D did not change although 1,25 di-hydroxyvitamin D declined slightly. Serum magnesium also declined slightly. Serum uric acid rose 13%–20% in the 20 μ g group and 20%–25% in the 40 μ g dose group. Within an average of 5 weeks after cessation of treatment, serum calcium, magnesium, and uric acid concentrations returned to or approached pretreatment levels. Serum creatinine and creatinine clearance were unaffected by PTH (1-34) treatment. Circulating antibodies were rare and did not have any discernible adverse effects.²²⁹

As that clinical trial was being conducted, a 2-year study in rats was simultaneously conducted to assess the near-lifetime exposure to PTH (1-34). The high dose used in this study (75 μ g/kg) was over 200 times greater than the therapeutic human dose. The Fischer 344 rats (60/sex/group) used in this study had a spontaneous rate of developing osteosarcoma. Doses of 5 and 30 μ g/kg were also given; all rats were treated for up to 2 years or to their spontaneous death; any rats alive at the end of 2 years were sacrificed. A necropsy was conducted on each rat. Bone mass markedly increased as measured by quantitative computed tomography and histomorphometry. Substantial new bone formation resulted in a large decrease in marrow space accompanied by altered bone formation. Osteosarcoma occurred in 3, 21, and 31 male rats and in 4, 12, and 23 female rats in the 5-, 30-, and 75- μ g/kg treatment groups, respectively. Focal osteoblast hyperplasia, osteomas, and osteoblastomas were also seen but less frequently. It was felt that the exaggerated effects of chronic stimulation of osteoblasts by PTH (1-34) resulted in the bone neoplasms. At that point in time, the authors felt that other related compounds that produce a similarly profound bone anabolic response in the rodent would also result in bone proliferative lesions following near-lifetime treatment. Thus, this issue would be expected with other PTH and PTH-related peptides, including PTH (1-84), PTH (1-38), PTH (1-31), PTHrP (1-34), and PTHrP (1-36), which have anabolic activity on the rat skeleton through interactions with the PTH/PTHrP receptor.²³⁰ An additional study was performed in 60 purpose-bred, mature herpes B- and simian retrovirus-free female cynomolgus monkeys who were estimated to be at least 9 years of age and skeletally mature, based on closed physes. The monkeys were injected with PTH(1-34) 0 or 5 μ g/kg/d for 18 months, and then followed for up to an additional 36 months. At the end of

18-month treatment period, 6 monkeys from each group were sacrificed to perform an interim analysis. The remaining animals were sacrificed 4–5 years after the initiation of the study. All monkeys underwent necropsies. There was no evidence of bone neoplasia in either the control or PTH(1-34) groups after 18 months of treatment or 36 months of follow-up.²³¹

The PTH (1-34) used in a rat toxicology study was negative in a battery of in vitro and in vivo genotoxicity tests; all tests were negative, supporting the conclusion that the bone proliferative lesions in the rat toxicology study were produced through a nongenotoxic, receptor mediated mechanism.²³⁰

The relevance of the bone proliferative changes observed in the 2-year rat toxicology study is uncertain, but there are several issues to consider: 1) rats were treated for 2 years, which is approximately 80%–90% of their expected normal life span; in comparison, the anticipated duration of therapy for humans is approximately 24 months, which represents only 2%–3% of their life span; 2) in terms of bone growth, rats were treated for about 25–30 bone-turnover cycles; again, in comparison, the expected treatment duration in osteoporotic women is 1–3 bone-turnover cycles; 3) there are fundamental differences in bone physiology between rats and humans, including longitudinal growth of bones throughout much of the rat's life and the near absence of osteonal remodeling in rats; and 4) the prolonged duration of treatment combined with the considerable sensitivity of the rat skeleton to the pharmacologic effects of PTH resulted in chronic hormonal stimulation of the osteoblast and highly exaggerated increases in bone mass that were in considerable excess of the effects observed in nonhuman primates and humans.²³⁰ Therefore, Vahle and his numerous coauthors concluded that in adult humans (with mature skeletons), in whom such exaggerated pharmacologic effects do not occur, it is unlikely that the risk of bone neoplasia would be increased by daily treatment with PTH (1-34) for a relatively small fraction of the normal life span.²³⁰

PTH (1-34), teriparatide, a recombinant human parathyroid hormone analog (1-34), [(rhPTH (1-34))], as brand name Forteo, was approved by the FDA on November 26, 2002 with an indication for treatment of postmenopausal osteoporosis in women at high risk of fracture. Because of a higher incidence of side effects in the 40- μ g dose, the 20- μ g dose became the approved dose. Because of the advent of the rat toxicology study results, the Neer study²²⁹ was terminated at 19 months (longest subject treated 21 months; personal communication). Subsequently, after further analysis of those data and their relation to human skeletal physiology, in a boxed warning, in which the rat osteosarcoma data was presented, it was decided that the use of the drug for more than 2 years “is not recommended.” Also, because of the uncertain relevance of the rat osteosarcoma finding to humans, it is advised to only use Forteo for patients for whom potential benefits outweigh potential risk. In addition, Forteo should not be prescribed for patients at increased baseline risk for osteosarcoma, e.g., those with Paget disease of bone or unexplained elevations of alkaline phosphatase, as well as pediatric and young adult patients with open epiphyses or prior external beam or implant radiation therapy involving the skeleton.²³² Subsequently, Forteo received approval for two

other indications: increase of bone mass in men with primary or hypogonadal osteoporosis at high risk of fracture; and treatment of men and women with osteoporosis associated with sustained systemic glucocorticoid therapy at high risk of fracture.²³²

While the clinical trial was bringing Forteo to FDA approval, there was great preclinical interest in fracture healing by the use of intermittent PTH (1-34) in rat and other fracture models. Among these studies, Andreassen and colleagues used 3-month-old female Wistar rats, given appropriate doses of hPTH (1-34) in a fracture model. External callus volume was increased 77% in the high-dose hPTH (1-34) group versus the lower-dose group and by 99% versus the vehicle group. The callus formation and the mechanical strength improvements were measured at 20 and 40 days after the fractures were created. A variety of mechanical strength tests were also increased in the high-dose treatment group. In conclusion, this early experiment showed that intermittent administration of high dose PTH (1-34) was able to improve fracture healing.²³³

Nakijima et al., with a different fracture model of the right femur in 2-month-old male Sprague-Dawley rats treated with PTH (1-34) of 10 µg/kg/day versus vehicle, showed at day 14 that bony callus, ultimate load to failure, BMC, and BMD were increased in the PTH group. Likewise, serum osteocalcin, a bone formation marker, was significantly higher in the PTH-treated group compared with controls, and the mRNA expression of bone matrix proteins was markedly increased. They were able to show that the anabolic effects of PTH on bone formation are mediated by IGF-I early in the healing process. In the later stages of fracture healing, the PTH effect was IGF-I independent. They concluded that “intermittent low-dose treatment with human PTH (1-34) may represent an effective strategy for the enhancement of fracture healing and could become the first systemic intervention for the repair of skeletal injuries.”²³⁴

Additionally, in 2003, Jiang et al. reported the results of the bone biopsies from the Fracture Prevention Trial.^{229,235} A total of 102 subjects participated in the biopsy study and yielded a total of 51 paired iliac crest biopsy specimens. Static parameters from the 2D histomorphometry and 3D trabecular structural parameters were measured, creating one of the iconic images of osteoporosis treatment. Increased trabecular and cortical thickness was observed in most posttreatment biopsies. Thus, teriparatide was able to improve both cancellous and cortical bone structure. The micro-CT images suggested that the increased cortical thickness resulted from increased bone formation at both the periosteal and endosteal surfaces.

Because of the preclinical work, clinical study, and the approval of teriparatide for postmenopausal fracture reduction, during the MLB season of 2004, it was used to treat a lefty-hitting baseball player who was hit by a pitch, fracturing the fourth metacarpal bone in his right hand. Team orthopedists predicted it would take 6–8 weeks to heal. He was started on 20 µg per day of teriparatide; his fracture was healed according to x-ray and CT scan in 3 weeks and he returned to play in 4 weeks. Thus began our experience in treating acute fractures, stress fractures, stress reactions, and delayed and nonunion fractures to speed and improve fracture healing. All of these uses are off-label. We have used teriparatide in over 2700 cases in our

practice, of which over 500 have been for the off-label uses, previously described.

In 2007, Schwartz reported on the first use of teriparatide in speeding fracture healing and initiating healing of nonunions in humans.²³⁶ A further report by the same group followed in 2008 that looked at teriparatide-induced stimulation of osteoblast function to stimulate stress fracture healing.²³⁷

Since that time, there have been several reports of different forms of PTH in fracture healing. Aspenberg et al. studied the use of teriparatide for acceleration of fracture repair in humans in a prospective, randomized, double-blind study of 102 postmenopausal women with distal radial fractures that were treated with closed reduction. The primary objective of the study was to compare the effect of 8 weeks of once-daily subcutaneous treatment with teriparatide 20 or 40 µg versus placebo on time to radiographic healing in postmenopausal women with a unilateral dorsally angulated distal radius (Colles) fracture. Radiographic healing was defined by cortical bridging in three of four cortices. A few other secondary endpoints were chosen for further evaluation. The estimated median time from fracture to first radiographic evidence of complete cortical bridging in at least three of four cortices was 9.1, 7.4, and 8.8 weeks in the placebo, teriparatide 20 µg, and teriparatide 40 µg groups, respectively. In actual fact, the time to healing was not shorter in the 40 µg group compared to placebo, but the time to healing was shorter in the 20 µg group compared to placebo ($p < 0.001$) and in the 20 µg teriparatide group as compared with the teriparatide 40 µg group ($p < 0.03$). Although the primary hypothesis that teriparatide 40 µg would shorten the time to cortical bridging was not supported, post hoc analyses suggested that the clinically approved teriparatide 20-µg dose significantly shortened median time to healing in three of four cortices compared with placebo. It was felt that the reason for this unexpected finding might be that, in the Neer trial, 40 µg was associated with a decreased BMD in the cortex of the radius shaft due to increased bone remodeling (what we would now call “cortical porosity”). They felt that teriparatide increased the number of early healers: at 7 weeks, approximately 10% of the placebo group had healed compared with 20% and 40%, respectively, for the teriparatide 40 µg and 20 µg groups. They also felt that teriparatide might reduce the risk of nonunion and that a reduction in healing time could also have “economic importance for the young, active patient.” Also, of importance, the 8-week treatment time resulted in the discontinuation of teriparatide before radiographic healing occurred in all patients. They concluded that the shortened time to healing with 20 µg of teriparatide compared with placebo suggested that fracture repair could be accelerated but that this might need to be shown with other types of fractures.²³⁸

Another key study was performed by Peichl and associates using PTH (1-84), the “complete” PTH molecule, to accelerate fracture healing in pubic bones and/or ischial rami of the pelvis of elderly osteoporotic women. Performed in Vienna, Austria, this was a randomized blinded study of 100 µg PTH (1-84) (which was not available in the United States at that time but is now approved as of January 23, 2015, in the United States, for the treatment of hypoparathyroidism) once-daily

as a subcutaneous injection. Older patients with pelvic pain were admitted to the hospital and underwent evaluation with DXA, X-ray of the pelvis, CT scan of the pelvis, and bone turnover markers, PTH level, and 25 hydroxyvitamin D level. For patients with pelvic fractures, the CT scan was repeated and functional progression was assessed every 4 weeks. All patients received 1000 mg of calcium and 800 IU of vitamin D. Twenty-one patients were treated with PTH (1-84) and 44 patients served as controls. Fracture healing was assessed on CT scans at weeks 0 (at least 2 days after the fracture) and at 4, 8, 12 weeks and later until evidence of fracture healing (defined as cortical bridging) was confirmed. The percentage of fractures that had healed at week 8 was considered to be the primary endpoint. Assessment of the CT scans was blinded to treatment arm. The median time from the fracture to the first signs of complete cortical bridging of the pelvic fracture on CT scan was 7.8 weeks for the treatment group, compared with 12.6 weeks for the control group ($p < 0.001$). At week 8, the primary endpoint, all fractures in the treatment group and four fractures in the control group had healed (healing rate 100% compared with 9.1%). The treatment group also demonstrated significant improvement in functional outcome compared with the control group. At week 12, all fractures in the treatment group and 30 fractures (30/44) in the control group were healed (68.2%). The mean time to healing for the fourteen fractures that had not healed by week 12 was 14.9 weeks (range 13–18 weeks). Laboratory monitoring was followed during the study. PTH was continued for a total of 24 months as the patients were being treated for osteoporosis.²³⁹

Thus, the Peichl study²³⁹ differed from the Aspenberg study²³⁸ in that in the former study, treatment was continued for 24 months, and in the latter study, treatment was arbitrarily given for only 8 weeks.

Since the time of these reports, additional studies have been reported by Ohtori et al. on other uses of teriparatide in orthopedic applications that show that teriparatide accelerates lumbar posterolateral fusion in women with postmenopausal osteoporosis and reduced pedicle screw loosening after lumbar spinal fusion surgery from a bone quality perspective in the same population.^{240,241}

As part of the development of the Forteo program, there has been monitoring of the potential occurrence of osteosarcoma from the beginning. In a letter to the editor of the *Journal of Bone and Mineral Research*, published online on November 13, 2006, Harper et al. reported one case of osteosarcoma at a time when >250,000 patients in the United States and >300,000 patients worldwide had been treated with an estimated background incidence of osteosarcoma in the general population of men and women ≥ 60 years of age of 1 in 250,000 per year. "The identification of this case does not change the risk/benefit profile for Forteo."²⁴² In 2010, Subbiak and colleagues reported a second case of osteosarcoma that occurred within a year of the previous reported case. This case, of note, was in a man with prostate cancer who received proton beam therapy to the bed of the prostate 9 years after the original diagnosis for local recurrence; he had taken Forteo for 2 months for osteoporosis when a 6 x 5.7 x 5.5 cm high-grade chondroblastic osteosarcoma was discovered. The authors felt, for a variety of stated reasons, that the osteosarcoma was probably due to the previous radiation therapy,

because the latent period of 7 years is characteristic of a radiation-induced osteosarcoma, the tumor occurred in the radiation field, and the short duration of Forteo treatment.²⁴³ An additional case was reported at the ASBMR 31st annual meeting as a poster (SU 0354),²⁴⁴ but upon consultative review, this was determined to be a neural sheath tumor and not an osteosarcoma (personal communication). Forslund et al. reported a unique case of a complex patient with malignant (multiple) myeloma after a course of teriparatide 20 μ g daily from June 2005 to January 2007. In September 2004, an MRI demonstrated multiple vertebral compression fractures with subsequent vertebroplasty. No evaluation of her serum protein (e.g., SPEP) was done at that time (although many would consider a presentation with multiple new vertebral compression fractures to be an indication for a thorough myeloma workup). In September 2006, her renal function was said to be normal, and in May 2007, she was found to have significant renal failure and proteinuria with a kappa M-spike with very high serum free kappa light chain level; the beta-2-microglobulin level was also very high. There was a question raised by the authors as to whether or not the gammopathy could have predated the course of teriparatide, but no conclusion about this status was made.²⁴⁵ Clinically, it seems certainly probable that myeloma predated the initiation of Forteo treatment. Please see the full online chapter for more information regarding potential side effects and risk for Forteo.

As part of the safety surveillance program, the FDA required the manufacturer to perform several postmarketing studies. In 2012, Cipriani et al. reported on the safety of osteoanabolic therapy after the first decade of use. They reviewed PTH mechanisms of action, PTH efficacy, PTH in men, PTH in glucocorticoid-induced osteoporosis, and PTH (1-84) use in hypoparathyroidism, its role in accelerated fracture healing, and, most importantly, safety. These relatively rare cases of osteosarcoma would appear to be consistent with the epidemiology of osteosarcoma in adults. The incidence of osteosarcoma was felt to be similar to unselected populations of adult humans who develop osteosarcoma and are not receiving PTH. The incidence of hypercalcemia with teriparatide was 11% and was observed 4–6 hours after the injection.²⁴⁶ Andrews and colleagues reported on the first 7 years of a 15-year mandated FDA postmarketing surveillance study. From June 2004 to September 30, 2011, 1448 cases of osteosarcoma (diagnosed 2003 to 2009) were reported to the 15 involved cancer registries in the United States. A subset of patients was interviewed. There were no valid reports of teriparatide use before the diagnosis of osteosarcoma was made. These authors emphasized the importance of understanding the potential latency period between the exposure to an inciting exposure and the appearance of an increase in the number of clinically observed cancer patients. In cancers where the time of the exposure is known, e.g., radiation-induced, the latency period has ranged from less than a year to more than a decade. The authors presumed that a study period of 15 years would be adequate to take any potential latency into effect. They stated that among the 16,000 patients who received teriparatide in controlled clinical trials and observational studies in the previous 15 years, the largest of which was 4000 patients treated for 2 years and followed for up to 2 years, no cases of osteosarcoma were reported. As of June 2012, over 1 million patients worldwide had received treatment with teriparatide with approximately 4 million patient-years. From that experience,

there were the three previously described cases of osteosarcoma of which one turned out to not be osteosarcoma and one occurred 2 months after initiating teriparatide (and this would be an unusually short latency period, making the previous radiation therapy a more likely etiologic factor; whether the teriparatide incited malignant change in a previous damaged tissue is unclear).²⁴⁷

There was also a similar study started a year later in five Nordic countries (Denmark, Finland, Iceland, Norway, and Sweden). Kellier-Steele reported the results of the first 12 years of the United States study through September 30, 2015, in which 939 osteosarcoma patients (or their proxies) were interviewed, and the results of the Nordic study that concluded in 2014 in which 112 cases were reviewed. In the United States study, one patient was identified who reported the use of Forteo prior to the diagnosis of osteosarcoma. No further details of this case are available. In the Nordic study, no patients were identified with a history of Forteo use prior to the diagnosis of osteosarcoma. The authors concluded, "Although there is a single case of prior FOR(S)TEO exposure in the US, evidence of a causal relationship between FOR(S)TEO treatment and OS [osteosarcoma] in humans has not been identified."²⁴⁸ As far as we know, no case of osteosarcoma has been reported after the use of PTH (1-84).²⁴⁹

In yet another study, Gilseman et al. reported on a patient registry linkage to multiple state cancer registries. This Forteo Patient Registry (FPR) was established in 2009 to estimate the incidence of osteosarcoma in US patients treated with teriparatide. This prospective registry was designed to link participants annually with state cancer registries. Patient enrollment is planned for 10 years (2009–2019) and annual linkage data collection for 15 years (2010–2024). For the eighth annual linkage in 2017, 63,270 patients contributed 242,782 person-years of follow-up. A total of 5268 adult osteosarcoma cases diagnosed since January 1, 2009 were available for linkage from participating state cancer registries. Projecting current enrollment rate to study end, it is anticipated that the completed study will be able to detect a four fold increase in the risk of osteosarcoma if one exists. To date, no incident cases of osteosarcoma have been identified among patients registered in the FPR.²⁵⁰

In the last 5–10 years, beyond the studies by Aspenberg²³⁸ and Peichl,²³⁹ there has developed a significant literature of anecdotal cases and review articles about utilizing the available anabolic agents off-label for various aspects of fracture healing, including acute fractures, delayed healing, and nonunions.^{251–254} In addition, several articles have approached the topic of how to evaluate the various products in order to create future adequate studies that might actually help achieve an indication for fracture healing.^{255,256} A full discussion of these issues is beyond the scope of this chapter. There has also been use of the available anabolic agents for resolution of stress reactions and treatment of stress fractures (again, off-label usage). In addition, in certain situations, we have utilized 40 µg of teriparatide per day for increased anabolic effect (also off-label usage; see below).

However, several of small studies deserve some comment. Johansson reported a series of 40 postmenopausal women with a proximal humerus fracture not suitable for operative treatment who were randomized to either daily subcutaneous injections with 20 µg of teriparatide for 4 weeks (starting within 10 days of the

fracture) or control treatment. The primary outcome was blinded radiographic findings of fracture healing and callus formation at 7 weeks. This study did not show any positive effect of teriparatide on the treatment of proximal humerus fractures, either radiographically or on pain and/or function. The author admitted that his study had several limitations: it may have been underpowered; the time of treatment may have been too short; it was not known if the optimal dose of teriparatide for fracture healing is 20 µg or 40 µg; and, finally, the study was not blinded because of lack of placebo pens.²⁵⁷ Bhandari et al. studied whether or not teriparatide would improve femoral neck fracture healing after internal fixation as measured by frequency of revision surgery, radiographic fracture healing, and other safety outcomes including pain control, gait speed, and safety. Two separate but identically designed double-blind placebo-controlled Phase III clinical trials to meet FDA criteria to gain a new indication were initiated to evaluate the effect of teriparatide 20 µg for 6 months subcutaneously versus placebo. The effect was to be evaluated at 24 months. The trials were conducted concurrently with a planned enrollment of 1220 patients per trial. However, the trials were stopped due to poor patient recruitment and enrollment, and only a total of 159 patients were randomized: 81 placebo and 78 teriparatide. The combined program had very low power to detect the originally expected treatment effect. There was no difference between the groups in the proportion of patients achieving radiographic healing at 12 months (75% [61 of 81] placebo versus 73% [57 of 78] teriparatide). One of the comments by the authors was that plain radiographs obtained at various intervals may be too crude for evaluation of femoral neck fracture healing.²⁵⁸

Several of our cases illustrate our approach to the problems of these studies.

Case 1

Thirty-seven-year-old black male, 6'3 3/4", 292 lbs, previous stress fracture right navicular healed in past. Complaining of left foot pain; 6/04, given cortisone injection; 7/13/04, NMBS & MRI left navicular fracture; low-intensity pulsed ultrasound bone growth stimulator initiated at 20 min/d; 9/10/04, CT: stress fracture mid portions left navicular bone; nonunion navicular fracture; 10/6/04, ORIF: take down cystic nonunion of navicular fracture; internal fixation w/2 cannulated screws; insert Osteogenic Protein – 1 (OP No1) and BMP – 7; 10/22/04, postop x-ray 2 screws across navicular non union; EBI bone stimulator 10 hrs/d; 12/16/04, post op CT "appears to be healing;" disuse osteoporosis; 3/15/05, CT still fracture lucency; 4/2/05, recurrent pain in foot; inject 1 cc beta-methasone (Celestone Soluspan); 7/05, 4–5 shock wave treatments; 7/26/05, new horizontal fracture through navicular bone; 8/18/05, short leg cast; using restart bone growth stim; 9/15/05, CT scan 2 navicular fractures (Fig. 4.6).

11/3/05, CT scan subtle progressive healing along the dorsal aspect of the vertical navicular fracture; osseous bridging over prior cleft; 1/27/06, referral from orthopedist for consideration of teriparatide treatment for fracture healing; office visit: informed consent obtained and Black Box Warning discussed; Citracal +D one BID started; a diagnosis of nonunion navicular fractures was made; teriparatide injection training was conducted; Lab drawn and obtained: Ca 10.0 mg/dL; 25 hydroxy vitamin D 35 ng/mL; osteocalcin 13.5 ng/mL (11.3–35.4); total alkaline phosphatase 108

FT

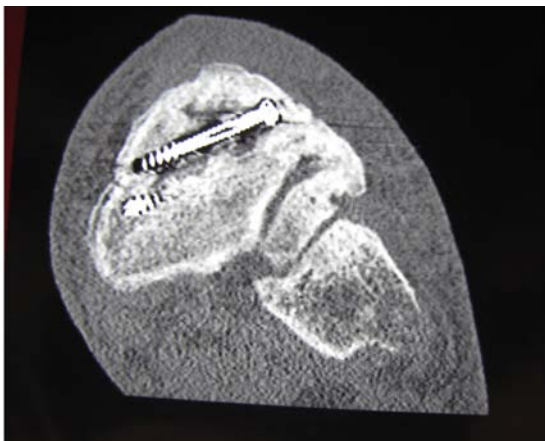


Fig. 4.6 CT navicular, Case 1.

U/L (30–115); BSAP 19.5 ug/L (7.7–21.3); Serum CTx 249 pg/mL (70–780); DXA (GE Prodigy Advance): LS 1.500 gm/cm², Z-score +1.7; Left Total Hip 1.447 gm/cm², Z-score +2.2; 1/3 Left Forearm (Radius) 0.874 gm/cm², Z-score +0.2; TBBM 1.607 gm/cm², Z-score +2.8; vertebral fracture assessment (VFA) no compression fractures. Trabecular Bone Score (TBS) 1.134; this value is less than 1.200 and is classified as “degraded” compared to a normal female Caucasian database; (now, less than 1.23; and, is, also, “degraded” compared to a normal male Black database [not officially approved by the FDA; used in research only, at this time]; retrospectively analyzed on 2-18-16); 1/30/06, teriparatide start date; dose 20 µg/day; continue bone growth stimulator (low-intensity pulsed ultrasound - LIPUS) but change to 20 minutes every 12 hours; 2/22/06, CT navicular bone: lucency with partial union distally; 2/22/06, increase teriparatide to 40 µg/day (2 consecutive injections from same pen with same needle); 5/6/06, Lab: PTH 59 pg/mL; 25 vitamin D 34 ng/mL; s-CTx 535 pg/mL; total alkaline phosphatase 158 u/L; BSAP 46.0 ng/L; 6/16/06, XR Left heel: fractures healed; 6/27/06 decrease teriparatide to 20 µg/d 8/8/06 total alkaline phosphatase 137 u/L; PTH 43 pg/mL; 25 vitamin D 20 ng/mL; osteocalcin 37.5 ng/mL; s-CTx 772 pg/mL; BSAP 52.4 ug/L 10/20/06 discontinue teriparatide. Teriparatide restarted in 2-07 until 10-07.

Case 2

Abstract

Nonunion of a fracture is defined as permanent failure of healing following a fracture, usually by 6 months. The FDA defines nonunion for investigative purposes as a fracture 9 months old that has shown no signs of healing for 3 months. Nonunions occur in 10%–20% of the 6,000,000 annual fractures in the United States. The morbidity of nonunions is extensive and expensive. The treatment of nonunions is usually surgical, but the development of osteoanabolic agents suggests there may be another approach. However, the duration of time of treatment to “heal” a nonunion is unknown. Numerous individuals suggest that this treatment may take weeks to a few months. We evaluated a 66-year-old morbidly obese white male who fractured his right medial malleolus after 7 weeks in an exercise class without obvious trauma; presumably, a stress fracture due to increased activity. A CT scan of the ankle 5 months later showed a nonunion. Because of morbid obesity and chronic obstructive pulmonary disease, surgical repair was deemed

inadvisable. A “secondary” osteoporosis laboratory workup showed prediabetes and hypogonadism. A DXA showed normal bone density. He was started on a low-dose pulsed ultrasound. A month later, he was started on teriparatide 20 µg subcutaneously daily. A month later, he was started on a heart-healthy diet and testosterone gel. He was followed with appropriate laboratory monitoring and serial CT scans at appropriate intervals. The CT scan performed at 1 year showed an incompletely healed medial malleolar fracture. A CT scan of the ankle performed 23 months after initiation of osteoanabolic treatment showed complete healing of the medial malleolar fracture.

History and Clinical Course

The patient, a 66-year-old, right-handed married white male, a medically disabled butcher, was seen for the first time on 9-2-11 for evaluation of nonunion of a right medial malleolar fracture. He stated that he didn’t know what happened to his ankle. He had been in a COPD exercise class for 7 weeks when his ankle “started to hurt.” He taped it up, but 1 week later, he saw his orthopedist. An x-ray in late April 2011 showed a fracture. No trauma. When he started the exercise program, “couldn’t hardly walk,” when done (7 weeks), “could walk for 1 hour without stopping.” Multiple previous fractures due to motorcycle accidents. Spinal fusion, 1983. Smoked 3 packs per day for 30 years, stopping 12–14 years ago. Intermittent prednisone for COPD. NSAID for 2–3 years; none for 6 months. “Probable” alcoholic; none for 15 years. Taking a multivitamin for years and vitamin D3 1000 IU “for a couple of months.” Tallest remembered height: 6’1”; height measured on wall-mounted stadiometer 5’9¼”. Weight 298lbs. BMI 43.7. Swollen right ankle. Testes small, soft, DRE negative. 9-3-11 Start Exogen low-dose pulsed ultrasound one treatment per day; 9-23-14 Start Heart-Healthy Diet; 9-24-11 Start teriparatide (Forteo) 20 µg subq daily; Increase vitamin D3 from 1000 IU to 2000 IU per day; 10-20-11 Start Androgel 1% 5 g pkt, apply one per day; 5-12-12 Increase vitamin D3 to 5000 IU per day; 8-26-13 Weight 266 lbs, down 32 lbs; 9-24-13 Stop teriparatide (Forteo), fracture healed; 8-21-14 Weight 261 lbs, down 37 lbs; Height measured on wall-mounted stadiometer 5’9½”; BMI 38; 1-year post-fracture healing follow-up.

DXA

These studies were performed on a Hologic Discovery A densitometer utilizing software version 13.3 (Table 4.9).

Imaging: 5-3-11; RT ankle XR: medial malleolar fracture, SAD type 9-8-11; CT RT ankle with reconstructions: nonunion of medial malleolar fracture which extends into the trabecular articulation; 12-13-11; CT RT ankle with reconstructions: interval healing of the vertically oriented fracture involving the medial malleolus without evidence for complete union; persistent nonunited fracture involving anterior caudal portion of the medial malleolus fracture with persistent articular extension; 2-13-12; CT RT ankle: Partially healed medial malleolar fracture with osseous bridging. Improved from 9-8-11 although appears similar to 12-13-11; 4-19-12; CT RT ankle with reconstructions: persistent incomplete union of the cortex of the articular surface of the medial malleolus. 6-18-12; CT RT ankle with reconstructions. partially healed medial malleolar fracture with osseous bridging noted. This has improved from 9-8-11 with persistent cortical break; 9-6-12; CT RT ankle with

reconstructions. Incompletely healed medial malleolar fracture; no interval change from prior CT of 6-18-12; 1-20-13; CT RT ankle with reconstructions. Incompletely healed medial malleolar fracture: no significant interval change since prior study from 9-6-12; 8-27-13; CT RT ankle. Healed medial malleolar fracture; no new fracture (Fig. 4.7).

Laboratory Results

9-8-11: Initial blood and laboratory workup; 12-18-11, 2-4-12, 4-31-12, 6-19-12, 9-7-12, 9-27-13, 8-5-14: Follow-up laboratory studies notes particularly the increasing and decreasing response of the anabolic BTOM, P1NP (Tables 4.10 and 4.11).

TABLE 4.9 Dual-Energy X-ray Absorptiometry (DXA)

	EXAM DATE: 9/2/11			9/13/2012	9/24/2013 ^A	8/25/2014
	BMD g/cm ²	T-Score	Z-Score	BMD g/cm ²	T-Score	Z-Score
AP Spine (L1–L4)	1.506	3.8	4.6	1.624	1.801	1.766
Femoral Neck (FN) (right)	0.954	0.2	1.2	0.899	0.990	1.067
Total Hip (TH) (right)	1.087	0.4	0.9	1.108	1.077	1.237
1/3 Forearm (FA) (right)	0.780	−0.7	0.4	0.787	0.799	0.799
VFA	No VCF			No VCF	No VCF	No VCF
TBS ^b	1.127			1.019		1.166

^APerformed on an outside Hologic Discovery A densitometer utilizing software version 12.7.3.2.

^bScore > 1.350 = “normal”; > 1.200, < 1.350 = “partially degraded”; < 1.200 = “degraded”

AP, anterior-posterior; BMD, bone mineral density; TBS, trabecular bone score (Medimaps, Geneva, Switzerland); VCF, vertebral compression fracture; VFA, vertebral fracture assessment

TABLE 4.10 Laboratory Results for Case Study 2

	9/8/11	12/18/11	2/4/12	4/31/12	6/19/12	9/7/12	8/27/13	8/5/14
CBC	Hgb 13.1: Hct 38.9	Hgb 13.1: Hct 38.9						
CMP	Glu 90: Creat 0.86	Glu 159						
HgbA1c	6.0%	6.3%		5.7%	5.7%		5.6%	5.8%
25 VD	31	38	33	26	37	40	46	54.4
1.25 VD	37 (15.75)							
SPEP	No M Protein							
Estradiol	< 20 (0.47)							27
TSH	0.89						0.64	1.010
ft4	0.7						0.7	1.16
Ttestost	90 (175–781)	857		683	>1500		1163	860 (348–1197)
Ftestost	14 (47.244)	229					283	19.0 (6.6–18.0)
PTH	42 (14.0–72.0)	35.4	43		36.9		26.7	24
TTG Ab, IgG	< 1 (< 6)							
TTG Ab, IgG	< 1 (< 6)							
Gliadin Ab, IgA	3 (< 19)							
Gliadin Ab, IgG	9 (< 19)							
Homocysteine	9 (< 19)						9	8.7
Carotene	8 (4–13)							18
Osteocalcin	90 (60–200)	38	44		52	58	41	7.4
P1NP	54 (22–105)	136	175	143	111	138	62	29
BSAP	14.4 (6.5–20.0)	17.3	21.9	20.8	16	21.5	12.0	8.0
sCTx	183 (87–345)	415	437	560	765	534	302	240
uNTx	28	38	39	57	45	44		20
Creat Ckear	113						102	
U Calcium	133	147		213			285	192
PSA				0.43			0.66	0.7
Ca	94	9.5			8.8	9.4		
P	3.0	3.7			2.9	3.2		2.5
Ferritin								88

TABLE 4.11 Laboratory Results for Case Study 2

	9/8/11	12/18/11	2/4/12	4/31/12	6/19/12	9/7/12	8/27/13	8/5/14
Osteocalcin	19 (11–50)	38	44		52	58	41	7.4
P1NP	54 (22–105)	136	175	143	111	138	62	29
BSAP	14.4 (6.5–20.0)	17.3	21.9	20.8	16	21.5	12.0	8.0
sCTx	183 (87–345)	415	437	560	765	534	302	240
uNTx	28	38	39	57	45	44		20



Fig. 4.7 CT case 2. (A) Initial stress fracture (arrow) and showing nonunion at 5 months postinjury. (B) CT showing incompletely healed medial malleolar fracture at 1 year post-initiation of teriparatide. (C) CT showing completely healed medial malleolar fracture at 2 years post-initiation of teriparatide. CT, computed tomography.

Conclusions

Current osteoanabolic therapy can heal fracture nonunions, but the length of time to do so may be prolonged.

Case 3

Professional basketball center; Seen in original consultation on 5-19-15; The last time his foot was entirely well was early in the 2011/2012 season; it began to be “sore;” it was evaluated with imaging, and he was told there was “a fracture;” (Jones fracture); he underwent surgery with placement of a screw. He played 3–5 games in 2011/2012 season starting after the All-Star Game but the foot “didn’t feel right;” imaging was performed and he was told his fracture “was not healed” and he was shut down for the rest of the season. He played the 2012/2013 season with his foot “pain free;” at the end of the season, an x-ray showed the screw was bent, and so a bigger screw was placed during the off-season, but he was told there was “still a tiny line.” He played 20 games of the 2013/2014 season but he had recurrent foot pain; a new screw was placed and a first metatarsal osteotomy was performed along with an iliac crest bone graft; he didn’t play again that season. He was given teriparatide for 2 months, from January to mid-March 2014; he was given a bone growth stimulator, which he used for a “period of time.” He played throughout the 2014/2015 season, but at the end of the season an x-ray showed “a line.” He was given a new, different bone growth stimulator to use along with the previous bone growth stimulator, which was restarted; he was also restarted on teriparatide; and he was given a PRP or stem cell

injection with another scheduled for a week after his first office visit. He had a vitamin D level performed 3 years previously, which he was told was “below average,” but he was not told to take vitamin D, nor was the level ever repeated. He was taking dairy products in his diet but no supplemental calcium. PMH, FH, SH, and ROS were otherwise unremarkable.

His DXA showed an AP Spine (L1–L4) Z-score +1.6; Right Femoral Neck Z-score +2.5. TBS = 1.458 “normal.” 25 vitamin D level was 25 ng/mL (normal: 30–100). He was treated with high-dose vitamin D3. His laboratory workup for causes of secondary metabolic bone disease was normal or negative. His bone turnover markers to measure bone resorption and bone formation were normal. A specimen to the Collagen Diagnostic Laboratory at the University of Washington showed no abnormality in bone genes analyzed. He was restarted on teriparatide 20 µg subcutaneously daily, one bone growth stimulator 20 minutes twice a day, and followed with appropriate lab and imaging. Imaging showed complete healing of the right fifth metatarsal fracture after 15 months. Teriparatide was continued through the end of the next season (Figs. 4.8 and 4.9).

Abaloparatide (Tymlos) PTHrP (1-34)

In April 2017 (4-28-17), the third anabolic drug, abaloparatide, which is indicated for treatment of postmenopausal women at high risk of fracture, was approved for patient use by the FDA as Tymlos. Abaloparatide is a novel synthetic analog of human parathyroid hormone related peptide (PTHrP [1-34]). PTHrP

Stress Fracture NBA Center
5-7-15

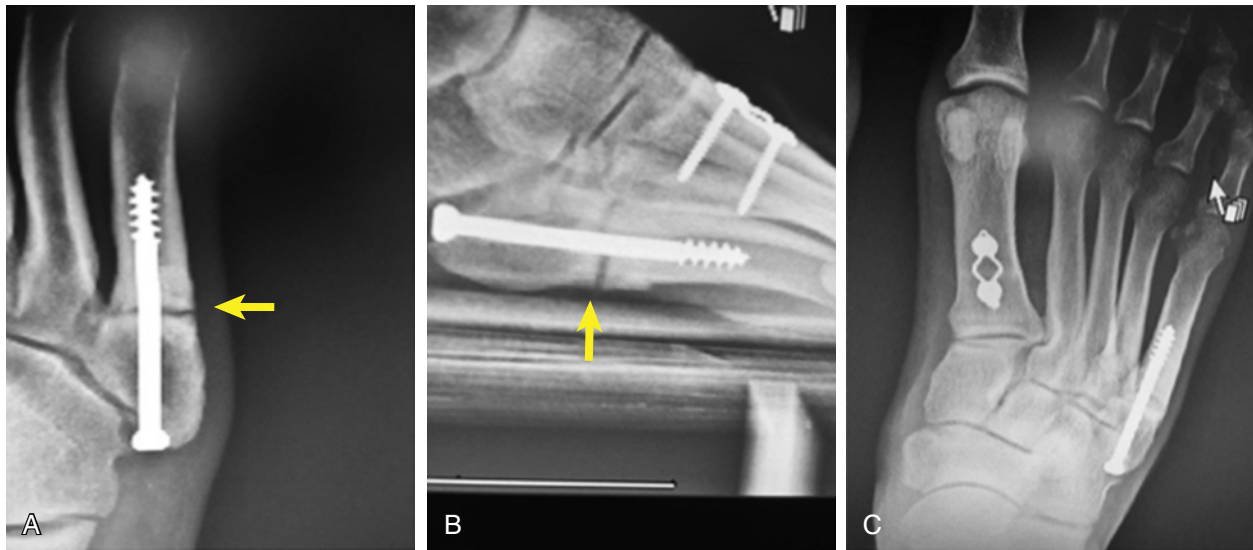
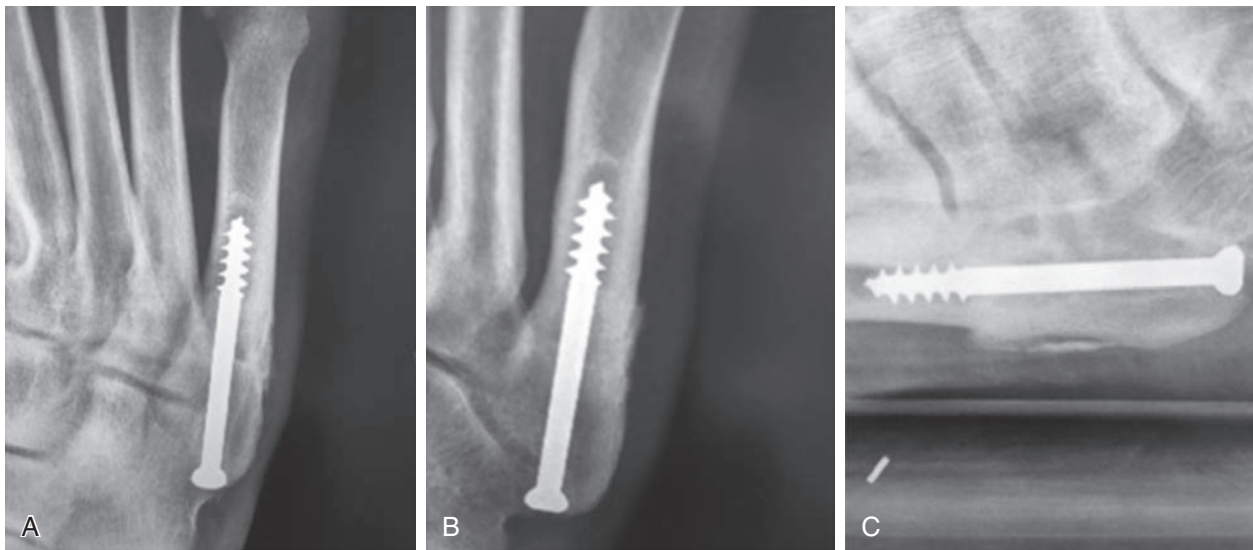


Fig. 4.8 Case 3,1 (A) 5th metatarsal fracture, S/P 3rd ORIF, nonunion, AP View. (B) 5th metatarsal fracture, S/P 3rd ORIF, nonunion, Lateral View. (C) X-ray of right foot.

Stress Fracture NBA Center
Patient B



X-ray August 2016

Fig. 4.9 Case 3,2 (A) 5th metatarsal fracture, healed after 15 months of teriparatide. (B) 5th metatarsal fracture, higher power view. (C) 5th metatarsal, lateral view.

is a paracrine-acting morphogenic factor that regulates cell proliferation and differentiation in developing bone and other tissues. It works through its action on the parathyroid hormone receptor type 1 (PTHr1), which mediates the actions of both PTH and PTHrP. Abaloparatide has 76% homology to human PTHrP (1-34) and 41% homology to human PTH (1-34) due to substituted amino acid residues in the peptide.²⁵⁹ Similar to teriparatide, a study to determine the carcinogenic potential of abaloparatide performed in Fisher F344 rats administered subcutaneous daily abaloparatide at doses of 0, 10, 25, and 50 $\mu\text{g}/\text{kg}$

of abaloparatide or 30 $\mu\text{g}/\text{kg}$ of PTH (1-34) as a positive control for up to 2 years. This study resulted in a dose- and time-dependent formation of osteosarcomas with a comparable response to PTH (1-34) at similar exposure.²⁶⁰

The registration trial, known as the Abaloparatide Comparator Trial in Vertebral Endpoints (ACTIVE), was a Phase III, double-blind, placebo-controlled trial in which 2463 postmenopausal women with low bone density and a significant percentage of prevalent vertebral and nonvertebral fractures were randomized to daily subcutaneous injections of placebo

($n = 821$), abaloparatide 80 μg ($n = 824$), or open-label teriparatide 20 μg ($n = 818$) for 18 months. Both abaloparatide and teriparatide reduced both vertebral and nonvertebral fractures. Of importance for this chapter are some of the details of the study. Daily administration of abaloparatide resulted in a 93% increase in the bone formation marker, P1NP, that was maximum at 1 month with a decline over the next 17 months so that at conclusion of this study, the P1NP was 45% above baseline. The bone resorption marker, s-CTX, was 43% above baseline at 3 months and was 20% above baseline at 18 months. This time course of bone remodeling from abaloparatide is somewhat different than that of teriparatide. Teriparatide reached its maximum bone formation at 6 months which plateaued until 12 months and then declined to 18 months at the conclusion of the study. The teriparatide curve of the bone resorption marker exceeded the respective curve of the abaloparatide bone resorption marker and was much higher than abaloparatide at the conclusion of the study. The BMD changes of abaloparatide at the LS spine were 5.9% at 6 months, 8.2% at 12 months, and 9.2% at 18 months; for teriparatide, the changes were 4.8% at 6 months, 7.4% at 12 months, and 9.1% at 18 months. At the total hip, the BMD in the abaloparatide patients increased by 2.1% at 6 months, 2.9% at 12 months, and 3.4% at 18 months; the corresponding changes in the teriparatide subjects were 1.3% at 6 months, 2.0% at 12 months, and 2.8% at 18 months, respectively. At the femoral neck, the abaloparatide increases were 1.5% at 6 months, 2.2% at 12 months, and 2.9% at 18 months, whereas the corresponding changes in the teriparatide arm were 0.8%, 1.4%, and 2.2%, respectively. There is a suggestion, but one that is not evidenced-based at this time, that fracture reduction might begin earlier with abaloparatide than with teriparatide. Adverse events of each arm were similar. Of importance, adverse reactions due to hypercalcemia (albumin-corrected >10.7 mg/dL) were 0.4% for placebo, 3.4% for abaloparatide, and 6.4% for teriparatide.²⁶¹ However, because the Tymlos arm used its specific pen and the teriparatide arm used its specific pen, the study was not blinded for the anabolic agent with which the subjects were treated. Also, because of the size of the population in each arm, it is not possible to draw some of the conclusions we might like to draw about the efficacy of one anabolic versus the other.

In a post hoc analysis of a Phase II trial, abaloparatide given at multiple doses was evaluated for its effect on TBS. After 24 weeks, TBS increased significantly by 4.21% in the 80 μg per day group. In the teriparatide 20 μg daily group, TBS increased by 2.21%.¹⁴¹ Saag et al. reported on the TBS changes in a study of patients treated with chronic glucocorticoid therapy-induced osteoporosis. In the teriparatide-treated patients, the TBS increased significantly by about 2% at 18 months and by 3.7% at 36 months. There was no increase in TBS in the alendronate-treated group.¹⁴⁰

At the present time, the preclinical fracture healing data are limited to two reports. In the first abstract, 96 12-week-old male Sprague-Dawley rats underwent creation of a right femur closed internally stabilized fracture. Rats were treated with 5 or 25 $\mu\text{g/kg/day}$ or vehicle. Micro-CT of the fracture calluses showed that the abaloparatide-treated rats had greater callus bone volume, bone volume fraction, and BMC at weeks 4 and 6 than vehicle. Semiquantitative histologic scoring of cortical bridging across the fracture gap showed significantly greater

bridging and significantly greater callus area in the high-dose group compared with vehicle. Three-point bending tests at week 4 showed that callus stiffness was 60% and 96% higher in the abaloparatide groups and 112% higher at 12 weeks. The authors concluded that abaloparatide enhanced fracture healing in the model of femur fracture utilized in this study.²⁶² In the second abstract, Chandler et al., the results were interpreted as showing early increases in callus stability, perhaps related to the augmentation of chondrogenesis and osteogenesis representing a potential pharmacodynamic profile for improving fracture healing.²⁶³

At the time of writing, there are no human fracture healing data available for abaloparatide. Whether there will be a human fracture healing study undertaken by the company has not been announced at this time. Thus, any use of Tymlos in fracture healing at this time would be off-label.

Bisphosphonates

Bisphosphonates were discovered about 50 years ago and were found to have extensive effects on bone metabolism. They have been used clinically for 40 years for the treatment of a variety of metabolic bone diseases associated with excess resorption since the FDA approval of etidronate (Didronel) on September 1, 1977.^{264,265} Over the subsequent time period, the mechanism of action has been delineated that bisphosphonates inhibit bone resorption by selective adsorption to mineral surfaces and subsequent internalization by bone-resorbing osteoclasts where they interfere with various intracellular processes. There are less potent nonnitrogen-containing bisphosphonates (clodronate [not approved in the US] and etidronate). Most of the clinical experience has been with the more potent nitrogen-containing bisphosphonates (pamidronate [approval date: 9-22-98]; alendronate [approval date: 9-29-95]; risedronate [approval date: 3-27-98]; oral ibandronate [approval date: 5-16-03] and intravenous ibandronate [1-9-06]; and zoledronic acid [approval date as Zometa: 8-20-01 and as Reclast: 8-17-07]). These nitrogen-containing bisphosphates inhibit a key enzyme, farnesyl pyrophosphate synthase, in the mevalonate pathway, thereby preventing the biosynthesis of isoprenoid compounds that are essential for the posttranslational modification of small guanosine triphosphate (GTP)-binding proteins (which are also GTPases) such as Rab, Rho, and Rac, the inhibition of protein prenylation, and the disruption of the function of these key regulatory proteins, explaining the loss of osteoclast function.^{266,267}

The clinical pharmacology of these drugs is characterized by poor oral absorption from the intestine ($<1\%$ – 4%) but highly selective localization and long-term retention in bone. Side effects, when properly used, are minimal, but in the last 10–12 years, the appearance of rare side effects (osteonecrosis of the jaw and atypical femoral fractures) has somewhat dampened the enthusiasm of their use, at least by the public if not physicians.^{268–270}

These drugs have numerous FDA-approved indications, including prevention and treatment of postmenopausal osteoporosis, male osteoporosis, glucocorticoid-induced osteoporosis, Paget disease of bone, hypercalcemia of malignancy, and numerous other oncologic indications. They have many off-label uses. They were originally given orally every 3 months (etidronate); to

orally daily (alendronate) to weekly (alendronate); to orally daily, weekly, and monthly (risedronate); to orally monthly and intravenously every 3 months (ibandronate); to monthly and yearly (zoledronic acid). The oral drugs have fastidious dosing recommendations: fasting (alendronate and risedronate) and postprandial (risedronate); and the intravenous drugs can be given as a bolus (ibandronate) or as a slow infusion (zoledronic acid).

Shima and colleagues reviewed the literature on the use of bisphosphonates for the treatment of stress fractures in athletes.² Controversy has existed over the years about whether or not bisphosphonates interfere with fracture healing, resulting in delayed healing or fracture nonunions. Little et al. suggested in a rat model that zoledronic acid could improve fracture healing by optimizing the amount and mineral content of the callus produced, which might prove to be of clinical benefit in obtaining fracture healing.²⁷¹

In 2005, five female intercollegiate athletes with bone scan positive lesions consistent with stress fractures were treated with intravenous pamidronate. Each patient received an initial test dose of pamidronate 30 mg intravenously over 2 hours; subsequently, the first three patients received 90 mg intravenously and the final two patients received 60 mg intravenously. They were given weekly infusions for a total of five treatments. The authors described marked improvement in pain within 48 to 72 hours during clinical evaluation and sport-specific participation beginning after the 30-mg test dose.²⁷² In another case series, Chambers reported the treatment of five lumbar pars interarticularis stress fractures in athletes with intravenous bisphosphonates. One received two infusions of pamidronate of 60 mg and four received 3 mg of ibandronate and three further infusions at 4-week intervals. They resumed play at 1–6 months after their final infusions. The longest follow-up was 45 months at which time that patient remained symptom free.²⁷³

In 2016, Eriksen reported the use of intravenous zoledronic acid to heal complicated stress fractures in the foot with delayed healing. Seven females and two males, aged 30–72, with nonunion of stress fractures for more than 12 months were studied. Intravenous zoledronic acid (5 mg) was given in two infusions 3 months apart. Pain was monitored and MRI was performed in six patients at baseline, 3, 6, and 12 months after the first infusion. All patients received calcium and vitamin D. They reported that all patients experienced clinical healing with significant reduction of pain at the fracture site with improvement of ambulation within 1–3 months after the first infusion. Four patients experienced further pain relief after the second infusion. At 6 months, all patients had normal ambulatory function and significant pain relief. He concluded that treatment with intravenous zoledronic acid represents a safe and effective treatment of delayed union of stress fractures of the foot, thus avoiding surgical intervention.²⁷⁴

In an iconic study, Milgrom, his team, and Burr looked at prophylactic treatment with risedronate on stress fracture incidence among infantry recruits in the IDF. Four hundred seventy-three new male infantry recruits, training on the same base between December 2002 and March 2003, were approached to participate in the study, and 324, median age 18.8 years, (range 18–28), agreed to do so. Subjects were randomly assigned to receive either risedronate or placebo.

Participants were given either 30 mg of risedronate or placebo daily for 10 days during the first 2 weeks of basic training before any significant training. After the 10-day loading period, treatment-arm subjects received a 30-mg maintenance dose weekly for 12 weeks. Side effects of medication were charted. Those with a suspicion of stress fracture were evaluated by the standardized IDF stress fracture protocol. For a variety of reasons, there were a lot of dropouts in the study. However, the results of the intention-to-treat analysis showed that prophylactic treatment with risedronate did not lower the incidence, time of onset, or severity of stress fractures.²⁷⁵

In 2009, Shima concluded his review with the statement: “...there is still no conclusive evidence to prove any effect of bisphosphonates on stress fracture healing in humans. Until the results of well-designed clinical trials are available, it is prudent to limit the use of bisphosphonates in the treatment of stress fractures.”² Despite the newer report by Eriksen,²⁷⁴ this conclusion still holds.

Bone Marrow Edema Syndrome

In the study by Eriksen, it is said that the subjects exhibited bone marrow (edema) lesions (BMLs) with and without fracture lines on MRI.²⁷⁴ Whether or not these lesions should be considered stress-related is not clear. Ringe et al., in their description in 2005, stated that BMLs constitute a rare disorder of localized high bone turnover of unknown etiology, self-limited; and that the lesion was characterized by the onset of disabling bone pain, usually at a single skeletal site, generally in the lower limbs, and especially the hips, in the absence of trauma. The entity has also been termed *locally transient osteoporosis* (LTO). The lesion is now diagnosed by MRI.²⁷⁶ The pathology of LTO has been evaluated in patients who underwent core decompression for the lesion. The specimens showed changes in the marrow and the bone. They showed marrow edema,^{277,278} thin seams of woven bone^{278,279} with active osteoblasts, thinning of the trabeculae, and osteoclastic bone resorption. There was no fat or bone necrosis.²⁷⁹ The lesion has been reported in the hip,²⁸⁰ the knee,²⁸¹ the ankle,²⁸² and foot,²⁷⁷ and has been treated with intravenous ibandronate²⁷⁶ and intravenous zoledronic acid²⁷⁸ to turn down what is essentially increased bone resorption and high turnover osteoporosis.²⁷⁶

In 1996, Schweitzer and White noted that altered biomechanics in weight bearing could produce marrow edema on MRI. They studied the hips, knees, and ankles of 12 asymptomatic volunteers (6 women, 6 men), age range 19–41, mean age 30 years, who, unilaterally, underwent the creation of a walking alteration by the placing of an extra-large, 9/16-inch longitudinal metatarsal pad underneath the lateral aspect of one foot to increase pronation. Images were obtained on a 1.5 T MRI system before (baseline), after 2 weeks of altered weight bearing, and 2 weeks after removal of the pad and return to normal ambulation. The MR images of 11 of the 12 volunteers showed changes of marrow edema in multiple bones consisting of a diffuse increase in marrow that varied between “subtle” and “intense.” Occasionally, the lesions appeared similar to stress fractures. At follow-up, the MR images returned completely to normal, although one subject demonstrated minimal persistent

marrow edema.²⁸³ Subsequently, in 1997, Lazzarini et al. asked, “Can running cause the appearance of marrow edema on MR images of the foot and ankle?” To determine this outcome, they imaged the feet and ankles of all runners of a university cross-country team. They found edema in 16 of 20 runners and in 4 of 12 nonrunners, but all subjects were asymptomatic. The total edema score and the number of bones with edema was significantly higher in the runners than in the nonrunners. Thus, running itself can account for marrow edema lesions. The authors concluded that the edema detected in these lesions on MRI might represent a continuum with stress fractures. Today, we would probably refer to these lesions as stress reactions.²⁸⁴

Another lesion in the spectrum of these bone marrow edema lesions is a “bone bruise” or “bone contusion,” which is an occult posttraumatic bone injury that may cause substantial clinical problems, since it is associated with acute and subacute pain and some degree of loss of function in a limb. These lesions are easily detected on MRI scans with decreased signal intensity on T1-weighted and increased signal intensity on T2-weighted images.²⁸⁵ The early time course²⁸⁶ and the later time course²⁸⁷ have been described. In 2012, Ucar et al. stated, “Currently, there is no definite consensus on the natural history of bone injuries secondary to minor knee traumas.”²⁸⁷ While it is generally thought that these lesions resolve in a short time, some persist for 6–24 months or longer.²⁸⁸ After trauma to the hip, knee, or ankle, individuals are sometimes left with persistent “bone bruise” (bone [marrow] edema) beyond the usual expected healing of the injury. These injuries produce persistent and/or lingering residual pain and discomfort that correlates with the bone edema seen on MRI, which does not resolve at the same pace as other symptoms or signs of inflammation. This entity reduces athletic performance and delays return to play. In these situations, an intravenous bisphosphonate (ibandronate or zoledronic acid) has been shown to reduce pain and disability and speed return to play. The ability to reduce bone pain in these benign disorders of increased bone turnover may be a property unique to intravenous ibandronate.²⁸⁹

From 2009 to the present, we have treated four professional basketball players and two intercollegiate basketball players with such a scenario as described previously, allowing earlier return to play. All were given 3 mg of intravenous ibandronate (the US FDA–approved dose) “push,” with one player receiving a second dose (3 mg) at 1 month after the first infusion. Pain began to subside within 7–10 days of the infusion. All returned to play after 1–8 weeks with reduction in pain and improvement in athletic performance based on clinical symptom and sign resolution. The MRI may or may not show improvement in the short term but will improve over the long term.²⁹⁰

Since our work on the use of intravenous ibandronate infusions in the treatment of elite athletes with bone marrow edema syndrome following a bone bruise, a number of further studies have been published.

Bartl and colleagues performed a prospective, observational, off-label study of intravenous ibandronate with a pain medication and partial weight-bearing group. The treatment group received three intravenous infusions of 6 mg of ibandronate monthly for 3 months versus a group that received

two 75-mg doses of diclofenac sodium per day for 3 weeks and nonweight-bearing status for 3 weeks, and then partial weight-bearing for 3 weeks. Standardized functional and pain scales were employed to evaluate the patients’ responses. MRIs of the affected regions were also obtained. Intravenous ibandronate produced “rapid and effective pain relief” based on the pain scores at 1 month, 6 months, and 12 months, with similar improvements in functional scores and improvements in MRIs compared with the pain medication arm. They concluded that intravenous ibandronate was an “effective treatment option for BMES [bone marrow edema syndrome] of the knee and ankle.”²⁹¹

Beckmann et al. studied 24 hips in 23 patients of which 12 were treated with off-label iloprost infusions coupled with core decompression, versus core decompression alone in bone marrow edema lesions. Iloprost is a synthetic analogue of prostacyclin PGI₂, which is a systemic and pulmonary arterial vasodilator. Iloprost was administered over 5–6 hours on 5 consecutive days with a starting dose of 20 µg on the first day, 30 µg on the second day, and 40 µg on the following 3 days. Core decompression and iloprost infusion decreased pain on the first postoperative day. The combination therapy was statistically significant over the monotherapies. There were fewer cases of persistent BMEL in the combined group as opposed to the monotherapy group radiologically.²⁹²

Baier and his group compared infusions of iloprost versus ibandronate. The investigators used the off-label iloprost infusion protocol of Beckmann versus the off-label ibandronate protocol of Bartl. It was felt that iloprost had certain advantages to the bisphosphonate, including rapid pain relief and a shorter time to complete the infusion protocol. Both medications helped the BMEL to resolve, and the differences from either were not significant.²⁹³ Fabbriani et al. reported a case of a 62-year-old man with a 2-month history of increasing pain in the left hip. MRI showed bone marrow edema. After further workup, which included finding significant vitamin D deficiency (9 ng/mL), and DXA of the proximal left femur that showed osteopenia, the patient was treated with teriparatide 20 µg subcutaneously daily. After 4 weeks, the patient was asymptomatic with no physical disability. An MRI showed almost normal signal intensity and DXA showed an 8% BMD increase in the left hip. From this one case, the improvement in the time course to resolution with an osteoanabolic drug as opposed to the previous antiresorptive or vasodilatory drugs seems impressive.²⁹⁴ Rolvien and his colleagues retrospectively studied 14 patients with atraumatic bone marrow edema syndrome who underwent a metabolic bone disease evaluation with 25 vitamin D levels, bone turnover markers, DXAs, and MRIs. Mean time from onset of pain and treatment with subcutaneous 60 mg of denosumab (Prolia) was 5.2 +/- 4.3 months (155 days). One patient had had a previous ibandronate infusion and two patients had had previous core decompression. Six to 12 weeks after injection, the bone marrow edema had resolved in 50% (7/14) of the patients and had improved in 6 other patients, thus demonstrating that denosumab is also an effective treatment for this syndrome.²⁹⁵

Simon and colleagues looked at the use of intravenous bisphosphonates and vitamin D in the treatment of bone marrow

edema in 25 professional athletes (3 women and 22 men). In five of the cases, a stress fracture was also present on the MRI in the region of interest. The time between the onset of pain and correct diagnosis was 106 ± 104 days. Sixty percent of the patients were considered vitamin D deficient; 44% had vitamin D levels between 20–31 ng/mL (insufficient), and 16% had vitamin D levels less than 20 ng/mL (deficient). All vitamin D abnormalities were corrected with high-dose vitamin D. After that was accomplished, all individuals were given 600 mg of calcium and 400 IU of vitamin D daily in tablets once a day. The treatment regimen consisted of an intravenous infusion of 3 mg of ibandronate. If there was no improvement, a second or third infusion was given after 4–6 weeks of the preceding infusion. Sixteen of the athletes (64%) noted pain reduction and improved mobility in the first 2 weeks after the first infusion; nine athletes got a second infusion, and two received a third infusion. All athletes returned to play at the previous level of function, but the group that only required one infusion returned to play at 78 ± 61 days; two infusions at 141 ± 47 days; and, three infusions at 164 ± 33 days.²⁹⁶

Bone Growth Stimulators

There is extensive literature on the development and use of bone growth stimulators. In the interest of full disclosure, I was involved in the development of a low-dose, pulsed ultrasound device and have used that device since it became clinically available about 1 month after its approval on October 5, 1994.

In a series of articles in Spanish (in 1983) and English (in 1987), Xavier and Duarte reported the acceleration of the normal fracture repair process and healing of ununited diaphyseal fractures in humans with the use of low-intensity ultrasound.^{297,298} In 1997, Heckman and colleagues reported on a multi-institutional, prospective, randomized, double-blind, placebo-controlled study of the acceleration of tibial fracture healing by a noninvasive, low-intensity pulsed ultrasound device. Ninety-six patients were recruited for the study but 13 were lost to follow-up for a variety of reasons, leaving 84 patients with 85 fractures. Another 17 subjects were excluded because of a variety of protocol violations, leaving 64 closed fractures (31 in the active-treatment group and 33 in the placebo group), and 3 open grade I fractures (2 in the active-treatment group and 1 in the placebo-treatment group). The fractures were treated with closed reduction and immobilization in an above-the-knee cast. Ultrasound treatment was started within 7 days after the fracture and consisted of one 20-minute period daily. Treatment was continued for 20 weeks or until the clinical investigator believed that the fracture was healed enough to discontinue the active or placebo treatment. The endpoint for the study was a healed fracture, as judged by clinical examination and on radiographic examination (three out of four cortices bridged). Also defined were several stages of intermittent healing, e.g., time to discontinuation of the cast, time to cortical bridging, and periosteal and endosteal healing. Mean time to a healed fracture clinically and radiographically was 96 ± 4.9 days for the active-treatment group compared with 154 ± 13.7 days for the placebo-treatment group. At 120 days after the fracture, 88% of the fractures in the active-treatment group were healed compared with 44% of

those in the placebo-treatment group. At 150 days, 94% of the fractures in the active-treatment group were healed compared with 62% of the placebo-treated group. There was variance for a number of other parameters: time needed for bridging of three cortices, time to complete cortical bridging, and time to complete endosteal healing, but all significantly favored the active-treatment group rather than the placebo-treatment group. Adverse reactions were minimal. However, in their discussion, the authors stated that the specific mechanism by which low-intensity pulsed ultrasound accelerated the fracture repair process was not known.²⁹⁹

Kristiansen and colleagues reported a study of healing of closed, dorsally angulated metaphyseal fractures of the distal aspect of the radius within 4 cm of the tip of the radial styloid using low-dose pulsed ultrasound where the fracture was initially treated by closed reduction and immobilization in a below-the-elbow cast. Eighty-three patients with 85 fractures were enrolled at 10 sites. Forty patients with 40 fractures were randomized to the active-treatment group and 45 fractures in 45 patients were randomized to the placebo-treatment group. Two patients had bilateral fractures, and one fracture was treated with an active device and one with a placebo device. Sixty patients fulfilled all aspects of the protocol. For both groups, the time from fracture to the start of treatment averaged 3 days. Time to a healed fracture was 61 ± 3.4 days for the active-treatment group compared with 98 ± 5.2 days for the placebo-treatment group; a significant acceleration of healing by 37 days (a 38% acceleration of healing). The percentage of organized trabecular healing at 5–16 weeks was significantly greater in the group treated with active ultrasound, and loss of reduction was significantly greater in the group treated with placebo device. Use of the active ultrasound device significantly reduced the time to healing in the group of smokers.³⁰⁰

In 2001, Rubin et al. conducted an extensive review of the literature. Numbers of studies pointed to the role of ultrasound on the biological processes of fracture healing, including its role on influx and efflux of ions into cells, increasing calcium incorporation in cartilage and bone cell cultures, and modulation of adenyl cyclase activity and transforming growth factor- β synthesis in osteoblastic cells, the increased release of platelet-derived growth factor, and the upregulation of aggrecan gene expression, all of which augment the processes of callus formation. These data suggest that besides moderating gene expression, ultrasound may increase blood flow through the dilatation of capillaries and increase angiogenesis to optimize fracture healing.³⁰¹ Rubin also reviewed Frankel and Lane's review of the patient registry of Exogen (Piscataway, New Jersey), which in June 2000 contained data on 22,300 patients; 10,500 patients had a 91% rate of healing; between 80% and 90% of the patients only had ultrasound as the new treatment, and 83% of the non-unions healed.³⁰¹

Over the last several years, there have been a number of meta-analyses of the last several decades of low-intensity pulsed ultrasound. These studies showed that LIPUS is effective for surgically managed, fresh, type C comminuted diaphyseal fractures of the lower limbs when there is appropriate stability at the fracture site.³⁰² In 2014, a National Institute for Health

and Care Excellence medical technologies guidance stated that the clinical evidence supports the use of Exogen bone-healing system in nonunion long bone fractures that have not healed after 9 months³⁰³; another study looked at treatment of chronic nonunion in a cohort of 767 patients where the heal rate was 86.2% with nonunion >1 year, 82.7% with nonunion >5 years, and 63.2% with nonunion >10 years³⁰⁴; in a systematic review, Rutten et al. evaluated 24 randomized trials in which time to radiographic fracture union was the most common primary outcome; their patient population (n = 429) had a mean reduction in healing time of 39.8 days, and the most reduction time was seen in fractures with a long natural healing time, but they could not show a beneficial effect of accelerated functional recovery or prevention of delayed union or nonunion³⁰⁵; in a retrospective, observational cohort of a convenience sample of patients with metatarsal fractures less than 1 year old enrolled in a registry, they found a heal rate of 97.4% for those treated with ultrasound, which was significantly better than for those treated without ultrasound (94.2%)³⁰⁶; Leighton et al. performed a systematic review and meta-analysis of 1441 nonunions treated with low-intensity pulsed ultrasound, which produced a heal rate of 82%, comparable to the heal rate of surgical treatment of noninfected nonunions.³⁰⁷

Schandelmaier and co authors, as part of the BMJ Rapid Recommendations process, created a systematic review of low-intensity pulsed ultrasound for bone healing. Their conclusions differed from other such studies; they felt that, based on moderate-to high-quality evidence from studies in patients with fresh fractures, LIPUS did not improve outcomes important to patients and probably had no effect on radiographic bone healing. However, they did state the applicability to other types of fractures is open to debate.³⁰⁸

Interestingly, there have been a number of studies over the last several years that looked at the effects of both low-intensity pulsed ultrasound and parathyroid hormone. Warden and colleagues,³⁰⁹ Naruse et al.,³¹⁰ and Mansjur et al.³¹¹ all reported data utilizing a rat model, which showed the combined treatment of PTH and LIPUS may accelerate fracture healing and enhance bone mechanical properties better than either single agent alone.³¹¹ We have been using the combination of a LIPUS unit and teriparatide for our patients with acute or chronic fractures or delayed union or nonunions.

Platelet-Rich Plasma Therapies

Please see the full online chapter for this information and [Chapter 29](#) for information on use of platelet-rich plasma.

Platelet-rich plasma (PRP) therapies are autologous blood products in which the platelets have been concentrated to a level greater than normal blood, and they have been used since the 1990s to promote bone and soft tissue healing.³¹² PRP can be manufactured at the bedside by centrifugation or filtering of a patient's whole blood to produce a small volume of fluid with a supraphysiologic concentration of platelets. Upon activation, the platelets contained in PRP release the following factors: ADP and ATP, angiopoietin-2 (Ang-2), connective tissue-activating peptide III (CATP III), epidermal growth factor (EGF), factor V, factor XI, factor XIII, fibrinogen, basic fibroblast

growth factor (bFGF or FGF2), fibronectin, insulin-like growth factor-I (IGF-I), osteocalcin, P-selectin (also called GMP-140), platelet-derived endothelial growth factor (PDECGF or thymidine phosphorylase), platelet-derived growth factor (PDGF), serotonin, transforming growth factor- β 1 (TGF- β 1), thrombospondin-1, vascular endothelial growth factor (VEGF), and von Willebrand factor (vWF).⁵⁶ A discussion of the production of PRP is beyond the scope of this chapter, but the concentration of platelets in PRP may vary according to the procedure used and the amount of plasma used to resuspend the platelets. Thus, the guidelines for PRP use need to be adhered to.³¹³

The Cochrane Collaboration has reviewed the topic on a number of occasions.^{314,315} In their 2012 review, Griffin and co authors found only one eligible study that met their selection criteria. Dallari and colleagues conducted a prospective, randomized, controlled study to evaluate enhanced tibial osteotomy healing. Thirty-three patients who had undergone a unilateral opening-wedge high tibial osteotomy for genu varum and osteoarthritis, with an opening defect of >1 cm on the medial side, were enrolled and randomly assigned to three groups: lyophilized bone chips with platelet gel were used to fill the defect in Group A; lyophilized bone chips with platelet gel and bone marrow stromal cells were used in Group B; and lyophilized bone chips alone were used in Group C. At 6 weeks, 12 weeks, 6 months, and 1 year after surgery, the patients underwent a clinical and radiographic evaluation. Histology and histomorphometry were also studied. The “final clinical outcome of lyophilized bone chips with added platelet gel or platelet gel and bone marrow stromal cells did not differ from that obtained with the use of bone chips alone.”³¹⁶ Their report concluded “that there remains insufficient clinical evidence to recommend the use of PRT [platelet rich therapies] in treatment of long bone fractures.”³¹⁴

However, in the Cochrane Collaboration report, the authors mention that one other study is currently under way involving hip fracture patients and “will provide further evidence concerning the use of PRT in the future.”³¹⁴ The study they were referring to was undertaken by three of the four authors of the Cochrane Collaboration report. This study, the Warwick Hip Trauma Study (The WHiT Study), was a randomized clinical trial comparing interventions to improve outcomes in internally fixed intracapsular fractures of the proximal femur. The protocol was reported in a first paper.³¹⁷ The design planned was a three-arm, single-center, standard-of-care controlled, double-blind, pragmatic, randomized clinical trial comparison between platelet-rich plasma and standard-of-care fixation versus standard-of-care fixation alone. The results of the study were reported in a second paper by the same authors and showed no evidence of a difference in the risk of revision surgery within 1 year in participants treated with PRT compared with those not treated with PRT.³¹⁸

Although the majority of studies are underpowered and consist of small case series or anecdotal studies, the role of PRT in soft tissue injuries may be completely different than its role in bone problems.³¹⁹

Stem Cells

Many of the stress fracture patients we see are referred for delayed union or nonunion. Therefore, any advances in fracture

healing are to be noticed, evaluated, and applied, if appropriate, to the problem of bone repair. Initial enthusiasm for repair by human embryonic stem cells has been stalled by many scientific, technical, political, and legal issues, whereas the search has developed for an “ideal” stem cell for clinical applications. Thus, the present approach has focused on mesenchymal stem cells (MSCs), which arise from blood, adipose tissue, skin, trabecular bone, and fetal blood, liver, and lung; they have also been identified in umbilical cord blood and placenta. Adult stem cells from the bone marrow are considered to have the highest multilineage potential. MSCs were able to differentiate into osteoblasts, chondrocytes, and adipocytes. These cells have been well characterized biochemically via cell surface markers. Autologous MSCs ease the accessibility of these cells for therapeutic needs. The use of stem cell–based therapies for the stimulation of fracture healing in nonunion has been an area of a great deal of research.³²⁰

Hernigou and Beaujean reported on the treatment of osteonecrosis of the femoral head with core decompression followed by an injection into the femoral head of aspirated anterior iliac crest marrow. The etiology of the osteonecrosis in 116 patients was steroids ($n = 18$), alcohol ($n = 32$), sickle cell disease ($n = 38$), organ transplantation ($n = 12$), idiopathic ($n = 10$), and “others” ($n = 6$); a total of 189 hips were treated. They were followed up for more than 5 years. Marrow was aspirated from the anterior iliac crests with the patient under general anesthesia. The aspirated marrow was pooled in plastic bags containing cell culture medium and anticoagulation solution. The aspirated material was reduced in volume to increase its cell count by removing red cells and plasma to leave the mononuclear cells; this resulted in a 150-mL marrow aspirate being reduced to a 30-mL suspension for injection. At the 7-year follow-up

(average), 20 of the 34 stage IV hips required total hip replacement; 14 of the lesser stage hips required replacement; most of these replacements occurred in the first 3 years after the initial procedure. They developed a procedure to count the stem cells injected, and there was a significant difference in the number of colony-forming units obtained from the iliac crest according to the etiologic factors of the osteonecrosis. Hips that received a low number of transplanted stem cells had a more significant risk of failure at the latest follow-up than hips that received a high number of transplanted cells.³²¹ Hendrich et al. also looked at a series of 101 patients of which 37 had osteonecrosis of the head of the femur, 32 avascular necrosis/bone marrow edema of other bones, 12 nonunions, and 20 other defects. In the nonunion, healing of the fracture could be achieved with injection of transplanted cells.³²²

In a review of studies on the role of stem cells in fracture healing and nonunion, Fayaz and associates put together a series of 208 nonunion cases of which 195 cases were tibial nonunions. They were treated with concentrated bone marrow aspirates prepared by a variety of different methodologies. Eighty-eight of 100 cases discussed had reported healing³²³ (Table 4.12). Steinert et al. reviewed 1279 patients from 31 different studies with nonunion, avascular necrosis, distraction osteogenesis of long bones, bone cysts, cartilage, and tendon lesions. They concluded that “delivery of MSCs for the repair of bone, cartilage, and tendon cells has shown safety and efficacy in several phase I clinical trials but large comparative prospective randomized clinical trials are required for adequate comparison of the MSC-based therapies to standard treatment modalities,” and “the ideal combinations of cell preparation, bioactive factor(s), and material(s) for each application have to be identified that also

TABLE 4.12 Clinical Evidence for Use of Mesenchymal Stem Cells (MSC) in Nonunion

Study and Year Bone Marrow Aspirate	Area of Treatment	Level of Evidence	No. of Patients	Mode of Administration and Carrier	Healing Times	Outcomes
Connolly and Schindell 1986 ⁷	Tibial nonunion	III	100	100–150 mL marrow osteoprogenitor cells	6 to 10 months	Better outcome compared with standard open iliac crest grafting.
Healey et al. 1990 ²¹	Patients with primary sarcomas that developed delayed unions or nonunions	III	8	50 mL marrow osteoprogenitor cell	4 to 36 weeks.	Good clinical outcomes achieved under difficult clinical circumstances
Giarg et al. 1993 ¹⁴	15 tibia, 3 humerus, and 2 ulna nonunions	III	20 cases	Percutaneous autogenous bone marrow grafting (15–20 mL of bone marrow)	5 months	In 17/20 cases, nonunion healed
Goel et al. 2005 ¹⁸	Tibial nonunion	III	20 cases	Percutaneous autogenous bone marrow grafting (15 mL of bone marrow)	14 weeks	In 15 cases, clinical and radiological bone union was achieved; 4 cases showed no sign of union
Concentrated bone marrow aspirate Hernigou et al. 2005 ²²	Tibial nonunion	III	60 cases	An average total of 51×10^3 fibroblast colony-forming units was inoculated into each nonunion site	12 weeks	Bony union was achieved in 53/60 patients.

From Fayaz HC, Giannoudis PV, Vrahas MS, et al. The role of stem cells in fracture healing and nonunion. *International Orthopaedics* (SICOT) 2011;35:1587-1597, Table 2.

meet the desired safety and cost requirements in a satisfactory manner.”³²⁴ Gomez-Barrena et al. also reviewed the role of cell therapy in bone fracture healing of delayed union and non-union. They concluded, “A major criticism on the available trials are the underreported results, which may reflect lack of protocol adherence, patient heterogeneity in small unicentric trials, confounding efficacy results in part due to patient or to protocol variability, or other issues. Many of these trials do not offer sufficient information about the cell product to correlate with the results in other trials and many are also impossible to reproduce in other centers due to lack of transparency. However, reliability is particularly challenged by the size and design of the currently available trials. Unless large, comparative trials, with well-defined cell products are published, evidence on this therapy will remain controversial or even negative.”³²⁵ Abou-El-Enein and others concluded in their paper that “cell therapies, especially autologous therapies, pose significant challenges to researchers who wish to move from small, probably academic methods of manufacture to full commercial scale. There is a dearth of reliable information about the costs of operation, and this makes it difficult to predict with confidence the investment needed to translate the innovations to the clinic, other than as small-scale, clinician-led prescriptions.... This evaluation illustrated the need for cooperative and collective action by the research community in pre-competitive research to generate the operational models that are much needed to increase confidence in process development for these advanced products.”³²⁶ Thus, although frequently utilized as a procedure of last resort, the role of stem cell transplantation in the healing of stress fractures, especially those with delayed union or nonunion, is unclear.

Romosozumab

(The following is a brief review, as the drug is undergoing evaluation by the FDA for registration at this time.)

Romosozumab is an antisclerostin monoclonal antibody. Sclerostin is a product of the osteocyte that inhibits osteoblast activity. The origin of the agent resulted initially from the recognition of van Buchem disease in South Africa and sclerosteosis, both of which affect the skeleton in that progressive bone overgrowth leads to gigantism, cranial nerve entrapment, and raised intracranial pressure, but these bone overgrowths also seem resistant to fracture.^{327,328} The genetic defect that produces these syndromes is identical and has been discovered as a deletion mutation downstream of the SOST gene.³²⁹ The mechanism of action for romosozumab is to bind to sclerostin, which results in an increase in bone formation. Li and colleagues showed that sclerostin antibody treatment increases bone formation, bone mass, and bone strength in a rat model.³³⁰ There have been a Phase 2 clinical trial and two Phase 3 clinical trials reported.^{331–333} All the studies showed a significant reduction in vertebral and nonvertebral fractures and increase in BMD when compared with alendronate or teriparatide. Of interest, there has been preclinical work in rats and nonhuman primates. In a study by Ominsky et al., the sclerostin-antibody-treated animals showed increased bone formation without increases in bone resorption and showed enhanced fracture healing.³³⁴ Liu and colleagues reported on another closed fracture model

in ovariectomized rats that also showed enhanced bone mass, bone strength, bone formation at the fracture site, and fracture healing. These studies suggested that sclerostin-antibody might have a role in healing osteoporotic fractures.³³⁵ Its role in healing stress reactions, stress fractures, delayed unions, and non-union is at present unknown but, as in the case of other anabolic agents, might result in off-label use in these settings.

Clinical Application

1. We would like our athletes to have a calcium intake of 1000–1200 mg through diet and/or supplementation; usually, we use calcium citrate (usually CitraCal w/D) as opposed to calcium carbonate, usually one tablet per day.
2. We would like our athletes to have a vitamin D 3 intake of 2000–4000 IU per day or vitamin D3 50,000 IU (Replesta), one tablet per week.
3. We would obtain a 25 vitamin D level by LC-MS/MS (e.g., QUEST Diagnostics, #92888) at baseline; probably, >30 ng/mL for prevention; probably, 40–60 ng/mL for fracture healing; probably, about 50 ng/mL for optimal athletic performance (based on studies that we have at this time).
4. Teriparatide (Forteo/Forsteo), rh PTH (1-34), a medication that stimulates bone growth, has been used off label to heal stress reactions, stress fractures, acute fractures, delayed unions, and nonunion.
5. Other anabolic drugs, such as abaloparatide (Tymlos) and romosozumab (if approved), need adequate further evaluation beyond preclinical studies to find their respective roles in fracture healing.
6. It is well established that bone growth stimulators, especially the Bioventus Exogen bone growth stimulator, using low-intensity pulsed ultrasound (LIPUS), are useful in helping acute fractures, stress fractures, delayed unions, and non-unions to heal.

DIET AND NUTRITION

It goes without saying that diet and nutrition are important for bone health. Over the last 20–30 years, there has been increased attention to diet and nutrition at all levels of athletics, including individuals or teams. The issue is: Do we have general principles that we can bring to patients, and specifically to athletes, especially those deemed to be at high risk for stress fractures? There is some controversy as to whether individual components of a diet or the dietary pattern are more important in the epidemiology of general health and bone health.³³⁶

To review healthy dietary patterns and their effect on fractures in postmenopausal women, the Women's Health Initiative (WHI) conducted an observational study (WHI-OS) that examined the indicators and natural history of important causes of morbidity and mortality in postmenopausal women. In its entirety, the WHI, initiated by the National Institutes of Health in 1991, consisted of three clinical trials and the WHI-OS; it enrolled over 160,000 postmenopausal women aged 50–79 years (at the time of enrollment). Women were recruited from October 1, 1993 to December 31, 1998, and the final study population included 90,014 women who were monitored through

August 29, 2014, with a median follow-up time of 15.9 years. Nutrient and food intake was derived from self-report through a WHI food frequency questionnaire (WHI-FFQ), and dietary quality was assessed using the alternate Mediterranean diet (aMED) score, the healthy eating index 2010, (HEI-2010), the alternate healthy eating index 2010 (AHEI-2010), or the dietary approaches to stop hypertension (DASH) score. These indices were used to assess the extent of adherence to the dietary patterns. Incident hip fractures and total fractures (except toe, finger, sternum, and clavicle fractures, which were all excluded) were assessed. During a median follow-up period of 15.9 years, 2121 hip fractures and 28,718 cases of self-reported total fractures were counted. The data supported an association between the extent of adherence to a healthy diet (and also a high-quality diet), Mediterranean diet, and lower hip fracture risk. Confounding issues of falls and low muscle mass did not change the analysis.³³⁷

There are studies of individual dietary components that might be of interest to the high-fracture-risk patient. Byberg et al. studied the intake of fruit and vegetables and their effect on the risk of hip fracture in a cohort of Swedish men and women. Men and women with a zero intake of fruit and vegetables had an 88% higher risk of hip fractures than those who took five portions per day, and taking more (eight) portions per day did not reduce the hip fracture rate further.³³⁸ Onion powder has been reported to reduce bone resorption in rats.³³⁹ Dried plums have also been shown to reduce bone resorption in osteopenic postmenopausal women.^{340,341}

The role of diet and nutrition in the prevention, causation, and healing of stress fractures is an issue that has been percolating for about 20 years. These studies have been undertaken in both soldiers and nonmilitary athletes. Much of this information concerns amenorrheic female runners or athletes with the female athlete triad (see also [Chapter 28](#) on the Female Athlete). In 2014, Mountjoy and co authors published the IOC (International Olympic Committee) consensus statement, *Beyond the Female Athlete Triad on Relative Energy Deficiency in Sport* (RED-S), to delineate the adverse effects of RED-S on the health of athletes. The syndrome of RED-S refers to impaired physiological function including, but not limited to, metabolic rate, menstrual function, bone health, immunity, protein synthesis, and cardiovascular health caused by relative energy deficiency.³⁴² Energy availability is calculated as dietary energy intake minus the energy cost of exercise relative to fat-free mass (FFM), and in healthy adults a value of 45 kcal/kg FFM/day equals energy balance.¹¹⁶ To quantitate this problem in amenorrheic athletes, energy deficits have ranged from -148 to -652 kcal/day, and amenorrheic athletes have lower energy intakes (200–900 kcal/day) than their eumenorrheic colleagues despite similar body weight, body composition, and training status. Kopp-Woodroffe et al. organized a study of four active amenorrheic females (age 18–35) who participated in a 20-week intensive comprehensive evaluation and intervention program. Exercise was reduced by adding a rest day each week. Each study case is reported in extensive detail. There was improvement in the participants' dietary intakes and energy balance, and in their intake of macronutrients and micronutrients.³⁴³

The first step in the therapy of female eating disorders is to restore normal menstrual cycling and increase BMD by modifying their diet and exercise behavior to increase energy availability by increasing energy intake, reducing energy expenditure, or both. Menstrual cycles may be restored by increasing energy availability to more than 30 kcal/kg FFM/day, but increases in BMD may require more than 45 kcal/kg FFM/day.¹¹⁶

Rosenbloom reviewed the role of nutrition in the prevention and treatment of stress fractures, making the point that the dietary guidelines for Americans identify four nutrients “of concern” for Americans, and three of the four—calcium, vitamin D, and potassium—are elements related to bone health. She looked at specific nutrients and emphasized the need for increased intake of protein because, beyond muscle building, it is an important component of bone. She states that protein foods popular with athletes include chicken, turkey, egg whites, cheese, nuts, dairy foods, and soy.³⁴⁴ She suggested the following strategies that she uses as a consulting sports dietitian:

- “Deliver nutrition seminars to teams on the importance of bone health, targeting incoming freshmen, female athletes, and those involved in sports with high risk for stress fractures (cross-country, track and field, basketball).
- Encourage athletes to keep 3-day food [diaries] at the beginning of each season.... Nutrition education materials, forms and videos are available....
- When analyzing food records and counseling athletes, show them where they could add an additional food or snack to increase any shortfall nutrients.
- Use social media (Twitter or Facebook) to educate athletes with quick nutrition tips or easy-to-fix recipes.
- Review all vitamin-mineral supplements taken by athletes: encourage athletes to bring the bottle of supplements they use as a teaching tool and to review for any possible banned substances (although banned substances are unlikely to be found in vitamin and mineral supplements, supplements that claim to be ‘fat-burning’ vitamins or ‘testosterone boosting’ could contain banned substances).
- Spend time with athletes in the dining hall to educate at the point of selection or to steer them to more nutrient-rich choices³⁴⁴ ([Tables 4.13 and 4.14](#)).

Nieves et al. recruited 125 runners from intercollegiate cross-country teams (n = 55), postcollegiate running clubs (n = 70), and road race participants who ran at least 40 miles per week during peak training times and had to compete in races. Eligible women were randomly assigned to receive an oral contraceptive or no intervention for 2 years. Extensive questionnaires were completed, including a modified version of the 97-item National Cancer Institute health habits, and a history food frequency questionnaire was used to estimate nutrient intake during the previous 6 months. A customized automated computer analysis program was used to calculate nutrient intake from the questionnaire. They estimated the relationship between the specific dietary factors and annual rates of change of BMD and BMC. The 125 runners were followed for stress fracture occurrence for a total of 2792 months (avg. 1.86 years per woman). Seventeen of the 125 participants had at least one stress fracture; 9 occurred in the tibia, 6 in the foot, and 2 in the femur; 4 had a second stress

TABLE 4.13 Nutrients Associated With Bone Health, Food Sources, and Recommended Intakes for Male and Female Athletes

Nutrient	Food Sources	Recommended Intakes ^a (per Day)
Protein	Eggs Lean meat Poultry Nuts Low-fat milk (dairy or fortified soy or rice milk) Low-fat yogurt Low-fat cheese	1.2–1.7 g/kg/body weight
Calcium	Low-fat milk (dairy or fortified soy, rice, or almond milk) Calcium-fortified orange juice Hard cheese such as Parmesan cheese Cabbage Broccoli Canned salmon Calcium-set Tofu	1000–1300 mg
Vitamin D	Wild salmon Egg yolks Fortified milk, margarine, and cereal	600 IU
Potassium	Bananas Low-fat milk White and lima beans Spinach Lentils	4700 mg
Vitamin K	Cooked greens (kale, spinach, collards) Broccoli Asparagus	60–75 µg
Magnesium	Whole grains Almonds Cashews Spinach Raisin bran Legumes (dried beans and peas)	310–410 mg
Boron	Fruit-based beverages Avocado Legumes Peanut Butter Peanuts	No recommendation for intake
Silicon	Grains Vegetables	No recommendation for intake

^aRecommended intakes are from the Institute of Medicine, Food, and Nutrition Board,²² Institute of Medicine, Food and Nutrition Board,²⁹ National Academy of Sciences,³² and Burd and Philips.³³

From Rosenbloom, C. Stress fractures in athletes: what is the role of nutrition in prevention and treatment? *Nutrition Today*; March/April 2013;48(2):81-87. Table 2.

fracture: 2 in the tibia, 1 in the foot, and 1 in the femur. The second stress fractures were not considered in the analysis. Higher intakes of calcium, skim milk, milk, and servings of dairy products per

day were each related to a reduced rate of stress fracture, with the strongest protection coming from higher skim milk consumption. Every additional cup of skim milk consumed per day was associated with a 62% reduced fracture risk ($p < 0.05$); every additional serving of dairy products consumed per day conferred a 40% reduction in risk. The authors divined four patterns of nutrients: pattern 1: higher consumption of dairy, lower consumption of fat; pattern 2: higher fruits and vegetable consumption, higher fiber and lower fat consumption; pattern 3: higher animal protein, higher fat, lower fruits and vegetable, lower fiber consumption; and pattern 4: higher protein (both animal and vegetable). They found that runners with a high-dairy and low-fat intake (pattern 1) had a significantly reduced risk of a stress fracture ($p < 0.05$). Calcium intake, skim milk, total milk intake, and number of dairy servings per day predicted significant gains in hip BMD and whole-body BMC; vitamin D intake predicted gains in spine and hip BMD; and animal protein predicted gains in whole-body BMD and BMC. There was a positive relationship between increased potassium and significant increases in BMD of the hip and whole body and in whole-body BMC. The authors felt that increasing dairy consumption might constitute a simple intervention that women runners could implement to reduce their risk of stress fracture.³⁴⁵ Examples of forms to assist in taking a nutritional history in an athlete are presented in [Figs. 4.10 and 4.11](#).

REST AND PHYSICAL THERAPY

A discussion of the role of physical therapy and rehabilitation is beyond the scope of this chapter. For details, see Rosenthal and McMillan.³⁴⁶ See also [Chapter 30](#) and [31](#).

RETURN TO PLAY (SEE ONLINE CHAPTER TO VIEW THIS ASSESSMENT AND RECOMMENDATIONS)

The decision to return to play is a complex one and has to be individualized to the underlying lesion, e.g., high-risk versus low-risk stress fractures, the progression to recovery, and the state of recovery. Every clinician dealing with sports medicine has had to make or will have to make this decision. In the absence of clear-cut evidence-based scientific data or protocols, the return-to-play decisions lack standardization and can be a source of confusion for clinicians, athletes, coaches, and administrators.³⁴⁷ Therefore, we have to gather what information we can from studies that try to review the stress fracture data.

Return-to-play considerations are more difficult for athletes with high-risk stress fractures versus those with low-risk fractures (see [Tables 4.1 and 4.2](#)). The discussion of the rehabilitation of the athlete with a stress fracture is beyond the scope of this chapter, but for those interested in developing their own rehabilitation program, Rosenthal and McMillan supply an excellent discussion.³⁴⁶

In 2002, the American College of Sports Medicine issued a consensus statement on the team physician and return-to-play issues. The statement reviewed establishing a return-to-play process including addressing the safety of the athlete, potential risk to the safety of other participants, functional capabilities of

TABLE 4.14 Bone-Building Snacks for Athletes

Snack	Bone-Building Nutrients in Snack
Raisin bran with fat-free milk and banana	Calcium, vitamin D, magnesium, potassium, protein, silicon
Almonds and strawberries in vitamin D fortified low-fat yogurt	Calcium, vitamin D, magnesium, potassium, protein
Lentil soup with whole grain crackers	Protein, potassium, magnesium, silicon
White bean chicken chili with whole-grain bread	Protein, potassium, magnesium
Peanut butter sandwich on whole grain sandwich thin with cranberry juice	Protein, magnesium, boron
Sports drink with string cheese and whole grain crackers	Protein, calcium, potassium, magnesium
Broccoli and carrots with Greek yogurt-based dip	Calcium, protein, potassium, silicon
6-in. sub on whole grain roll with sliced turkey topped with spinach and tomato with low-fat milk	Calcium, protein, vitamin D, potassium, magnesium, vitamin K, silicon
Low-fat chocolate milk	Calcium, protein, vitamin D, potassium
Scrambled egg with spinach and cheese in a whole wheat tortilla	Protein, vitamin D, potassium, vitamin K, magnesium, silicon
Yogurt parfait with mixed fruit and granola	Calcium, protein, vitamin D, potassium
Bean burrito with avocado slices	Potassium, magnesium
Calcium-fortified orange juice and wheat bran cereal with fat-free milk	Calcium, protein, potassium, vitamin D, magnesium, silicon
Trail mix with almonds, cashews, and dried fruit & grape juice	Protein, magnesium, calcium, boron
Air popped popcorn with melted margarine and Parmesan cheese	Calcium, vitamin D

From Rosenbloom, C. Stress fractures in athletes: what is the role of nutrition in prevention and treatment? *Nutrition Today*: March/April 2013;48(2):81-87. Table 3.

the athlete, functional requirements of the athlete's sport, and federal, state, local, school, and governing body regulations related to returning an injured or ill athlete to practice or competition. They also discuss evaluating, treating, and rehabilitating injured or ill athletes, and returning an injured or ill athlete to play. The later phase should confirm the following criteria: the status of the anatomical and functional healing; the status of recovery from acute illness and associated sequelae; the status of chronic injury or illness; the athlete must pose no undue risk to the safety of other participants; restoration of sport-specific skills; psychosocial readiness; ability to perform safely with equipment modification, bracing, and orthoses; and compliance with applicable federal, state, local, school, and governing body regulations.³⁴⁸

Beyond the general consensus statement, one must deal with the issues of return to play specifically revolving around the issue of stress fractures. Diehl and colleagues, in their series of articles developing the classification system, also dealt with issues of return to play.²⁶ They divided treatment into that of low-risk stress fractures and high-risk stress fractures (see Tables 4.1 and 4.2). They felt that most low-risk fractures could be successfully treated with rest for 2–6 weeks followed by a gradual increase in activity level of limited weight bearing, progressing to full weight bearing performing low-impact activities such as biking, swimming, or pool walking or running, and use of an Alter-G machine. After the patient can perform low-impact exercises without pain for prolonged periods of time, high-impact activities can be started; usually a program of increasing jogging followed by sport-specific activities. In the high-risk stress fracture patient, surgical intervention may be advisable, or even necessary, including open reduction and internal fixation with or without bone grafting, PRP, stem cell injection, or anabolic drugs.²⁴

Creighton and co workers created a three-step decision-based model to help a physician resolve issues in return to play. In the risk evaluation process, step 1 is evaluation of the health status: this considers patient demographics, symptoms, personal medical history, signs (physical examination), laboratory tests (including imaging and, in our evaluation, blood and urine tests, especially for bone turnover markers), functional tests, psychological state, and potential seriousness. Step 2 is evaluation of participation risk, which involves type of sport, position played, limb dominance, competitive level, and ability to protect. Step 3 is decision modification, which involves timing and season, pressure from athlete, external pressure, masking the injury, and fear of litigation. The authors felt that the decision-based return-to-play model would provide a basis for research into the individual factors and components that, when integrated, provide clinicians with an evidence-based rationale for return-to-play decision making.³⁴⁷ The ethical issues involved in these return-to-play decisions have been addressed and are of tremendous importance.^{349,350}

In 2015, the Swiss Sport Physiotherapy Association along with the International Federation of Sports Physical Therapy and the British Journal of Sports Medicine organized a world congress to issue an updated statement on return-to-sport (which they considered a more generic term that was intended to apply to all sports and all athletes rather than to only team sports and athletes). Although previous statements had focused on the team physician as the central figure in the decision-making process, this document was more athlete-centered and placed the athlete in the position of an active decision maker.³⁵¹

Utilizing the Kaeding-Miller stress fracture classification system (discussed in detail in the Classification section²⁹), Miller and colleagues looked at the expected time to return to athletic participation after stress fracture in Division I collegiate



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Nutrition Initial Note

Patient's Name:
 DOB:
 DOS:

S:

History of Present Illness:

Weight History:

Max Weight: Date:
 Min Weight: Date:
 CBW: Height:
 UBW:
 Goal Weight:

Social:

ED Behaviors: Denies bingeing, purging, laxatives, diuretics, diet pills, chewing/spitting

DIETARY:

24 hour recall:

7 Day Food Record Notes:

Food Groups	Description of foods consumed in each food category
DAIRY	
PROTEIN	
CARB	
FRUIT	
VEGETABLE	
FAT	
SNACKS	

Fluids: Water: Soda: Shakes:
 Juices: Coffee/Tea: Smoothies:

Alcohol:

Drugs:

Food Allergies:

LMP: **PMP:** **Menarche:** **Diagnosis:**

Body Image:

Preoccupied with food, weight, shape:
 Percentage of time thinking about it:
 Fear of gaining weight:

Fig. 4.10 Example of forms to assist in taking a nutritional history in an athlete. (Courtesy of Wendy Sterling, MS, RD, CSSD) Courtesy of Wendy Sterling, MS, RD, CSSD, CEDRD-S.

athletes. They evaluated 57 stress fractures in 38 athletes over a 3-year period with mean age 20.48 years (range 18–23 years) with 10 athletes who sustained recurrent or multiple stress fractures. The mean time to return to unrestricted sport participation was 12.9 ± 5.2 weeks (range 6–27 weeks). A trend toward increased time to return to sport was noted in women (13.9 ± 5.7 weeks) compared with men (11.3 ± 3.8 weeks). They felt that

the classification system was a reliable prognostic tool for communicating injury severity between clinicians.³⁵²

Nattiv and colleagues studied the correlation of MRI grading of bone stress injuries with clinical risk factors and return to play in a 5-year prospective study of collegiate track and field athletes. Two hundred eleven male and female Division I cross-country and track and field participants enrolled in the study at



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Nutrition Initial Note

Patient's Name:

DOB:

DOS:

Distorted body weight:

Exercise Currently:

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

E

Exercise History:

Cramper? **Yes** **No**

Supplements:

Sleep:

Mood:

Self-harm:

Family History:

Mom:

Dad:

Past Medical History:

Past Surgeries:

Bowel Function:

GI Symptoms:

Medications:

Calcium:

Multivitamin:

Other:

TEAM:

Therapist:

MD:

Coach/Trainer:

Anthropometrics:

WEIGHT GOAL:

Height: % Height

Weight: % Weight

BMI: % BMI

MBW (@ 50th percentile for BMI): % MBW:

ASSESSMENT:

PLAN:

1.

x Wendy Meyer Sterling, MS, RD, CSSD

#859803

Fig. 4.11 Example of forms to assist in taking a nutritional history in an athlete. (Courtesy of Wendy Sterling, MS, RD, CSSD) Courtesy of Wendy Sterling, MS, RD, CSSD, CEDRD-S.

the time of the preparticipation at the start of their seasons and were followed prospectively to the end of the study at 5 years for bone stress injuries. When a bone stress injury developed, the athlete was seen first by an athletic trainer with regard to their injury and then a team physician. If a bone stress injury was suspected, a radiograph was ordered; if negative, an MRI was obtained; sometimes an MRI was the initial study. They utilized

a modified MRI grading system described by Fredericson¹⁸ and Arendt.²⁵ Thirty-four (12 males, 22 females) sustained 61 bone stress injuries during the 5-year study period. The most frequent sites of injury were the tibia (51%) and metatarsals (21%). Weeks to full return to sport for grade 4 bone stress injuries (31.7 ± 3.7 weeks) were significantly higher compared with grade 3 (18.8 ± 2.9 weeks, $p = 0.055$), grade 2 (13.5 ± 2.1 weeks,

$p = 0.001$), and grade 1 (11.4 ± 4.5 weeks, $p = 0.008$) injuries. Weeks for return to sport for grades 3 and 4 bone stress injuries were significantly higher than grades 1 and 2 injuries (23.6 ± 2.4 weeks versus 13.1 ± 2.0 weeks, respectively, $p = 0.002$). Among grade 3 and 4 injuries, time to return to play was significantly longer for those at bone sites high in trabecular bone versus sites high in cortical bone (38.1 ± 6.4 versus 18.8 ± 2.1 weeks, $p = 0.005$). In those with grade 1 and 2 injuries, there was no difference in time to return to play between injuries at bone sites high in trabecular bone versus cortical bone (17.1 ± 9.1 versus 12.7 ± 1.6 , $p = 0.75$). In addition to MRI grade, total body BMD and bone stress injuries of trabecular bone regions of interest in the femoral neck, pubic bone, and sacrum were the most important independent predictors of return to play. While many studies have found that a lower BMD is predictive of increased risk for stress fracture, this study found that a lower BMD is predictive of return to play (longer recovery time). Clinically, the authors concluded that trabecular bone stress injuries were associated with a significantly longer time to return to play than cortical bone stress injuries, which should help physicians and training staff to utilize a more gradual progression of return to play with activities like Alter G machines or endless pools.

PREVENTION

In 1987, Giladi, Milgrom, and their cohorts, from their studies on the IDF, showed a statistically significant difference in the mean mediolateral width of the tibia at different measurement levels in recruits with and without stress fractures. Standard radiographs were taken using a magnification ruler of total tibial width and cortical width in both anteroposterior and mediolateral planes at three levels in each bone: at 8 cm above the ankle joint, at the point of the narrowest mediolateral width, and at the point of the narrowest anteroposterior width. During the basic training, 91 of the 295 recruits (31%) were found to have one or more stress fractures. Of the total of 184 stress fractures, 51% were in the tibial diaphysis, 5% in the tibial plateau, 21% in the femoral diaphysis, 9% in the supracondylar region, and 4% in the femoral condyles. All of the tibial and femoral diaphyseal fractures were in the medial cortex. For example, at the narrowest mediolateral measurement, those with stress fractures measured 23.8 ± 2.1 mm ($n = 86$) and those without stress fractures measured 24.6 ± 1.8 mm ($p = 0.001$). It was felt that wider tibias should have greater resistance to bending, and resistance to compression and tension would also be greater, since a larger cross-sectional area results in better distribution of the strain forces created by activity over a greater area. The authors stated that the decreased tibial width was “the first physical parameter of the bone to be identified as a risk factor” for stress fractures.⁴³ Thus, the concept arose of preventing stress fractures by building wider bones³⁹ and, therefore, stronger bones.

The role of prevention of stress fractures and the mechanisms underlying the attempt to do so has been reviewed in a variety of preclinical trials. Bone desensitizes rapidly to a loading (dynamic strain stimulus) regimen where bone cells accommodate to routine loading and the exerciser develops a situation that Turner calls “diminishing returns.”³⁵³

Umemura and colleagues looked at the effect of different jumping regimens in 5-week-old rats who were divided into a control group and five jump-trained groups who performed 5, 10, 20, 40, and 100 jumps per day. They jumped 5 days per week for 8 weeks. Each jump took about 3 seconds. After the 8-week trial, the rats were sacrificed and the right femur and tibia were dissected and subjected to a number of tests, including bone mass and bone morphometry. Measuring the fat-free femur and tibia weights, it became apparent that there was a significant difference between controls and the 5-jump threshold. There was not much difference between the 5-jump group and the other jump groups, although the 100-jump group weight was slightly greater (21.9% increase). The cortical area and periosteal perimeter were markedly increased by the jump training. For these and other reasons, jump training (which took a few seconds to 5 minutes in the 100-jump group) is more effective in producing bone hypertrophy than running (which in these experiments usually takes 30–60 minutes).³⁵⁴

Robling, Burr, and Turner, in a further attempt to identify the exact mechanical signal to which bone adapts, studied the effect of partitioning a daily mechanical stimulus into discrete loading bouts and showed this improved the osteogenic response to loading. As stated previously, the osteogenic response to dynamic loading desensitizes after relatively few cycles.^{355,356} These authors studied whether the bone formation response to mechanical loading could be increased by applying multiple bouts of four-point bending within a 24-hour period as opposed to one continuous loading event. Sixty-three adult female Sprague-Dawley rats were randomly divided into 7 groups ($n = 9$) including four bending groups, two sham bending groups, and one nonloaded control group. A standardized limb-bending device was employed. Load cycles (360 per day) were applied to the rat right tibia on days 1, 3, and 5 of the experiment. The groups differed from each other only in the distribution of the delivered cycles over the course of the day. On each loading day, one group of rats received 60 cycles of bending in each of six discrete loading bouts (60×6), each bout separated by 2 hours; another group received 90 bending cycles in four discrete loading bouts (90×4), each bout separated by 3 hours; the next group was given 180 cycles of bending twice a day (180×2), with the bouts separated by 6 hours; and the last bending group received all 360 bends in one bout (360×1). The left tibia served as a control, and there were also sham bending groups. All bending groups exhibited significantly greater mineralizing surface, mineral apposition rate, and bone formation rate in the loaded tibias than in the unloaded tibias. By applying the 360 bending cycles in four discrete bouts (90×4) rather than one bout (360×1), the following increases occurred: 71% increase in relative mineral apposition rate, 80% increase in relative bone formation, and 94% increase in mineralizing surface. The 180×2 and the 60×6 groups had lesser changes. Thus, the results show that 360 cycles per day cause a much greater bone formation response if those cycles are divided into different bouts with an interruption. By adding a rest period between bouts, the bone cells appear to lose some of their desensitization and regain some of their mechanosensitivity. For the same number of cycles, providing more “rest” periods result in more bone formation due to an increase in mineralizing

surface and mineral apposition rate. In conclusion, the authors remark, “These data support the concept of a saturation curve for bone cell mechanosensitivity and suggest that physical activity programs aimed at maintaining or improving bone mass can be optimized by scheduling mechanical loading bouts (exercise sessions) so that most of the load cycles occur during a time when the bone cell network is highly mechanosensitive. This probably would entail several shorter intervals of daily exercise, rather than a single sustained session.”³⁵⁷

The same authors extended their work on desensitization (or adaption), recovery periods, and the restoration of mechanosensitivity to dynamically loaded bone. As they showed previously, recovery periods, as marked by intervals between bending cycles, are necessary to restore mechanosensitivity to mechanodesensitized cells, but they questioned how much time cells require in vivo to become fully resensitized after a loading bout. They took another group of rats and exposed them to the same 360 loading cycles with the same loading device. Then, they introduced recovery periods of 0.5 seconds, 3.5 seconds, 7 seconds, or 14 seconds. The 0.5 second group was loaded for 18 seconds; the 3.5 second group was loaded for two minutes; the 7 second group was loaded for 4 minutes; and the 14 second group was loaded for 8 minutes. Each bout consisted of 90 load cycles. Among the bending groups, the four daily loading bouts were separated by 0, 0.5, 1, 2, 4, or 8 hours of recovery. There were both short-term and long-term recovery experiments. Longer recovery periods resulted in higher bone formation rates. In tibias allowed 8 hours of recovery between each of the loading bouts, bone formation rates were 125% greater than the rates found in rats that had received four bouts with no recovery time between bouts (0-hour group) and 102% greater than the rates found in animals who had received four loading bouts with 0.5-hour recovery time between bouts. Calculation of the recovery curve beyond 8 hours revealed almost no further increase in sensitivity with increasing recovery duration. “Thus, a recovery period of approximately 8 h[ours] is sufficient to restore full mechanosensitivity to the cells.” Mineralizing surface and bone formation rate were significantly higher (66%–190%) in the 14 second group than in any of the other three bending groups. The results in the short-term experiment suggested the existence of a short-term recovery threshold somewhere between 7 and 14 seconds. By increasing the duration of recovery between loading bouts, a greater degree of sensitivity is restored to the cells. Approximately 8 hours of recovery is adequate to restore full sensitivity. Although suboptimal, shorter recovery periods (0.5–1 hour) allow more bone formation than no recovery period at all (0-hour group). The 2001 paper stated that the cellular mechanisms involved in mechanosensory loss and recovery and those involved in short-term recovery phenomenon are unclear.³⁵⁸ But in the context of newer information discussed elsewhere in this chapter, it is possible that osteocytes, known mechanosensors and mechanotransducers of the skeleton, play a role. The authors conclude: “Physical activity programs used as prophylaxis for bone loss might be met with greater success if appropriate recovery periods were structured into exercise programs.”³⁵⁸ The same might be said for programs developed to prevent stress fracture occurrence.

Continuing the development of this line of research, Robling and colleagues studied bone structure and strength after long-term loading and found it was improved the most if loading was separated into short bursts. With a similar study protocol, they studied *ex vivo* strain gauging, peripheral QCT, micro-CT, and mechanical testing to the rat ulna diaphysis curvature after their dynamic loading of 360 cycles delivered in one single bout and 90 cycles delivered in four bouts with 3 hours between each bout. Three-dimensional reconstructions of the micro-CT whole ulna slices showed that the right (loaded) limb was significantly different from the left (nonloaded) limb. The loaded bones had significant mediolateral thickening of the diaphysis near the midshaft and the distal shaft. The 90 x 4 group had significantly greater improvement in the measurement of geometric properties, e.g., the maximum and minimum second moments of area (I_{max} and I_{min}), than the 360 x 1 group. Percent difference (right versus left) in ultimate force and energy to failure was also significantly different, by 35% and 75%, respectively, in the 90 x 4 group. So, biomechanical properties were increased by loading. The loaded ulnas exhibited 5.4% and 8.6% greater areal BMD (DXA) in the 360 x 1 and 90 x 4 groups, respectively. These “small” gains in aBMD and BMC resulted in “very large” increases in ultimate force (64%–87%) and energy to failure (64%–165%). The authors summarized their work by stating that “it might be possible to significantly enhance fracture resistance through mechanical loading (e.g., exercise) even if some of the noninvasive measurements of bone mass or density (e.g., DXA) reveal only slight changes. For example, most exercise intervention studies yield differences in aBMD of only a few percent at most, but it is not known how such intervention affects fracture susceptibility. [Their] data suggest that bone strength can be enhanced substantially from small changes in BMD or BMC if bone is added to the mechanically appropriate (high strain) sites.”³⁵⁹

Hughes and her group also dealt with clinical issues for prevention of stress fractures. They reviewed the data on building wider bones. The only modifiable, nonpharmacological way to increase the width of long bones is via physical training. Thus, the hypothesis was proposed that before beginning either basic training in the military or formal training as an athlete, pretraining might be instituted to stimulate adaptive bone formation.³⁹ Two early studies seemed to indicate this was not so: Mustajoki et al. in 1983 felt their prospectively obtained data on 103 men from the Parachute Ranger School of the Finnish Defense Forces suggested that previous physical activity (not specifically stated as to what activity they were referring) did not affect the risk of stress fractures in military recruits.⁴⁶ Swissa and colleagues conducted an early IDF study of 295 new male infantry recruits, where 79 (28%) out of 279 recruits who were interviewed did not participate in any sport activity prior to basic training, 160 recruits (57%) participated in running and jogging, and the remaining 58 recruits (21%) participated in various sports, usually basketball or soccer. In their analysis, no correlation was found between pretraining sport activity and stress fractures.⁴⁷ Newer studies and reviews contradict these findings and support the original hypothesis: Milgrom and Shaffer³⁶⁰ and Milgrom et al.⁴⁵ in further carefully documented

studies of IDF recruits found, as previously discussed, that the incidence of stress fractures was 28.9% among those who did not play ball sports and 13.2% among those who played ball sports for at least 2 years before induction in a first epidemiologic study (1988 induction group); 27% among those who did not play ball sports and 16.7% among those who played ball sports in a second epidemiologic study (1990 induction group); and 18.8% among those who did not play ball sports and 3.6% among those who played ball sports for at least 2 years in a third epidemiologic study (1995 induction group). Playing ball sports for less than 2 years had no protective effect; swimming provided no protective effect; the number of kilometers run per week was also not protective. The major ball sport that was protective was playing basketball three times per week. In the chapter by Milgrom and Shaffer, they state that over the three periods of time involved in these studies, the sport of basketball replaced soccer as the primary ball sport in the country, and this resulted in a significantly lower incidence of stress fractures.³⁶⁰

The only two papers that try to deal with the application of the principles of prevention to reduction in stress fractures both apply to the IDF.^{45,355} I have been unable to find any papers that apply this basic science information to an athlete population, but we can make some inferences to help from the basic science information so far developed. In 2008, Finestone and Milgrom reviewed their 25-year effort to lower the stress fracture incidence in the IDF. The first step toward lowering the incidence of possible stress fractures is to develop as comprehensive a list of risk factors for stress fractures as possible. Narrow tibias were found to be a risk factor for both tibial and femoral stress fractures. The reason the narrower tibia is a risk factor is that increasing the diameter of the bone from 2 to 2.5 cm increases the bending and rotational strength by 126% and the compression strength by 51%. External rotation of the hip greater than 65° was found to be a risk factor. The narrow tibia and hip rotation are independent risk factors, and combining them allowed profiling of those at high risk for stress fractures. As previously discussed, basketball was found to be a deterrent and long-distance running was not. They found a discrepancy between the laboratory bench studies as reported and the *in vivo* human bone strain recordings, so this led them to the hypothesis that tibial and femoral stress fractures are mediated by bone remodeling.⁴⁵ The authors then took a look at the amount of sleep the recruits had and its effect on stress fracture incidence. Among the sleep-deprived recruits, bone turnover markers increased by 170%, and in those who slept in a vertical position they increased by 68%. Because this study was performed in the early 1990s, urinary hydroxyproline was measured as a bone turnover marker.³⁵⁶

The understanding of the importance of sleep and bone metabolism is increasingly recognized.^{176,361,362} Studies suggest that there is a diurnal circadian rhythm for serum C-telopeptide (s-CTX), indicating sleep is essential for normal bone function and that sleep or circadian disturbance could directly affect bone physiology and metabolism.¹⁷⁵ Swanson et al. studied bone turnover markers after sleep restriction and circadian disruption as a mechanism for sleep-related bone loss. They recruited 11 healthy men, aged 20–65 years, to a complex sleep study that included both a “sleep satiation”

component and a “forced desynchronization” component in which the sleep-wake cycle was separated from the internal circadian cycle. This was accomplished by scheduling recurring 28-hour sleep-wake cycles with a 21.47-hour wake episode and a 6.53-hour sleep opportunity (equivalent to 5.6 hours of sleep opportunity per 24 hours) over approximately 3 weeks. Therefore, participants experienced circadian disruption (i.e., a mismatch between the internal clock and the external environment). Serum for this study was obtained hourly from the participants who were admitted to individual suites in the Intensive Physiological Monitoring Unit of the Center for Clinical Investigation at Brigham and Women’s Hospital between 2007 and 2010 over a 24-hour period on days 5 and 6, at the baseline and at the end of the forced desynchrony period, referred to as postintervention. They measured four bone biomarkers, including serum C-telopeptide (s-CTX) as a marker of bone resorption, the N-terminal propeptide of type I procollagen (PINP) as a marker of bone formation, and sclerostin and fibroblast growth factor 23 (FGF 23) as measures of osteocyte function. Without going into further description of the details of the sleep studies, the authors found a lower PINP and no change in s-CTX after 3 weeks of circadian disruption and sleep restriction creating a “bone loss window.” Their study suggests that the changes were greater in the six younger individuals (20–27 years, mean 23.5) than the four older (55–65 years, mean 58.75) individuals. “If sustained, these alterations in bone metabolism induced by sleep/circadian disruption could result in suboptimal peak bone mass, bone loss, osteoporosis, and fracture.”³⁶² The decrease in PINP is “of similar magnitude to the expected increase in PINP seen with responders to teriparatide treatment,”³⁶² indicating that bone formation may be preferentially affected by sleep and circadian disturbances. Perhaps an important reason for microdamage accumulation is that, while bone resorption cannot clear all the microdamage that occurs, the defect may be that bone formation cannot repair the deficit left by the bone resorption. In the IDF studies, not all soldiers responded equally; 40% of the recruits in the sleep-deprivation and vertical-sleep groups had increased bone markers (“responders”) while 60% did not have increased bone turnover measurements (“nonresponders”).¹⁷⁶ While the increased occurrence of stress fractures in basic training is likely due to multiple different factors, “these data may explain part of the pathophysiology underlying these injuries, and the incidence of stress fractures during basic training may be partially mitigated by sleep extension or at least minimizing the interval during which recruits are exposed to sleep/circadian disturbance.”³⁵⁶ Therefore, the IDF decided to strictly enforce a minimum 6-hour sleep requirement during basic training.³⁵⁶ Qvist and colleagues, in their study of the circadian variation in the serum concentration of s-CTX and the effects of gender, age, menopausal status, posture, daylight, serum cortisol, and fasting, found that s-CTX should be obtained in the fasting state.¹⁷⁵

The effect of sleep deprivation on athletic performance has definitely been shown in multiple settings.³⁶³ Mah and coauthors from the Stanford Sleep Disorders Clinic and Research Laboratory studied 11 healthy students from the Stanford University men’s varsity basketball team. The athletes maintained their habitual sleep-wake schedule for a 2- to 4-week baseline period followed by a 5–7-week sleep extension period, with a minimum goal of 10 hours in bed each

night. Total objective nightly sleep time increased during sleep extension compared with baseline by 110.9 ± 79.7 minutes ($p < 0.001$). Subjects demonstrated a faster timed sprint following sleep extension (16.2 ± 0.61 seconds at baseline versus 15.5 ± 0.54 seconds at the end of sleep extension, $p < 0.001$). Shooting accuracy improved, with free throw percentage increased by 9% and 3-point field goal percentage increased by 9.2% ($p < 0.001$). The Psychomotor Vigilance Task and the Epworth Sleepiness Scale both decreased following sleep extension ($p < 0.001$), and the Profile of Mood States improved with increased vigor and decreased fatigue subscales ($p < 0.001$). The players also reported improved overall ratings of physical and mental well-being during practice and games. The authors concluded that “optimal sleep habits and obtaining adequate sleep will play an important role in peak performance in all levels of sports.”³⁶³

There is, as yet, no reference that shows that poor sleep habits in athletes leads to an increased incidence of stress fractures; this is an area that needs research. If we look at the IDF findings, it certainly seems like poor sleep hygiene could potentially contribute to poorer bone health and stress fractures. What it means is that physicians and health care professionals taking care of stress fracture patients need to ask about sleep habits. Sleep health should be incorporated into training programs, based on the individual needs of the athletes, and if there is a recognized or perceived problem, the athlete may need to be referred to a sleep health professional.

In 2017, Milgrom and Finestone summarized all their work, again, on stress fracture interventions aimed at prevention of stress fractures as it applied to a single elite infantry unit.³⁵⁶ Of course, the concept of prevention is based upon not exceeding the loading or repair potential of bone. It is easier to deal with these issues in a military unit, whereas it is impossible to know, at our present state of knowledge, what these thresholds are in an individual athlete, but there may be more variability of body type, body composition, and bone density in a NFL team than in an IDF elite infantry unit.¹²³ Studies were undertaken during the 14-week basic training of the same elite IDF infantry unit in 1983, 1988, 2002, 2006, 2007, 2011, 2012, 2013, and 2015. In all of the studies, the basic training was done in the winter (daily January temperature about 60–70° F). In 1983 and 1988, the training was performed on a topographically very hilly base; but, starting in 2002, the training was moved to a topographically flatter base—a significant first change. The cumulative formal march distance in 1983 and 1988 during the 14 weeks of basic training was 548 km (340.5 miles); beginning in 2002, the march distance was decreased by one-third to 348 km (216.2 miles). So the second change that was made in the IDF training was a decrease in the recruits’ cumulative marching and running.³⁵⁶ These two changes had a statistically significant association with a decreased incidence of stress fractures.³⁵⁶ Another change introduced was the development of an authorized training protocol to be followed by the units. By multivariate analysis, the only stress fracture training intervention that had a statistically significant association with lowering the occurrence of stress fracture during the observation period of 1983 to 2015 was restricting training to the “authorized training protocol.” In 2011, three changes were made to the training regimen. First, a mandatory 7-hours-a-night sleep regimen was enforced; even though this had not been studied

prospectively, it was thought this would help. Second, there was a change in the infantry boot. Third, they added a physical therapist to the unit.

Again, some of these changes would be of value to the elite athlete but others might not be applicable. In addition, in an IDF study, infantry recruits whose march distance was increased more gradually during basic training than a control group sustained the same incidence of stress fractures but over a more extended period of training.³⁵⁶ Although the role of some of the rat studies is unclear, the study that shows an interval in the training regimen for 3 hours probably has some relevance to the human condition. But whether a variation of a high-intensity interval training (HIIT) is better than the usual training programs currently employed for the prevention of stress fractures is unclear.

Clinical Application

1. The preclinical science studies delineate some information about ways to exercise, such as breaking up a session into multiple shorter exercise periods per day with several-hour intervals between sessions (recovery periods).
2. Very modest changes in aBMD or BMC can translate into large changes in mechanical properties because mechanical loading tends to add bone to the most structurally relevant sites. This does not necessarily happen with pharmaceutically induced bone formation. For example, teriparatide (Forteo) adds bone primarily to the endocortical and trabecular surfaces where it contributes little resistance to bending.³⁶²
3. Warden et al. set forth steps for targeting exercise toward the skeleton to increase bone strength. They made some suggestions: Step 1: start young; Step 2: select dynamic, high-impact exercises; Step 3: exercise the bones you want to strengthen; Step 4: exercise briefly but often; and Step 5: continue exercising as you age.³⁶⁴

Burr (personal communication), in response to a question about designing the ideal exercise program to reduce stress fractures, felt that “a small amount of exercise, several times per day (with 4–6 hrs between bouts) is the most beneficial for building bone because bone cells have a refractory period after a fairly small amount of loading. Therefore, to build bone specifically, requires perhaps a few hundred cycles of jogging/running, repeated 3–4 times per day with 4–6 hr intervals between [bouts]”. This would also prevent the muscle fatigue–related negative effects of longer periods of exercise that cause increased strain and strain rate on bone, and can cause bone damage.

CONCLUSION: STRESS FRACTURES

Lessons Learned After 20 Years of Treating Stress Fractures, Delayed Unions, and Nonunions

1. In cases of acute fracture, a modest metabolic/endocrine workup is indicated, including 25 hydroxy-vitamin D, parathyroid hormone, calcium, and phosphorus that should be obtained in addition to appropriate imaging.
2. In cases of delayed union or nonunion, a comprehensive metabolic/endocrine workup is indicated, as proposed, and includes bone turnover markers, at least P1NP and sCTX.

3. Teriparatide should be initiated as 20 µg subcutaneously daily; after 6–12 months, if there has been no improvement in fracture healing, consideration should be given to increasing teriparatide to 40 µg per day.
4. LIPUS should be used twice a day (every 12 hours) until fracture is healed.
5. Treatment of an acute fracture or delayed union or non-union fractures should combine teriparatide and LIPUS bone growth stimulator.
6. Fracture healing is defined as four-point bridging on CT scan.
7. Four to 8 weeks of teriparatide is too short a period of time for treatment: bone turnover markers have not increased in response to teriparatide, because the time interval is too short; this may be different with abaloparatide and/or romosozumab.
8. Treatment of an acute fracture with teriparatide and LIPUS can be stopped when the acute fracture is healed.
9. Teriparatide for one season after a stress fracture is healed; can stop LIPUS when fracture is healed.
10. If a stress fracture is caused by an accumulation of microdamage, then to reduce or heal the accumulation of such damage requires a medication that heals or reduces the amount of microdamage by bone remodeling, such as teriparatide (or perhaps abaloparatide or romosozumab). Whether other newer drugs will help stress fracture healing may depend on the proportion of remodeling to modeling of the particular agent.

Bone stress injuries comprise a significant percentage of sports-related injuries (and military injuries), and as this chapter demonstrates, as our understanding of the pathology (starting in 1855 with the first description by Breithaupt), epidemiology, pathogenesis, diagnostic tools (from the original x-rays by Stechow to MRI, DXA, TBS, and Osteoprobe), surgical repair, calcium, vitamin D, anabolic bone pharmacologic agents, bone growth stimulators, and nutritional influences on fracture causation and prevention and healing evolves, it is likely that the approach to prevention and treatment will continue to improve over the next decade. It is possible that the treatment in the next few years and decades will be markedly different than it is today.

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Problematic Stress Fractures of the Foot and Ankle

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PEARLS

1. Subtle, unexplained pain in the foot or ankle in an athlete can be a stress fracture.
2. Stress fractures of the medial malleolus may be associated with pathologic varus coming from the knee, ankle, or hindfoot. If surgery is warranted, the underlying biomechanics leading to the stress fracture need to be addressed.
3. Navicular stress fractures can occur in both the competitive and recreational athlete.
4. Metatarsal base stress fractures in elite athletes must be treated aggressively because they can place one's career at risk.

INTRODUCTION

A stress fracture is a complete or incomplete fracture of bone secondary to failure over a prolonged period due to repetitive microtrauma and submaximal stresses. While these fractures are relatively rare, they can pose a significant barrier to performance and function in the athlete and non-athlete alike if not properly diagnosed and managed in a timely fashion.¹

Stress fractures differ from acute fractures in that their course generally is more gradual with oftentimes an elusive radiographic appearance. Stress fractures of the foot and ankle are common in running athletes, especially those who jump. For example, track athletes, ballet dancers, and basketball players have a high incidence of stress injury.^{2,3} Many studies have implicated biomechanical factors, such as leg-length discrepancies, cavus foot deformities, and limb malalignment. Women have a higher incidence of stress fractures, and amenorrhea often is a concomitant finding in female

athletes with these injuries.⁴ (See also [Chapter 3](#)—Stress Fractures: Their Causes and Principles of Treatment; [Chapter 4](#)—Metabolic Assessment and Treatment in the Athlete; and [Chapter 28](#)—Unique Considerations for Foot and Ankle Injuries in the Female Athlete.)

The insidious onset of ill-defined foot and ankle pain is the main culprit in the delay of diagnosis. Understanding the biomechanics leading to such injuries, the means of diagnosis, and the execution of treatment should be requisite in the armamentarium of physicians treating athletes.

STRESS FRACTURE OF THE TARSAL NAVICULAR

Tarsal navicular stress fractures account for one-third of all stress fractures.^{2,5} However, navicular stress fractures remain an elusive and poorly understood facet of dorsal midfoot pain in sports. Primarily diagnosed in running athletes,² its incidence has risen from 0.7%–2.4% in the 1980s^{6,7} to more recently^{1,2,5} at 14% to 35% of all foot and ankle stress fractures. It is unclear why such the rapid rise in the incidence; it may be due an increase in better imaging modalities and a higher index of suspicion.⁸ Navicular stress fractures are not to be taken lightly. In an epidemiological NFL Combine study, players with these injuries had a greater probability of not being drafted and not competing in at least two NFL seasons when compared with matched controls.⁹

Anatomy and Presentation

The tarsal navicular serves as a keystone in the medial longitudinal arch and consequently is subjected to tremendous forces

through the foot. Moreover, nutrient arteries arising from both the anterior and posterior tibial arteries create a generous supply of blood to the medial and lateral thirds of the navicular. The result is a poorly vascularized zone in the middle third of the bone as described by Waugh in 1958.¹⁰ Recently, this arterial anatomy has been called into question, as a recent study demonstrated a robust intraosseous vascular supply in 59% of adult cadaveric specimens.¹¹ Factors other than vascularity may predispose the navicular to a stress injury, such as the location of the navicular, sandwiched between the talar head and the cuneiforms. Lateral shear forces are generated during running, as the medial aspect of the navicular has its stress shared by the talar head.¹²

Misdiagnosis of stress fracture of the tarsal navicular generally is the rule rather than the exception, as the average time to diagnosis is 6–8 months.^{13,14} There are several sources of midfoot pain that are more common, including anterior tibial and posterior tibial tendinitis, spring ligament injury, Lisfranc sprain, and degenerative joint disease.¹⁵ Therefore, unrelenting symptoms in the seemingly normal midfoot merit further diagnostic workup.

Towne et al. first reported stress fracture of the navicular in humans in 1970.¹⁶ In this series of two patients, each was a distance runner who had experienced midfoot pain with swelling and failure to respond to conservative therapy. Plain radiographs were negative, and only specialized studies were able to reveal the occult fracture.

Clinically, an athlete complains of a slow but progressive onset of medial dorsal foot pain that radiates along the medial arch. Initially, this is experienced only during sports and is relieved by rest. Certain activities can increase the pain, such as cutting, sprinting, pushing-off, and jumping. After time, the

onset of pain during activity occurs sooner and rest does not offer a respite. Eventually, the dorsomedial pain (over the “N” spot) limits sports and as activities of daily living. (Fig. 5.1)

Imaging

Stress fracture of the tarsal navicular is often overlooked due to radiograph's poor sensitivity (33%).⁵ An anteroposterior view of the foot can show: sclerosis of the proximal border of the navicular; a short first metatarsal; metatarsus adductus and hyperostosis; and stress fracture of the second, third, and fourth metatarsals. Most fractures are linear, lie in the middle third of the navicular, and can be complete or partial.¹⁷ Oblique or supinated radiographs can be useful (Fig. 5.2).

Radionuclide bone scanning has 100% sensitivity and high positive predictive value.^{13,18–20} Uptake generally will appear in the shape of the navicular on the plantar view. Although radionuclide scanning can assist in localizing the area of concern to the navicular, definitive diagnosis and definition of the fracture pattern require magnetic resonance imaging (MRI) or a computed tomography (CT) scan.

The typical fracture is an incomplete fracture in the central one-third with the fracture line extending obliquely from dorsal lateral to plantar medial on CT scan.¹⁷ A useful CT classification,²¹ based on coronal cuts is as follows:

Type I: Dorsal cortex fracture

Type II: Extension into the body

Type III: Extension from dorsal to plantar cortex

While MRI scans have a high sensitivity, CT scans can more accurately diagnose a navicular stress fracture.^{14,18} However, MRI is better able to show edema patterns and a medullary extension of the fracture.



Fig. 5.1 Examination of a foot for the “N” spot.



Fig. 5.2 An oblique radiograph of the tarsal navicular demonstrates a stress fracture.

Treatment

Treatment at our respective institution is based on CT findings, athletic participation level, and the patient's functional status.⁸ Complete elucidation of the fracture pattern is important in dictating management of the athlete. Patients with incomplete and nondisplaced/incomplete fractures (Type I) typically respond well to conservative management. We typically treat patients in a nonweight-bearing cast for at least 6 weeks, followed by a protected weight bearing in a cam boot for 2–4 more weeks until pain is no longer present.

Athletes with Type II or any patient with Type III fractures, patients with displaced fractures, or those who have failed nonoperative management benefit from bone grafting with open reduction and internal fixation (ORIF). We prefer to use either two 4.0-mm cannulated screws or two cannulated headless compression variable pitched screws to provide fixation after gently debriding and reducing the fracture. Most of these athletes will return to sport within 5 to 7 months. High-performance career athletes and the treating surgeon may elect a more aggressive approach to nondisplaced fractures. In a recent study by Saxena et al.¹⁴ patients who underwent ORIF had a return to activity 4.6 months compared with those who had undergone nonoperative treatment, who had an average return to activity of 4.0 months.

STRESS FRACTURE OF THE BASE OF SECOND METATARSAL

A base of the second metatarsal stress fracture is an often misdiagnosed condition that seemingly is exclusive to elite-level ballet dancers. However, fractures of the other metatarsals also are seen in new military recruits and running athletes.²² The unique biomechanics of ballet dancing, coupled with the high incidence of hypoenestrogenism among female performers (see Chapter 28 on unique considerations for foot and ankle injuries in the female athlete), generates an environment conducive to stress fracture of the base of the second metatarsal. High-level ballerinas generally have a narrow window of opportunity and short-lived careers; therefore, rapid diagnosis and treatment are essential. Outcomes from treatment of second metatarsal fractures are excellent, and this injury usually is not considered to be a career-threatening disability.

Anatomy and Presentation

The most common presentation of stress fracture of the second metatarsal is the insidious onset of midfoot pain. However, ballerinas will report intermittent sudden onset of pain after an increase in training or after a jumping maneuver. Many performers will be able to “dance through” the pain and often do not present until 2 to 6 weeks after the onset of symptoms.^{23,24} Hamilton²⁵ reported five risk factors for stress fracture in the ballet dancer. They include amenorrhea, anorexia nervosa, cavus foot, anterior ankle impingement, and a Morton's foot (short first metatarsal).

Examination of the foot can be confusing rather than revealing because patients will exhibit generalized tenderness of the midfoot with palpation and motion. It is oftentimes hard to distinguish pain localized to the base of the second metatarsal versus the Lisfranc joint.²⁶

The nature of this injury is due primarily to the unique biomechanics of ballet and specifically to the incredible stresses placed on the midfoot when the dancer is in the *en pointe* position. When *en pointe*, the ballerina (male dancers dance only on *demi-pointe*) stands on the tips of her toes with the foot in maximal plantar flexion.¹³ Consequently the mechanical axis of the lower extremity is directed straight through the plantarflexed foot. The middle cuneiform serves as a keystone in an arch-type configuration. The base of the second metatarsal is countersunk into this keystone. Furthermore, the plantar ligaments create tensile forces about the second metatarsal at push-off during normal gait. This anchor of the proximal second metatarsal generates a substantial stress riser at the junction of the metaphysis and diaphysis when the dancer is *en pointe*. Treatment can be as simple as restricted dance with a moratorium on *en pointe* maneuvers until union is achieved.

Imaging

The evaluation of the painful foot in a ballerina must include weight-bearing views of the foot and ankle. O'Malley et al.²³ recommended a specialized view called the posteroanterior dancer's view. The dancer's foot is placed with the dorsum on the cassette to eliminate overlap of metatarsals. Approximately 30% of plain films will demonstrate a stress fracture. Bone scintigraphy is positive in 100% of second metatarsal stress fractures; yet Harrington et al.²⁶ reported positive bone scans in two of their patients diagnosed with synovitis of the second tarsometatarsal joint. The role of MRI has not been clearly defined, but it is used routinely in our practices, as it can show edema as early as 2–8 days following the onset of symptoms.²⁴ CT with fine cuts also is an effective method to demonstrate a stress fracture of the base of the second metatarsal. Differentiation can help to direct a less disruptive management routine for professional dancers. For example, nonsteroidal treatment and dance modifications for traumatic synovitis may seem more attractive to a professional dancer than 6 weeks of rest. A large cohort of patients experienced good results following external shockwave therapy.²⁴

Treatment

The timing of this injury, in concert with the goals and aspirations of the dancer, should lead the clinician in treatment. Patients usually can expect a full recovery in approximately 6 to 8 weeks. Initial management should include cessation of all dance activity and application of a hard-soled shoe. Pain at the base of the second metatarsal then serves as a barometer for return to activity. The dancer may begin working out but should delay return to jumping and *en pointe* maneuvers. The rate of recurrence can be as high as 12%. Ballerinas should be reassured that this is rarely, if ever, a career-ending injury.

If a patient fails nonoperative management, we prefer to perform an open reduction internal fixation of the metatarsal shaft. We use a minifragment plate with 2.0-mm screws. After fixation, the patient is placed into protected weight bearing for 6 weeks. The patient can return to dance at 8 weeks.

Case Example

An 18-year-old, college-level, female basketball player presented to the sports medicine clinic with a long-standing history of left

midfoot pain that had gotten acutely worse. The pain was exacerbated by play and persisted the majority of the season. She had a history of a similar injury that was treated successfully in high school.

Examination demonstrated bilateral pes planovalgus deformities with tenderness over the base of the second metatarsal. Pain was reproduced with motion of the second, third, and fourth tarsometatarsal joints. Plain radiographs and a CT scan (Figs. 5.3, 5.4, and 5.5) showed a chronic stress fracture at the base of the second metatarsal. She was given a walker boot for daily activity



Fig. 5.3 An anteroposterior (AP) oblique radiograph shows a chronic stress fracture of the base of the second metatarsal.



Fig. 5.4 An oblique radiograph shows a chronic stress fracture of the base of the second metatarsal.

and a rigid shank for her shoe to wear during play. The boot was worn during off times, and the shank was worn during games. She successfully completed the season without limitations. Follow-up images showed a nonunion of the second metatarsal and a healed third metatarsal fracture. At last follow-up, she continued to play at the collegiate level asymptotically.

STRESS FRACTURE OF THE MEDIAL MALLEOLUS

There is a paucity of literature regarding this unusual injury as this was only first described in 1958. Medial malleolar fractures represent a rare stress fracture injury, as they account for only 0.6% to 4.0% of all lower extremity stress fractures.^{2,27}

Anatomy and Presentation

The majority of patients with stress fracture of the medial malleolus are running and jumping athletes who present with gradual onset of pain over the medial ankle. Pain is exacerbated by activity and is localized to the medial malleolus.²⁸ In 2002, Shabat and colleagues performed a review of the 23 cases reported in literature.²⁹ Males were affected disproportionately higher than females (78%). The average age of the affected patients was 24 (range, 15–60).

Shelbourne et al. identified three criteria for the evaluation of medial malleolar stress fracture.³⁰ They include tenderness over the medial malleolus with an ankle effusion, pain during activity preceding an acute episode, and a vertical line from the tibial plafond proximally.

Physical examination often demonstrates edema of the medial malleolus with bony tenderness. Patients often will have normal motion in the ankle and subtalar joints and should not have tenderness of the lateral ankle or posterior tibial tendon. Analysis of hindfoot alignment is critical to assess any varus deformity that may exacerbate stresses on the medial malleolus.

The ankle is exposed to repetitive dorsiflexion and external rotation of the tibia during running. During heel strike, and as the hindfoot dorsiflexes, the forefoot pronates; the navicular becomes abducted relative to the talar head.³⁰ The talus is thus forced to internally rotate into the medial malleolus causing the

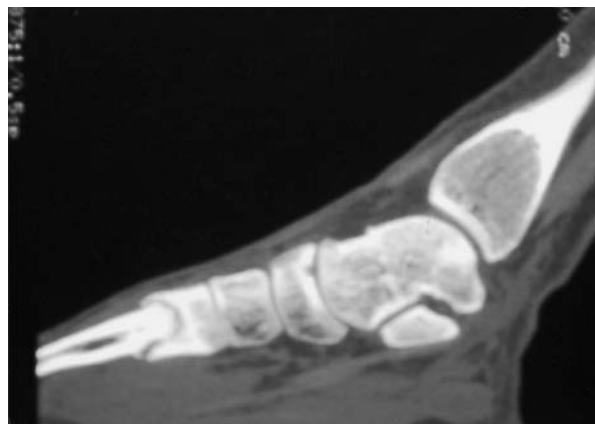


Fig. 5.5 A sagittal computed tomogram (CT) shows a chronic stress fracture of the base of the second metatarsal.

tibia to externally rotate. Consequently, the fracture line usually starts anteriorly at the medial tibial plafond and extends superomedially into the metaphysis. This vertical type of fracture pattern is also seen in Lauge-Hansen adduction injuries.

Theoretically, a varus malalignment at the ankle biomechanically places more sheer and rotational stress along the medial malleolus,^{31,32} though most case reports note that a majority of their patients did not have any mechanical alignment abnormalities.^{28,33}

Shelbourne et al. used strict criteria to diagnose a medial malleolus stress fracture.³⁰ The patient must have: tenderness over the medial malleolus and a joint effusion; pain during activities before an acute medial sided ankle pain; and a vertical fracture propagating from the medial tibial plafond.

Imaging

Plain film radiography is requisite in the diagnosis of medial malleolar stress fractures and can be more useful with other problematic stress fractures of the foot. There may be a small area of fissuring along with cysts at the junction of the tibial plafond and the medial malleolus.¹² When one has normal radiographs, a triple phase bone scan, CT, or MRI can be useful. We routinely use MRI in our diagnosis, as it can be used to accurately identify stress reactions and subtle fracture lines (high-intensity signal on T2-weighted and decreased T1-weighted images at the plafond-medial malleolus junction).³⁴ MRI use was supported in the case series²⁷ in which medial malleolar stress fractures picked up all edema patterns while initial radiographs were negative.

Treatment

Initial management of stress fracture of the medial malleolus should include cessation of sport, with nutritional and endocrine interventions when appropriate. Recreational athletes with small fracture lines can be treated nonoperatively in a short-leg cast or removable boot. Patients treated conservatively should not return to sport until they are asymptomatic, a period of time that averages 6 weeks.

Conversely, many authors prefer operative management of this injury, citing the possibility of nonunion and faster return to sport as incentives.

The objective of operative management is to create a construct that counters the tensile forces of the medial malleolus and allows quick rehabilitation. Standard AO technique should be used with either cancellous or cortical lag screws positioned perpendicular to the fracture line. Some surgeons advocate the use of lag screws through a buttress plate. Patients treated with internal fixation return to sport on average at 4.5 weeks and have evidence of union by 4.2 months.²⁹ The elite or professional athlete may prefer this option because it portends a faster return to activity and theoretically reduces the risk of nonunion or complete fracture.

A malleolar nonunion can lead to significant lost playing time and potentially can be career ending. Isolated medial ankle pain with normal radiographs merits further workup with either bone scintigraphy or MRI, followed by an appropriate scheme of management tailored to the athlete's goals and aspirations.

CASE STUDY 5.1

An elite-level, male, college basketball player began to note pain in the antero-medial distal ankle early in the season. As the season progressed, he had to stop playing because of recalcitrant pain. Physical examination demonstrated tenderness along the anteromedial aspect of the tibia and pain with dorsiflexion. Plain films (Figs. 5.6 and 5.7) showed a small lucency in the anteromedial plafond that may have been consistent with an osteochondral defect. An MRI (Figs. 5.8 and 5.9) did not show a definitive chondral lesion; however, there was high signal in the anterior and medial tibial plafond, suggesting a stress fracture of the medial malleolus. The patient was treated conservatively, and he sat out the remainder of the season. He returned the following year and played successfully without incident.



Fig. 5.6 An anteroposterior (AP) radiograph in an elite college athlete does not show obvious fracture of the medial malleolus.



Fig. 5.7 A lateral radiograph in an elite college athlete does not show obvious fracture of the medial malleolus.



Fig. 5.8 T2 coronal images show increased signal in the anterior medial malleolus. Note that this area appears normal on the initial plain radiographs.



Fig. 5.9 T2 sagittal images show increased signal in the anterior medial malleolus. Note that this area appears normal on the initial plain radiographs.

CASE STUDY 5.2

An elite-level, male athlete experienced debilitating pain in the ankle. Examination and imaging were consistent with a stress fracture of the medial malleolus. Axial CT images clearly demonstrated involvement of the antero-medial tibial plafond (Fig. 5.10). The player was not able to return to preinjury performance after nonoperative management. Therefore he was treated with internal repair of the vertical fracture fragment (Fig. 5.11).

STRESS FRACTURE OF THE HALLUCAL SESAMOIDS

Located on the plantar surface of the great toe metatarsophalangeal (MTP) joint, the hallucal sesamoids are an often neglected and inadequately respected pair of tiny bones that reside in the flexor hallucis brevis. They are capable of causing an enormous amount of pain, discomfort, and disability in the running and jumping athlete. Stress fractures of the sesamoid is an unusual and rare diagnosis that requires clinical and radiographic perseverance on the part of the treating clinician.

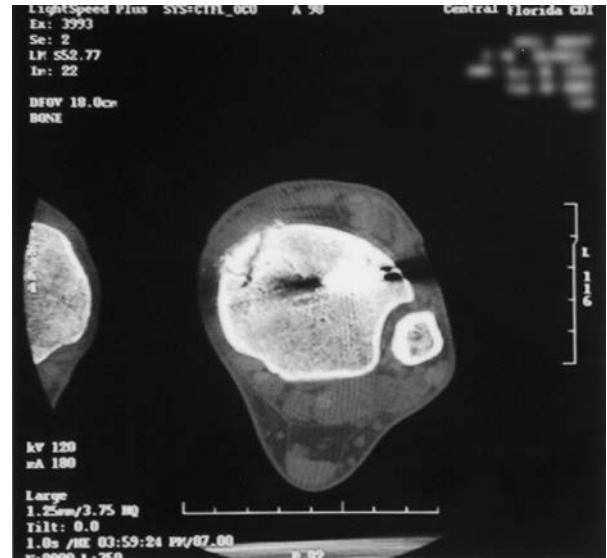


Fig. 5.10 Axial computed tomography (CT) scan showing stress fracture of the antero-medial tibial plafond.



Fig. 5.11 Internal repair of vertical stress fracture of the medial malleolus.

Anatomy and Presentation

Rarely will a patient report focal pain of his sesamoid bones. Rather, he or she often describes a gradual onset of pain about the plantar surface of the great toe. This pain often is exacerbated by dorsiflexion of the hallux. In some instances, pain may be replaced by paresthesia of the great toe. Conversely, the patient may recall a specific incident in which he or she experienced a loud pop or snap on toe-off. Key features of the history should include changes in activity level, adequacy of footwear, and other important risk factors for stress fracture previously described.³⁵

Physical examination should include a detailed, segmental analysis of the hindfoot, midfoot, and forefoot. Cavus are predisposed to sesamoid injury because of the increased load placed on the first metatarsal head. Direct palpation of the sesamoid will elicit pain. The tibial (medial) sesamoid is most commonly involved. Furthermore, one may note decreased dorsiflexion of the first MTP joint and pain with range of motion. The corollary to this finding may be decreased strength of plantarflexion of the first toe.³⁶

The hallucal sesamoids increase the mechanical advantage of the flexor hallucis brevis. The sesamoid location subjects them to enormous amounts of force when the phalanx is dorsiflexed

and planted. The medial sesamoid is injured more often, owing to its larger size and more demanding role in weight bearing.

Imaging

Plain radiographs of the foot are not often helpful in the diagnosis of sesamoid stress fracture. The clinician first must understand that a standard lateral view is essentially useless, and an anteroposterior (AP) of the foot is infrequently revealing. Medial and lateral oblique views of the sesamoids will more clearly visualize the tibial and fibular sesamoids, respectively. Several patients will have normal radiographs or the appearance of a bipartite sesamoid. The role of scintigraphy, CT, and MRI continues to evolve.

Many authors have recommended the use of bone scintigraphy (or now SPECT/CT scan) in the evaluation of sesamoid pain. However, the ordering physician must communicate the need to perform oblique scans because a traditional anteroposterior bone scan of the foot can reveal first MTP activity that can obscure the sesamoids. A study of army recruits³⁷ found no difference in sesamoid bone scan activity between soldiers in basic training for several weeks in comparison with sedentary adults. They cautioned readers about the interpretation of increased uptake in the sesamoid, warning that this may be normal physiologic activity for this bone.

Perhaps axial imaging serves a more important role to the surgeon who potentially will treat the patient with excision of one of the sesamoid fragments. CT is an excellent modality for detection of sesamoid stress fractures. However, obtaining only axial images of the sesamoid can result in a false negative by “skipping” the fracture line. This error can be prevented by supplementing axial CT images with longitudinal cuts through the sesamoid.³⁶

Improved availability of high-quality MRI may supplant the use of CT and bone scan because it enables the treating physician to obtain axial and longitudinal images, as well as indicators of stress fracture such as edema. Imaging facilities must use the appropriately sized coil for imaging of the sesamoids to ensure the proper resolution. High-resolution MRI of the sesamoid will show fragmentation and marrow changes in the face of acute stress fracture. Although MRI may not clearly define stress fracture versus avascular necrosis or chronic nonunion, this point is moot because treatment ultimately will be the same.

Treatment

In our practices, we favor a conservative approach consisting of a nonweight-bearing, short-leg cast for 6 to 8 weeks. Return to jumping and running activity should be graded on the basis of symptomatology. Furthermore, custom orthoses designed to unload the first MTP joint, such as a dancer’s pad or a metatarsal bar, can be instituted after completing a course of casting. Unfortunately, nonunion and delayed union of the hallux sesamoids is a common occurrence.

Management of the recalcitrant sesamoid fracture is surgeon specific and may include bone grafting and ORIF or excision of the sesamoid. Authors have reported excellent results for all types of procedures. Potential pitfalls of operative intervention include digital nerve injury and weakness of the great toe flexor.

A study of six patients reported good or excellent outcomes in dancers and in a long jumper treated with a partial excision of the medial sesamoid.³⁶ Athletes should expect a full recovery but should remain nonweight bearing for 4 to 6 weeks in the postoperative setting, followed by protection of the first MTP joint for another 4 to 6 weeks and a gradual return to activity by 3 to 4 months. A recent meta-analysis³⁸ demonstrated that internal fixation shows the best return to full-level sport rates with low rates of complications.

Surgeons and patients will find that diligent treatment of these seemingly diminutive and insignificant bones can lead to a full recovery and return to competitive sport.

CASE STUDY 5.3

A 30-year-old, recreational athlete presented to a foot and ankle surgeon after a several-day history of right forefoot pain. The pain was associated with a long walk and progressed significantly in the week before the office visit. Examination demonstrated edema of the first MTP joint and pain with dorsiflexion. The patient was exquisitely tender over the tibial sesamoid. Plain x-rays showed a fracture of the tibial sesamoid (Fig. 5.12). This was confirmed with CT. She was placed in a compressive boot with no weight bearing on the forefoot for 6 weeks. She was progressively weaned out of the boot and back to full weight bearing. At last follow-up she had full return to activity and radiographic evidence of callous formation.

STRESS FRACTURE OF THE FIFTH METATARSAL

A constant stream of dialogue exists in the literature regarding the history and treatment of fracture disorders of the proximal fifth metatarsal. Accordingly, misuse of the eponym “Jones fracture” is both propagated and defied. True stress fractures in this anatomic location in fact represent an entirely different injury, with its own mechanism and behavior, and should not be confused with the traditional Jones fracture or an avulsion fracture



Fig. 5.12 A plain radiograph demonstrates stress fracture of the hallux sesamoid.

of the tuberosity (Fig. 5.13). Cavus feet have been radiographically implicated in increasing the risk for developing fifth metatarsal stress fractures.³⁹

Anatomy and Presentation

The history and presentation of this injury are useful in discerning the diagnosis of stress fracture over an acute Jones fracture. DeLee et al.⁴⁰ described three criteria for a fifth metatarsal stress fracture: prodrome of pain in the lateral foot, ultimately leading to debilitating pain; radiographic evidence of stress fracture; and no history of previous fracture and treatment of the fifth metatarsal. Consequently, patients often report a prolonged period of pain on the lateral border of the foot that may be exacerbated by a jumping or running maneuver.

Variations in the anatomy of the proximal fifth metatarsal are described and can be misleading clues for diagnosing fracture of the tuberosity. These variations include the os peroneus, the os vesalianum, and the secondary ossification center of the tuberosity. The os peroneum is a sesamoid bone located in the tendon of the peroneus longus that may occur in up to 15% of normal feet. The os vesalianum is a similar sesamoid, with a less regular shape, occurring only 0.1% of the time. The secondary ossification center or apophysis of the fifth metatarsal does not appear until after age 8 in females and age 11 in males. The apophysis may be present only in up to 50% of feet. This structure can be differentiated from a fracture because the physeal line runs parallel to the shaft of the bone. Conversely, a fracture in this anatomic location generally is in a plane orthogonal to the diaphysis of the bone.⁴¹

Fractures of the base of the fifth metatarsal are subdivided into three types. They include type I tuberosity avulsion fractures, type II Jones fractures, and type III stress fractures of the diaphysis.⁴² Stress fractures are subdivided further into types A, B, and C, which corresponds to early stress fracture, delayed union, and nonunion. This classification scheme is useful because it is anatomically based and describes separate fractures with differing mechanisms.

Imaging

Radiographic diagnosis of fifth metatarsal stress fracture typically is not as elusive as the other bones of the foot and ankle. Patients who present early in the course of their lateral foot pain may have normal radiographs. The first feature to appear is thickening of the cortex and a small periosteal reaction.⁴³ Type I fractures⁴⁴ (acute/chronic) are characterized by a straight line at the junction of the proximal and middle third of the diaphysis. The bone ends are sclerotic, there is minimal periosteal reaction, and there is no widening. Type II fractures (delayed unions) will demonstrate widening with hypertrophic periosteum and a wide band of radiolucency across the diaphysis. The

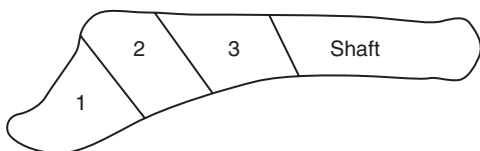


Fig. 5.13 The three zones of injury at the base of the fifth metatarsal. Modified from: Lawrence SJ, Botte MJ. Jones' fractures and related fractures of the proximal fifth metatarsal. *Foot Ankle*. 1993;14:358-65.

medullary canal may be sclerotic. The type III fracture (non-union) differs in that the bone ends will appear to be entirely sclerotic, as though the medullary canal were nonexistent.

The clinical and plain radiograph diagnosis of fifth metatarsal stress fractures rarely requires the use of bone scan or MRI. Scintigraphy will demonstrate increased uptake within 72 hours of acute injury but is less specific. As in other stress fractures, MRI will clearly demonstrate a fracture line with surrounding edema and signal change.

Treatment

Management of fifth metatarsal stress fractures is determined on the basis of the needs and goals of the athlete, as well as the radiographic classification of the injury. Surgeons may opt to be more aggressive in professional athletes, who are dependent on a rapid return to play. Conversely, patients may advocate a less invasive approach to initial management. All athletes with this injury should be counseled on the pitfalls that may be encountered, including nonunion and temporary disability. Authors favoring conservative management have reported lackluster results.^{40,43} Specifically, patients are prone to prolonged immobilization and nonunion. Improved results have been demonstrated with surgical intervention. As such, this modality is advocated in most athletes who desire early definitive treatment.⁴⁵

Torg et al.⁴⁴ have demonstrated that acute, nondisplaced stress fractures of the fifth metatarsal can be treated successfully with nonweight-bearing immobilization. The importance of compliance with nonweight-bearing status should be emphasized for the first 6 to 8 weeks, as weight bearing has been shown to diminish healing. The management of type II delayed unions is less clear. Nonweight-bearing immobilization is effective but prolonged, and the specter of nonunion is not unreal. We tend to be more surgically aggressive with athletes and opt for intramedullary fixation.⁴⁶

Nonunions demand intramedullary fixation (or tension band wiring⁴⁷) with or without biologic supplementation.⁴⁸ Nonunions have also been treated with pulse electromagnetic fields and bone grafting. However, patients should be aware that persistent nonunion is a potential complication and possibly is related to screw diameter.⁴⁹

Stress fracture of the base of the fifth metatarsal is a debilitating injury that requires expertise in diagnosis on behalf of the treating surgeon. Mistaking this injury for a less benign fracture, such as a tuberosity avulsion, can result in painful nonunion and significant loss of playing time. Therefore commensurate management demands a thorough understanding of the anatomy of the fifth metatarsal and the variable fracture patterns existing in this location. Athletes treated correctly can often expect an excellent prognosis.

CASE STUDY 5.4

A 22-year-old, college-level, female soccer goalie noted lateral border of the foot pain after kicking a soccer ball. Physical examination was consistent with fifth and fourth metatarsal tenderness. Plain films demonstrated a fracture at the base of the fifth metatarsal (Fig. 5.14). She underwent percutaneous screw fixation with a 4.5-mm shaft screw (Figs. 5.15 and 5.16) and had full return to sport 6 weeks postoperatively.



Fig. 5.14 Stress fracture of the base of the fifth metatarsal in a female soccer player.



Fig. 5.15 Anteroposterior (AP) radiograph after percutaneous fixation with a 4.5-mm shaft screw.



Fig. 5.16 Lateral radiograph after percutaneous fixation with a 4.5-mm shaft screw.

CONCLUSION

Poorly defined foot and ankle pain in the athlete can be a frustrating condition for athletes and physicians. Stress fractures represent a subset of maladies of the foot and ankle that require diligence on behalf of the diagnostician. Careful history and physical examination will illuminate mechanisms of injury specific to each fracture type and risk factor, such as weight loss, amenorrhea, and eating disorders. Moreover, stress fractures often require advanced imaging modalities, such as CT, bone scintigraphy, and MRI. Therefore a global approach to care of the athlete is advised. This should involve activity modification, improvements in training, nutritional and psychological counseling, as indicated, and definitive orthopedic intervention. Accurate diagnosis and successful treatment of problematic stress fractures of the foot and ankle is a rewarding and attainable goal for all physicians.

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Ankle and Midfoot Fractures and Dislocations

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INTRODUCTION

Fractures and dislocations of the foot are among the most common injuries in the musculoskeletal system. Sports-related lower limb fractures and dislocation are less frequent than those of the upper limb, but they are particularly problematic for athletes because they often result in significant periods of non-weight rehabilitation. The disability and time away from sports resulting from these injuries warrant close attention to diagnosis and management.

CLINICAL EVALUATION

In evaluating patients with trauma to the ankle, it is essential to obtain a thorough, detailed history to direct the physical and radiographic examination.

Physical examination should be meticulous and systematic. Because of the high incidence of coexisting injuries or fracture/dislocations in the injured limb, careful examination and palpation of points of tenderness should be performed to detect evidence of occult injury. Evaluation of range of motion of the ankle, subtalar, mid-tarsal, and metatarsophalangeal joints is incorporated into every routine examination. A careful motor examination, both intrinsic and extrinsic, as well as a sensory examination are performed. Vascular examination, including Doppler studies, is essential. Radiographs are guided by the examiner's history and physical examinations. Oftentimes, the injured athlete is unable to localize the injured part, so a more global radiographic survey is performed. Standard views of the foot include anteroposterior (AP), lateral, and oblique views. The oblique view, for example, is particularly useful for evaluating joints, such as the calcaneal cuboid joint, that typically are hidden or poorly examined in AP view. Specialty views, such as

axial views of the heel, Broden's view of the subtalar joint, and stress views of the foot, also are helpful in certain circumstances. Because of the complexity of the anatomy and lack of uniform appreciation or interpretation of the foot and ankle radiographs, adjunctive studies, such as computed tomography (CT), bone scan, and magnetic resonance imaging (MRI), can be of tremendous value. These also are particularly useful because of the subtle nature of many foot and ankle injuries.

RADIOGRAPHIC EVALUATION

Standard radiographic examination of the ankle includes three views: AP, lateral, and mortise. These radiographs allow the clinician a clear view of the relationship of the three bones that comprise the ankle mortise—tibia, fibula, and talus. The use of measurements of mortise width; medial or tibiofibular clear space; talocrural angle; "Shenton's" line of the ankle (that space that demonstrates a mirrored congruity between the lateral talar wall and the corresponding curvature of the distal medial fibula); and talar tilt all are helpful in determining the subtle abnormalities of the ankle mortise (Fig. 6.1). When in doubt, the clinician also may obtain contralateral views to determine that which constitutes normal anatomy for that particular patient, because there tends to be a high degree of variability in what is considered normal from patient to patient.

Mortise views should demonstrate relative congruity of the joint space circumferentially—medial tibiotalar, dorsal tibiotalar, and lateral fibulotalar. The distance between these subchondral bone margins should be equivalent. In addition, a congruous relationship should exist between lateral talus and medial fibula, the so-called Shenton's line of the ankle. Abnormalities, as evidenced by incongruity, provide clues to malalignment resulting from bony or soft tissue injury.

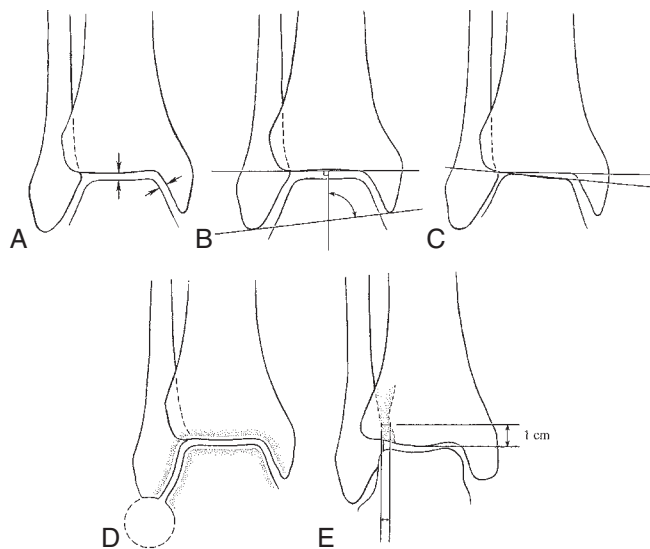


Fig. 6.1 A to F, Schematic representation of radiographic parameters. (A) Medial clear space should equal the articular distance at any point around the mortise. (B) Talo-crural angle. (C) Talar tilt. (D) “Shenton’s line” of the ankle. (E) Tibio-fibular clear space. (F) Tibio-fibular overlap. (From Myerson MS: *Foot and ankle disorders*, St Louis, 1999, Mosby.)

The medial clear space of the ankle as viewed on AP or mortise radiographs is the distance between the medial talar wall and lateral portion of medial malleolus. The measurement of the medial clear space continues to play a significant role in determining the stability of ankle fractures, and thus the need for operative interventions. Although this is a linear measure, it reflects a rotational (external) abnormality of the talus with respect to the tibia. Injury leading to abnormality of this relationship with measurements of less than 1 mm or greater than 4 mm has been shown to correlate with poor outcomes, including chronic pain, instability, and arthrosis.¹⁻³ There remains significant variability in the literature with respect to the correct method of measuring the medial clear space. It is our practice to measure the medial clear space on a weight bearing or external rotation stress mortise view at a level 5 mm below the medial talar dome. It is imperative that the ankle is not plantar flexed in radiographs in which the medial clear space is evaluated, as the narrower posterior talar dome will often produce a falsely elevated measurement of medial clear space widening.

The talocrural angle helps to define the appropriate fibular length. This is measured as the angle between the line parallel to the distal tibial joint surface and another line drawn between tips of the medial and lateral malleoli. Normal values average 83 ± 4 degrees. Differences of more than 2 degrees to the contralateral normal side suggest fibular shortening.

Talar tilt is measured by determining the angle between articular surface lines drawn parallel to the distal tibia and proximal talus. Although uniform agreement on what is considered normal does not exist, a side-to-side difference of more than 5 degrees (or 2 mm) is considered pathologic.

Syndesmotic space probably is the most confounding of all radiologic measures. Measurements should be performed to

account for the space existing between the medial edge of the fibula and the lateral edge of the tibial incisura, determined at 1 cm proximal to the joint line to ensure reproducibility. Average distance should be less than 5 mm but may vary up to 6 mm in larger individuals. Another measure of syndesmotic integrity is the tibiofibular overlap. The distance between the medial fibula and the lateral edge of the anterior tibia should be 10 mm (see Fig. 6.1).

Ancillary studies, such as CT scanning and MRI, are used liberally to provide more information regarding ankle relationships and stability.

TREATMENT

Generic goals in the treatment of fractures and dislocations of the ankle are as follows:

- Avoiding stiffness and loss of mobility.
- Removing bony prominences, which may result in pressure phenomena.
- Restoring the articular surfaces.

Any fracture or dislocation of the foot or ankle that results in focal skin pressure or evidence of neurovascular compromise must be addressed immediately. Manipulation or even open reduction must be carried out to reduce the potential sequelae, including skin necrosis, neuropraxia, ischemia, and/or pressure-induced necrosis of articular surfaces, because of abnormal loading secondary to malpositioning after fracture or dislocation.

Even anatomic restoration does not guarantee optimal functional outcome, but it certainly provides the athlete with a significantly reduced risk of morbidity associated with sequelae of delayed or untreated injury. However, injuries that present without gross distortion of anatomy or imminent threat to the viability of the limb may be treated better after an appropriate “cooling down” period. This is not to say that they should be splinted and ignored, but a short period should be devoted to rest, ice, compression, and elevation (RICE) to allow the soft tissue integrity and oxygenation to reestablish itself, particularly before the clinician embarks on any invasive procedures.

The evolution of treatment of the traumatized ankle of the athlete has directed more attention to aggressive intervention than to “benign neglect.” Recognition of the fact that long periods of immobilization after trauma may lead to muscular atrophy, myostatic contracture, reduction of joint mobility, associated connective tissue proliferation leading to scarring, synovial adhesion, and cartilage degeneration has prompted a more aggressive approach to ankle injuries, using appropriate surgical intervention to stabilize injuries and institute earlier range of motion and weight bearing when possible. These tenets provide for the ability to institute potential prevention against previously disabling factors such as disuse osteopenia, limb atrophy, proprioceptive losses, and chronic, persistent pain.⁴⁻⁸ Introduction of early range of motion, physical therapy modalities, appropriate splinting, and bracing, as opposed to casting, allows for the earlier restoration of function and avoidance of complications. The static accumulation of hematoma, fluid

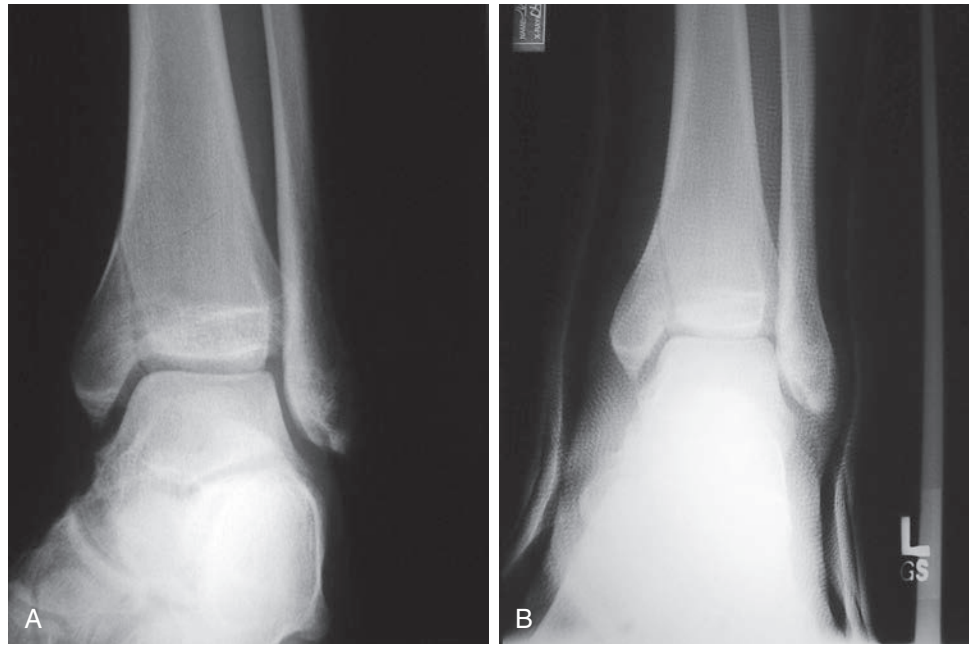


Fig. 6.2 (A and B) Medial malleolar fracture in a 16-year-old basketball player. The athlete elected to undergo nonoperative treatment and healed uneventfully in 6 weeks.

extravasation, and resultant articular and tendinous adhesions is far less with treatment that promotes earlier rehabilitation.⁴ This type of treatment also helps to prevent disabling sequelae, such as arthrofibrosis and regional pain syndromes.⁷

Although the realm of athletically related ankle injuries is too vast to be encompassed in this chapter, the more common injury patterns encountered are addressed. Diagnostic and management controversies are discussed and elucidated for the reader. Rather than a trauma compendium, this is meant to be a guide for the treatment of frequently occurring sports and athletic injuries to the ankle for one's reference and perusal.

ANKLE FRACTURES

Medial Fractures

Isolated medial fractures are unusual but not rare. Fracture patterns may vary from vertical, oblique, or horizontal, depending on the mechanism of injury. Isolated, minimally displaced medial malleolar fractures may be treated with 6 weeks of non-weight bearing cast immobilization. Good functional outcomes and less than 5% rate of non-union have been demonstrated in studies of a general population (Fig. 6.2, A and B).⁹ However, radiographs demonstrating talar tilt or subluxation in association with a medial malleolus fracture should raise suspicion for a “bimalleolar variant” in which lateral ligamentous injury has occurred in deference to bony injury. Patients with radiographic signs of instability of the ankle should be treated operatively. In the athlete, even in the absence of clear signs of instability given the risk of sequelae and potential for instability and abnormal mechanics with medial malleolar fractures, it is the author's practice to repair all but those that are non-displaced. Even those demonstrating minimal (<2 mm) displacement carry some advantage to stabilization, such as reliable fixation, early

range of motion, lack of immobilization, and potentially early return to activity.

As evidenced by Ramsey and Hamilton,¹⁰ as well as Yablon,¹¹ ankle stability is dependent on medial integrity. Michelson and others^{2,12-21} have shown that the talus will not shift abnormally with integrity of medial structures. Therefore, attention should be directed to anatomic restoration of the medial ankle if it is disrupted. Repair may be performed percutaneously with cannulated screw fixation but should be reserved for absolutely anatomic reductions. Any incongruity, as evidenced by articular irregularity, necessitates open repair with restitution of the articular surfaces. The author prefers open techniques because radiographs often may disguise an occult malreduction. Often, anterior/posterior reduction appears anatomic, but evaluation via live fluoroscopy will demonstrate some degree of articular step-off with internal rotation toward a mortise view. The author prefers an open reversed J incision with attention to interposed periosteum and unrecognized comminution at the fracture site. Additionally, open reduction affords the opportunity to inspect the articular surface, which provides useful prognostic information. Fixation is dictated by fracture pattern. Most often, one or two partially threaded cancellous screws are sufficient; however, with a more vertical fracture pattern, several screws with washers or even a small one-third tubular anti-glide plate will be indicated (Fig. 6.3). Recent attention has promoted the development of fracture-specific implants, and a variety of medial malleolar hook plates now exist as well to provide more options to the surgeon for better fixation in an effort to secure the fragment(s) and gain earlier mobility.

Once wound healing is stable, range of motion and resistance exercises are instituted. Weight bearing is restricted until 4 weeks and is advanced on the basis of symptoms. Results generally are good.

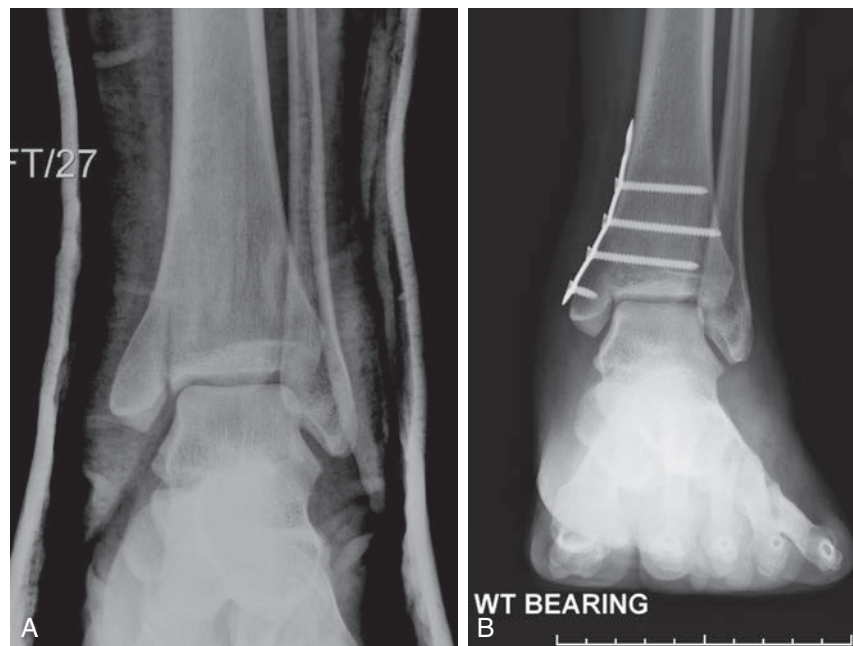


Fig. 6.3 (A and B) Vertical shear type medial malleolus fracture. Anti-glide plating provides excellent stability allowing early range of motion and weight bearing.

Posterior Malleolus Fracture

The posterior malleolus encompasses the posterior projection of the distal tibia plafond. It is bordered medially by the groove for the posterior tibial tendon and laterally it includes the posterior aspect of the tibial incisura. The lateral tubercle of the posterior malleolus serves as the origin of the posterior inferior tibiofibular ligament (PITFL), which plays an important role in the stability of the distal tibia-fibular syndesmosis. Ogilvie-Harris and colleagues²² showed in a cadaveric study that the PITFL was responsible for 42% of the stability of the syndesmotic ligament complex. Thus, any posterior malleolar fracture (PMF) involving the posterior tubercle has the potential to result in an unstable syndesmotic injury. It is also important to recognize that large triangular posterior malleolar fragments may involve a significant portion of the tibial incisura, and associated malreduction of this fragment has the potential to result in malreduction and altered biomechanics of the syndesmosis (Fig. 6.4A).^{23,24} Despite these anatomic considerations, the role of anatomic reduction and fixation of PMFs continues to be a topic of considerable debate.

Large PMFs have been implicated in posttraumatic arthritis, and thus fractures involving greater than 25%–33% of the posterior tibial articular surface have traditionally been recommended for operative reduction and fixation.^{25–29} More recently, many authors have advocated a more aggressive approach, recommending fixation of even small fractures of the posterior malleolus based on a potential for syndesmotic malreduction and posterior micro-instability of the tibiotalar joint.^{27,30–32} Some surgeons have suggested this is conceptually akin to a bony Bankart lesion in the shoulder.

Several cadaveric biomechanical studies have attempted to elucidate the importance of the posterior malleolus fracture size to ankle stability and force transmission. Initial

studies suggested that degeneration after posterior malleolus fracture malunion may result from decreased contact area and increased contact pressures at the tibiotalar joint.^{26,28,33} However, more recently, these results have been called into a question by a number of studies that suggest that posterior malleolus fracture malunion effects force transmission by changing the area of contact without a decrease in contact area or change in peak contact pressures at the joint.^{34,35} It has also been noted that, in addition to changing the contact area, larger PMF place increased stress on the lateral ligamentous structures including the anterior tibiofibular ligament (ATFL) and anterior inferior tibiofibular ligament (AITFL), particularly with ankle dorsiflexion.³⁴ It is postulated that posttraumatic arthritis following posterior malleolus fractures, rather than resulting from increased contact pressures, may result from a change in the pattern of force distribution resulting in forces in areas of the joint that may not typically be subjected to loading.

While our understanding of the biomechanics of posterior malleolus fractures continues to progress, the current cadaveric studies may oversimplify the biomechanics of the ankle, particularly when the posterior malleolus fracture is a component of a more complex injury pattern including adjacent bony and ligamentous injuries. Thus, when making treatment decisions for posterior malleolus fracture, it is important for the clinician to take into account the totality of the ankle injury. Below we outline our treatment algorithm for PMFs.

Posterior malleolar fractures are typically easily identified on a lateral radiograph, but their characteristics are best evaluated with computed tomography. Computed tomography allows for a more accurate determination of fracture size, articular impaction, comminution, syndesmotic disruption, and medial extension, in many cases changing the decision to treat as well as the

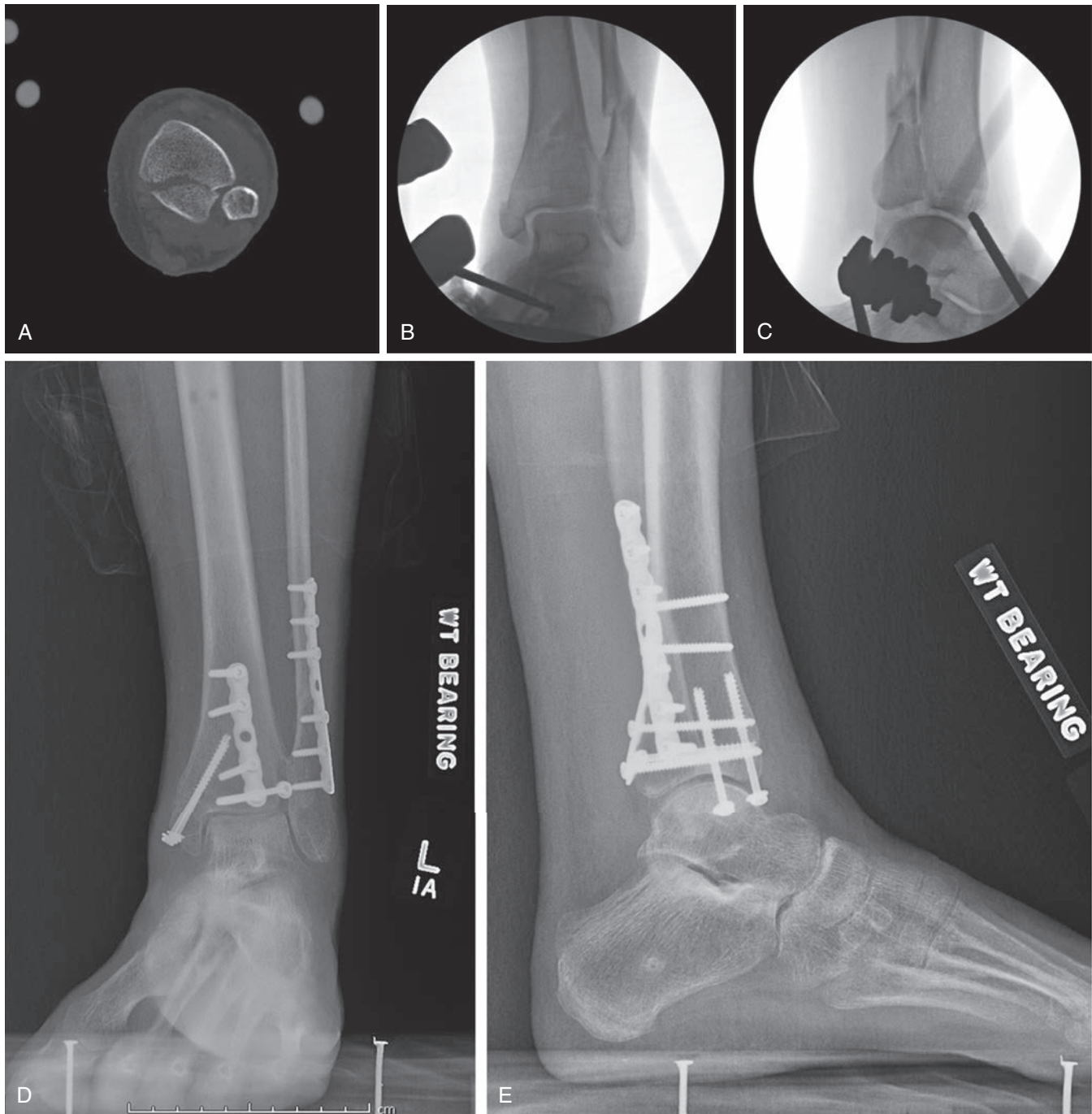


Fig. 6.4 (A through E) (A) Displaced posterior malleolus fracture resulting in malalignment of the tibial incisura and syndesmosis. (B and C) Intraoperative radiographs showing a displaced trimalleolar ankle fracture. (D and E) Postoperative radiographs with buttress anti-glide plating of the posterior malleolus. Note the syndesmosis is stable after fixation of the fracture.

operative approach.³⁶⁻³⁸ It is our practice to obtain CT scans of nearly all fractures involving the posterior malleolus.

Isolated fractures of the posterior malleolus are rare, with an estimated incidence of 0.5% to 1% in the general population.^{39,40} Non-displaced, isolated PMFs may be considered for nonoperative treatment, with 6 weeks of non-weight-bearing cast immobilization. Our experience would indicate that these fractures are exceedingly rare in the athletic population, and when encountered, we recommend CT evaluation to better quantify

the degree of displacement and evaluate for syndesmosis injury as well as to look for occult concomitant injuries. Isolated PMFs are often fairly large, and if any degree of displacement or instability is present, we advocate for open reduction and internal fixation. This approach ensures anatomic reduction of the joint surface and restores the anatomy of the syndesmosis, while allowing for early range of motion and protected weight bearing. Patients with large isolated posterior malleolus fractures with stable fixation may initiate weight bearing as soon

as 2 weeks postoperatively and advance as symptoms allow. Immobilization may be discontinued at 6 weeks and return to play is allowed after completion of a proper ankle rehabilitation program, as soon as 8–12 weeks postoperatively.

More commonly, posterior malleolus fractures are treated as a component of bimalleolar or trimalleolar fracture variants. Treatment of posterior malleolus fractures in this scenario is often more complex and is dependent on the overall stability of the ankle joint. As mentioned above, fractures involving greater than 25%–33% of the articular surface were traditionally recommended for surgical reduction and fixation, based on early biomechanical studies in which larger fracture fragments were thought to result in greater instability and increased joint contact pressures. The concept of fracture size is likely less important in making treatment decision as is the requirement to restore structural integrity to the ankle joint. As noted by Bartonicek,²³ restoration of the anatomy of the fibular incisura and the distal tibia-fibular syndesmosis may be more clinically significant in restoring function and limiting arthritis. We continue to treat these fractures aggressively. Our indication for surgery include a PMF involving greater than 25% of the articular surface, disruption of the distal tibia-fibular syndesmosis, and we tend to fix fractures of any size in the presence of a posterior dislocation.

We prefer to approach these fractures through a posterior lateral approach to the ankle. This approach allows for easy visualization of the PMF for reduction and fixation. In cases in which an intercalary fragment must be reduced, the PMF can be hinged on the PITFL or the piece can be pushed inferiorly with a tamp or freer. We prefer to use a buttress plate due to the better biomechanical strength of this construct (Fig. 6.4, A through E). Alternatively, cannulated screws with washers may be used. It is still debatable as to whether screw placement is better from either the front or back.

Postoperatively patients are immobilized in a splint for 7–10 days. Patients in which the posterior lateral approach is utilized may develop significant stiffness, and we prefer to place these patients into some form of removable immobilization and initiate physical therapy as soon as 1 week after surgery. Weight bearing may be initiated as soon as 4 weeks postoperatively for solidly fixed fractures, but is typically delayed until 6 weeks. Return to play typically commences between 10–14 weeks if aggressive rehabilitation is undertaken.

Lateral Fractures

Isolated lateral malleolar fractures present one of the most challenging management dilemmas in the realm of sports injuries. Associated syndesmosis widening or medial injury, bony or ligamentous, make the choice of treatment fairly simple and obvious.^{18,41–43} However, fibular fractures at any level without concomitant injury or significant radiographic displacement generate varied and controversial opinions as to what is considered appropriate intervention.

On one hand, arguments may be made that surgery is unnecessary because, even though the lateral stability is compromised, it is not completely diminished. Intact medial structures, specifically the malleolus and deltoid ligament, provide primary resistance to lateral talar translation, thus limiting or

preventing abnormal ankle mechanics. Several studies support displacement, lateral or posterior, of up to 5 mm without significant compromise in clinical outcomes.^{42–46} Physiologic loading studies of the normal and compromised ankle suggest that the medial structures are, in fact, most important for stability.^{2,11–20,46,47} It also has been shown by CT analysis that fibular displacement occurring as a result of an external rotation force with intact medial structures (Lauge-Hansen SER2) is the result of internal rotation of the proximal fragment.¹⁹ This implies that the distal fibula maintains its relationship with the mortise and that no functional incongruity is present (Fig. 6.5, A through D). Clinical studies have supported this notion, demonstrating good results with up to 30-year follow-up on nonoperative treatment of isolated lateral malleolar fractures.^{43,44,48,49}

Alternatively, an argument may be made for repairing all but nondisplaced fibular fractures, the rationale being that even small increments of displacement may lead to fibular shortening or mortise widening.^{4,11} Early mechanical testing suggested that the lateral talar displacement of as little as 1 mm would significantly increase contact pressures in the tibiotalar joint, thus creating a potential predisposition to early arthritic changes.¹⁰ In addition, it was shown that the talus would routinely follow the displacement of the fibula, thus lending itself to anatomic malpositioning and subsequent abnormal loading stresses (Fig. 6.6, A through F).¹¹

However, these studies^{10,11} are some of the most often misquoted or misinterpreted in the literature. These analyses were performed in vitro and, as such, focused specifically on the relationship between the fibula and talus after eliminating all other attachments. There was no medial restraint to motion; thus, even though the results can be viewed as reliable and truthful, they bear limited clinical applicability because the contribution of the medial osseous and ligamentous structures was ignored. Appropriate interpretation of these studies suggests that abnormal ankle mechanics may be encountered when a fibular fracture exists in the face of medial deficiency. In these cases, operative treatment should be used.¹⁸ However, these studies fail to speak to the long-term, clinical consequences of a truly isolated lateral malleolar fracture.

More practical arguments for operative fixation in the athlete are more reliable reduction in the face of unclear medial injury; anatomic bone-to-bone contact, facilitating primary bone healing; faster recovery times; and earlier return to weight bearing; stabilizing weight bearing; rehabilitation; and shorter duration of pain. All are anecdotal, and none have been demonstrated in a prospective comparison study of operative versus nonoperative treatment specific to this injury pattern.

Controversy persists surrounding the process of decision making. Despite evidence to the contrary, many surgeons perform, and athletes elect to undergo, repair of the injured lateral malleolus, presumably for fear of abnormal and untoward results of pathologic mechanics and to resume activity as quickly as possible. A large body of clinical evidence favoring this faction is the demonstrated lack of reliability of reproducible medial tenderness on clinical examination in disclosing the presence or absence of deltoid ligament injury.⁵⁰ It is unclear as to what degree of deltoid injury in the face of the fibular fracture will allow for



Fig. 6.5 (A and B) Nondisplaced distal fibula fracture that this athlete elected to treat without surgery. (C and D) Note that, despite clinical healing, radiographs still disclose a fracture line at 4 months. The athlete was asymptomatic and back to full activity.

clinical instability.¹¹ Therefore many surgeons ascribe to the philosophy that it is better to be aggressive, especially in someone whose livelihood may depend on the anatomic function of an ankle or lower extremity. Again, the perspective is anecdotal but reasonable. Surgical treatment often is pursued, as detailed later.

Nonoperative management consists of immobilization until swelling and pain allow motion, usually about 10 to 14 days. Subsequent weight bearing ensues in a walking boot, again, when symptoms abate. In most instances, athletes are back to protected weight bearing somewhere between 3 and 4 weeks. The walking boot is maintained until full weight bearing and nearly normal range of motion are restored. Physical therapy focuses on maintaining muscle tone, joint mobility, and proprioception during the healing phase. Return to activity is dictated by relief of pain, normal symmetric joint range, and strength equal to 80% of that in the normal, unaffected side.

Sports-specific activities are resumed with protective taping or bracing as necessary. Radiographs are monitored frequently in the first month to ensure no displacement, but after 4 weeks these typically are not helpful as long as no changes are noted, specifically no mortise widening.

Should one embark on the surgical management of the isolated lateral malleolus fracture, operative principles of anatomic restoration and rigid fixation apply. The goal is to allow early mobilization and quick recovery. Debate still exists regarding the use of interfragmentary fixation combined with lateral buttress plating versus posteriorly placed, anti-glide fixation. Lateral plating is technically easier, whereas posterior plating theoretically provides greater mechanical stability.^{51,52} Both seem to perform well clinically. No current consensus exists, and the method remains the preference and comfort level of the surgeon.

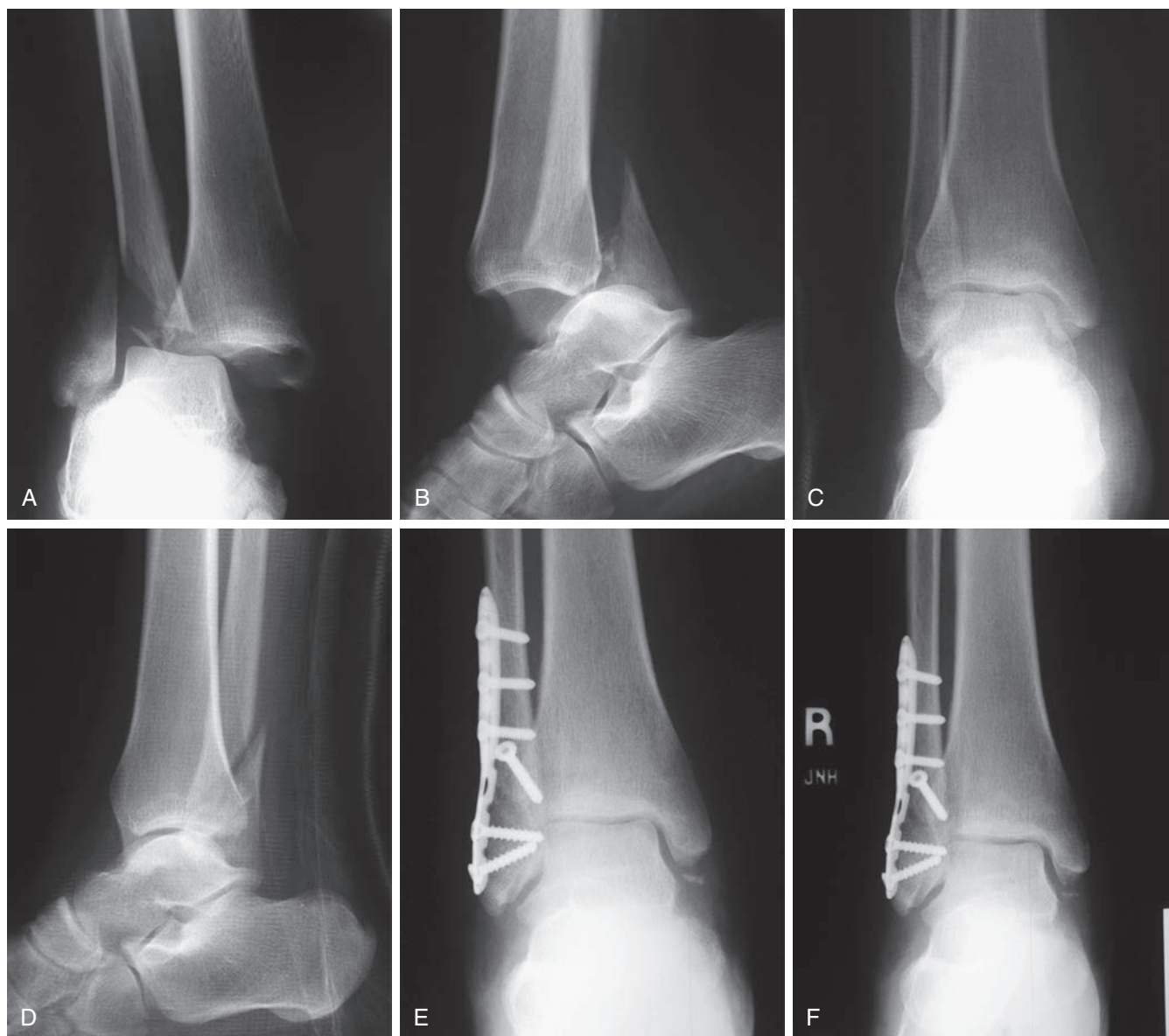


Fig. 6.6 (A and B) Displaced fractures of the fibula with mortise widening require open reduction and internal fixation, with possible attention to the deltoid ligament if the mortise remains widened. (C and D) Even after anatomy is restored through closed reduction, stability is in question. (E and F) Open reduction and internal fixation (ORIF) ensures anatomic restoration of the joint and allows early institution of joint motion and therapy.

Weber A variant lateral malleolus fractures present another clinical dilemma. The majority of these fractures represent stable injuries, and may be treated with a short period of non-weight bearing immobilization. Early range of motion is initiated once swelling and tenderness has diminished, and weight bearing is usually possible within 10–14 days of the injury in a fracture boot. In cases with significant displacement at the fracture site or athletes requiring a more expedited return to play, surgical stabilization may be permissible. Depending on the size of the fracture, a single 4-mm or larger intramedullary screw or a hook plate provides stable fixation. Postoperative recovery commences with a short period of non-weight-bearing immobilization. Weight bearing is allowed at 10–14 days in a fracture boot, and an ankle rehabilitation program is initiated. Return to play is allowed as early as 6–8 weeks postop if managed aggressively in the postoperative period.

Bimalleolar/Trimalleolar Fractures

Little debate exists regarding treatment of bimalleolar/trimalleolar ankle fractures. In an athletic population, uniform agreement exists regarding the need for operative intervention (Fig. 6.7, A through D).^{41,42}

Some caveats do exist, however. Particular attention should be paid to the fibular length and rotation. Any degree of malreduction may lead to abnormal mechanics and possibly could hasten the advance of degenerative arthritis.

High fibular fractures associated with bimalleolar fracture patterns should be stabilized rigidly and anatomicallly. All injuries should be tested for syndesmotic stability, but especially those demonstrating a medial soft tissue injury. This test can be done by directly visualizing the syndesmotic ligaments while applying a laterally directed pull on the fibula with a towel clamp, reduction tool, or other grasping object. Any laxity in



Fig. 6.7 (A through D) Bimalleolar and trimalleolar fractures require open treatment.

tibiofibular stability associated with a fibular fracture more than 3.5 to 4.0 cm from the joint should be stabilized with syndesmosis fixation (Fig. 6.8, A through D).¹²

Syndesmosis fixation remains a topic of debate and is discussed in more detail in the syndesmosis chapter (see Chapter 15). In the athletic population, the author prefers to use one or two suture-buttons to secure the syndesmosis. Suture-button fixation of the syndesmosis allows for stable fixation with the advantage of allowing some rotational motion at the syndesmosis (see Fig. 6.9, D). In addition, the use of a dynamic implant obviates the need for hardware removal in the case of pain or screw failure. Alternatively, a 3.5-mm screw with three cortex fixation and a plate long enough to incorporate the screw proximally to the distal-most hole (see Fig. 6.8, D). If screws are removed, routine screw removal is performed after 12 weeks on

the basis of biomechanical evidence of abnormal ankle mechanics in the face of restricted talofibular motion.⁵³ This reduces the risk of a free-standing screw hole as a stress riser and theoretically allows quicker, safer, and more reliable return to activity.

Trimalleolar fractures at least should have the medial and lateral components repaired. Fixation of the posterior fragment of tibia as discussed above remains controversial, and stabilization should be considered in patients with large fragments, significant displacement, syndesmosis injury, and intercalary fragments.

It is also important to discuss the controversies surrounding deltoid ligament repair in trimalleolar equivalent fractures. Traditionally, these injuries have been treated indirectly with restoration of the ankle stability by way of anatomic stabilization of the lateral malleolus fracture and syndesmosis. This

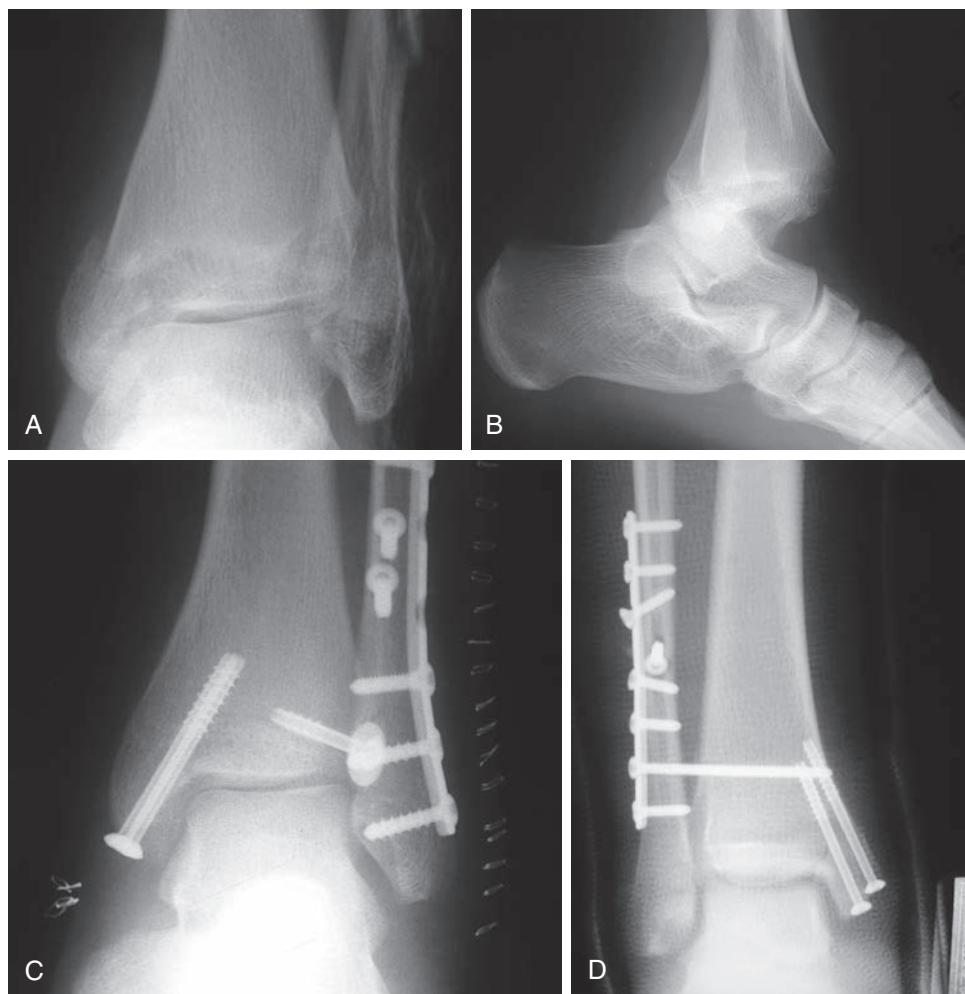


Fig. 6.8 (A through D) Syndesmosis repairs should be performed at the level of injury occurrence and be based on the stability of the joint after malleolar repair. (A and B) Displaced bimalleolar fracture in an adolescent wrestler. Note the avulsion of the anterior inferior tibiofibular (AITF) ligament from the distal tibia. (C) Malleolar repair with a screw in the syndesmotomic fragment. (D) More traditional fixation for a higher-level fibula fracture and persistent tibio-fibular widening.

approach has resulted in satisfactory clinical outcomes in many studies.^{45,54-56} Nonetheless, many of the level IV studies published on this matter do not clearly define what a satisfactory outcome is, and also do not elaborate on the clinical findings in those patients with unsatisfactory outcomes. Stromsoe et al.⁵⁵ published the only high-quality study on the subject in which 50 patients with ankle fractures were randomized to direct primary repair of the deltoid or in situ healing. They found no clinical difference in outcomes between the direct repair group or non-repair group, and recommended against primary repair of the deltoid. When analyzing this body of evidence, one must be cognizant that the population described in these studies may not reflect the same athletic population that many of us are charged to treat. In many athletes, the goals and expectations regarding level of activity, early return to play, and need for an elite level of function exist, and thus a more aggressive approach to stabilizing the ankle maybe necessary.

In treating athletes with a trimalleolar equivalent fracture we recommend the following algorithm. We begin with anatomic reduction and fixation of the fibula fracture with either a lag

screw and neutralization plate or a posterior anti-glide plate (Fig. 6.9, A through D). The syndesmosis is then evaluated and stabilized in patients with clinical or radiographic evidence of syndesmotomic instability. Valgus and rotational stress testing is again performed to determine if any persistent instability of the deltoid exists. If stress testing produces either increased talar tilt or medial clear space, then the deltoid is primarily repaired, restoring the stability of the ankle mortise. We typically repair only the superficial deltoid, which is commonly avulsed from the medial malleolus. Deltoid repair is typically carried out through drill tunnels or with a suture anchor device. End-to-end repair may be carried out in cases of midsubstance rupture. The deep deltoid is not typically repaired but may be repaired with anchor to post reinforcement if desired.⁵⁷

Postoperative rehabilitation after bimalleolar and trimalleolar ankle fractures typically includes a 7–10-day period of immobilization followed by transition to a fracture boot with initiation of early range of motion. Early mobilization is particularly important in patients with fixation through a posterior lateral approach, as this approach may result in

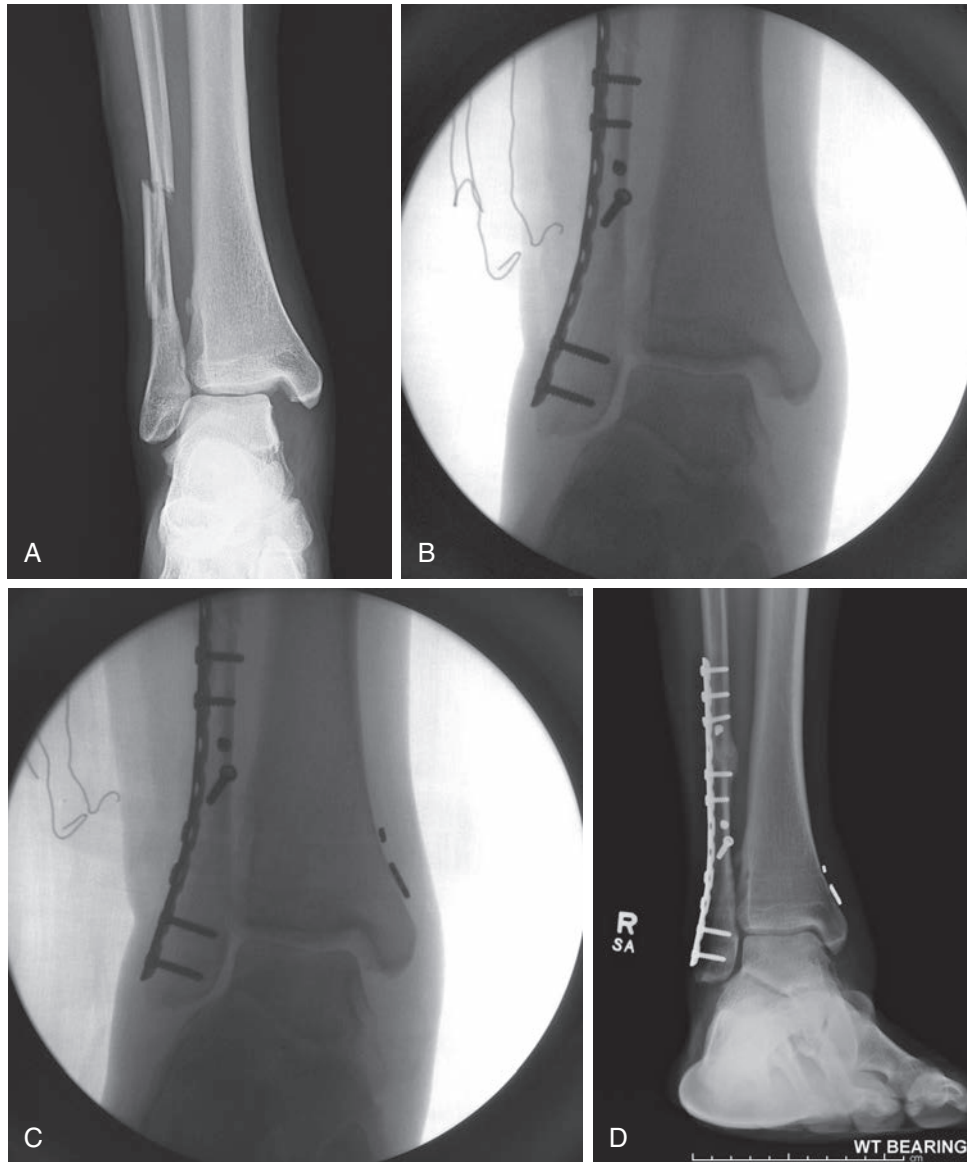


Fig. 6.9 (A through D) (A) Preoperative radiograph of a trimalleolar equivalent ankle fracture in a collegiate football player. (B) External rotation stress after fibula fixation reveals medial clear space and syndesmosis widening. (C) Valgus stress after reduction and fixation of the syndesmosis reveals valgus tilt and medial clear space widening. Deltoid repair is performed to stabilize the medial ankle. (D) Final radiograph at 3 months postop reveals restoration of the ankle mortise and syndesmosis alignment.

significant stiffness at the ankle and contracture if the FHL tendon is immobilized for prolonged periods. Weight bearing is typically delayed until 6 weeks in patients requiring fixation for unstable syndesmosis injuries. In the absence of significant syndesmosis instability and with stable bony fixation, the patient may be allowed to weight bear at 3 weeks postoperatively.

Arthroscopic Evaluation of Acute Ankle Fractures

Despite anatomic reduction, many patients with operatively treated ankle fractures report residual pain, stiffness, and dysfunction at intermediate- to long-term follow-up.^{58,59} Several authors have suggested that these poor results may be due to unrecognized ligamentous or intra-articular injuries resulting from the initial trauma. The incidence of intra-articular

chondral lesion in the setting of acute ankle fractures has been reported to be as high as 88%, with most series documenting lesion in at least 60% of ankle fractures.⁶⁰⁻⁶⁴ Several studies have shown that intra-articular lesions are more common in patients with ankle fracture dislocation and higher-energy variants such as Lauge-Hansen SER IV and PER IV fractures.^{61,62,65}

Traditional means of open reduction and internal fixation (ORIF) allows for a limited evaluation of the ankle joint, especially in presence of an intact medial malleolus. Ankle arthroscopy has been proposed as an adjunct allowing the surgeon to fully evaluate the joint for intra-articular lesion, assist in reduction, and examine the deep deltoid ligament and syndesmosis ligaments. Intra-articular findings may help to guide treatment, but also assist the physician in setting patient expectations and provide potential prognostic information.

Despite the numerous theoretical advantages of arthroscopically assisted treatment of ankle fractures, high-quality studies are lacking to support its use. As mentioned earlier, numerous case series have documented the high incidence of pathology identified with arthroscopically assisted ankle fracture ORIF, but to date no studies have shown that this practice results in a significant improvement in patient outcomes.^{65,66} Nonetheless, the practice of using arthroscopy to assist in treating acute ankle fractures has been shown to be safe, and results in a nominal increase in operative time, typically between 10–15 minutes.⁶⁵

The author uses arthroscopy liberally in treatment of ankle fractures, and finds it to be particularly useful in treating ankle fractures in athletes. Missed intra-articular lesion and inadequately treated syndesmosis and deltoid injuries may result in a delayed postoperative rehabilitation or potentially require reoperation. This can have serious consequences in an athlete attempting to return to play or in those that make a living off their sport.

Patients undergoing ankle arthroscopy at the time of their ankle fracture fixation are positioned supine (Fig. 6.10, A through D). The author typically uses a noninvasive distractor to gain access to the joint, but alternatively the procedure can be done with manual traction. The anterior medial portal is established and a spinal needle is used to provide outflow and guide placement of the anterior lateral portal. Hematoma is always present in the joint in the acute setting, and can be easily cleared with an arthroscopic shaver. After clearing the hematoma, the tibiotalar joint surfaces are inspected for chondral injuries. Unstable articular lesions are debrided, and microfracture may be performed if subchondral bone is intact. Small PMFs and PITFL injuries not identified on radiographs are easily identified with arthroscopy and provide clues to potential instability of the syndesmosis. Injuries to the deep deltoid are easily identified, while the superficial deltoid is more difficult to visualize.

Pediatric Ankle Fractures

Pediatric ankle fractures constitute a wide variety of patterns and complexity. However, these often are encountered in the growing population of high school, junior high, and primary school athletes.

Salter-Harris (S-H) fractures not involving the joint adhere to principles of all generic, pediatric fracture management protocols (Fig. 6.11). Closed anatomic reduction often is successful simply by reversing the mechanism of injury. Cast immobilization typically is effective for management, and bony remodeling usually compensates for any minor malalignments. Immobilization usually is required for 6 to 8 weeks, at which point gradual weight bearing and range of motion may be advanced as tolerated. Any articular incongruity necessitates open management (Fig. 6.12, A and B).

Complexity increases in the diagnosis and management of the adolescent variants of the Tillaux (S-H III) and triplane (S-H IV) fractures. These typically occur in the 12- to 14-year age range as the medial tibial physis begins to close, creating an irregular stress distribution and resistance to forces applied across the ankle (Fig. 6.13).

Tillaux and triplane fractures are considered adult, and issues regarding treatment should be viewed as such (Fig. 6.14). The focus of treatment should be based on congruity of articular reduction, because the complications surrounding these injuries arise from nonanatomic incongruous relationships, leading to early degenerative changes rather than the more popular but erroneous presumption of growth arrest. Abnormalities or asymmetry in growth actually are rare and not terribly consequential in these scenarios.

Any question of articular irregularity should be settled by obtaining advanced imaging studies, specifically CT scanning, to eliminate the possibility of articular step-off. Separations of more than 2 mm in distance along the joint surface, regardless of congruity, should be repaired. No compromise should be accepted at the articular surface for fear of early degenerative changes.

Percutaneous techniques using large reduction clamps or devices and cannulated screw fixation are acceptable, but the surgeon must be certain of anatomic restoration and no interposed tissue. If there is any question regarding adequacy of reduction, open treatment is required. Alternatively, arthroscopic assistance is an increasingly attractive adjunct for this purpose, to avoid larger open procedures. Once stability is ensured, motion may be introduced; however, weight bearing should be withheld for 6 to 8 weeks until healing is confirmed.

Ankle Rehabilitation

The primary focus of rehabilitation following ankle fractures is prevention of stiffness, muscle atrophy, and loss of range of motion. In the immediate postoperative period, patients are typically immobilized in a short leg splint to allow for stabilization of the wounds. At 1–2 weeks following surgery patients are transitioned to a fracture boot and are allowed to initiate early range of motion. Early weight bearing in a fracture boot is typically allowed for stable fracture patterns after the first postop visit, while weight bearing for more unstable fracture patterns commences between 4 to 6 weeks postoperatively or when radiographic healing is noted. In the presence of stable fixation and progressive healing, most patients can be transitioned to a lace-up style ankle brace at 6 weeks postop or after they are walking comfortably in a boot. Regardless of weight-bearing status, it is our practice to initiate physical therapy as soon as the patient transitions to a fracture boot. Again, the primary focus of the physical therapy is on early range of motion, strengthening, and edema control. Patients most prone to stiffness, such as those with a posterior lateral ankle approach, often require more intensive therapy to regain range of motion. Aquatic therapy or mobilization on an anti-gravity treadmill such as the AlterG may be helpful to normalizing gait and strength. Evidence-based measures for return to play are lacking for patients undergoing ankle surgery. It is our practice to allow patients to return to play when strength and range of motion are 80% of the contralateral side. Physical therapist and athletic trainers are often utilized to help make decision regarding return to play, employing test such as the Y balance test and single-limb hop test, which provide an objective measure of the strength, proprioception, and coordination of the affected extremity.

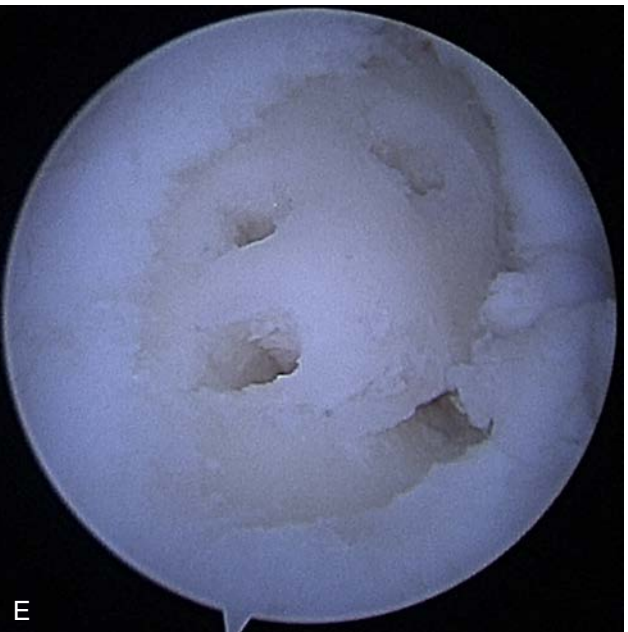
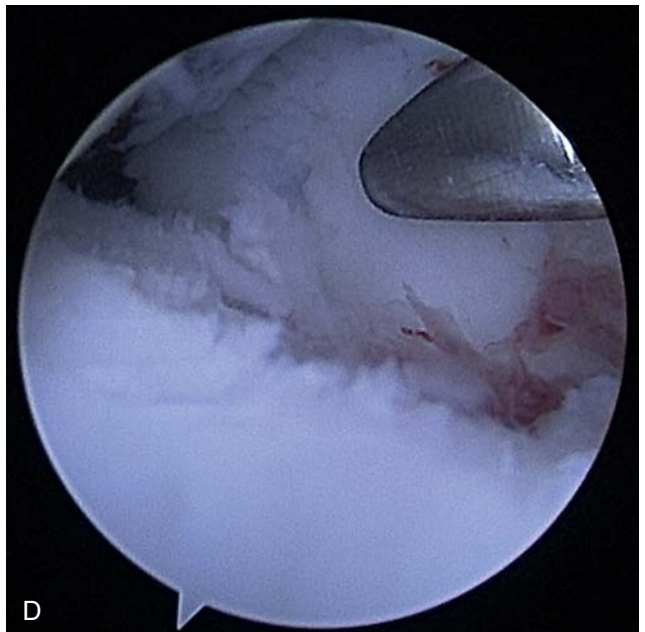
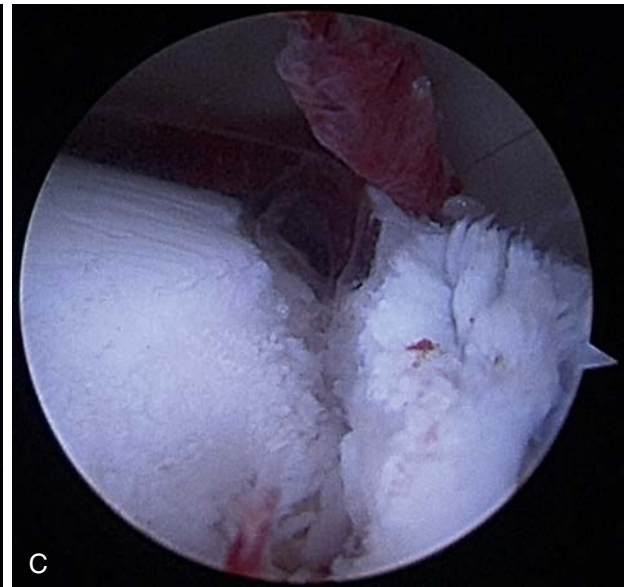
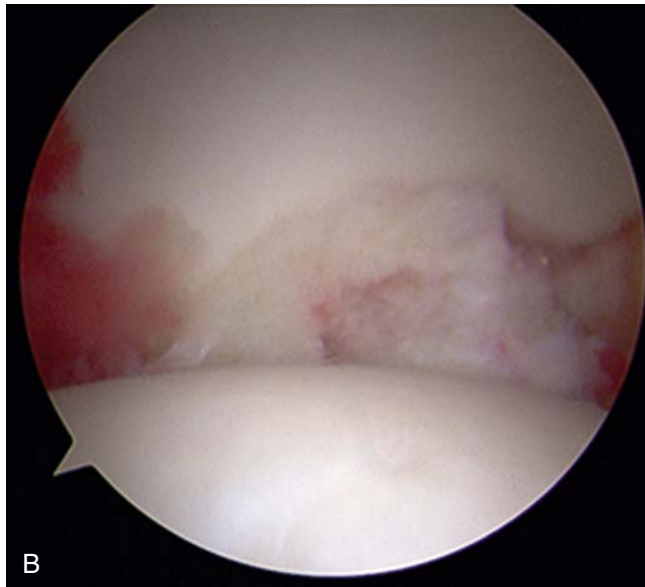


Fig. 6.10 (A through E) (A) Typical positioning for arthroscopic assisted treatment of ankle fractures. The well leg holder is removed after completion of the arthroscopy and ORIF commences. (B) Arthroscopic image showing the deltoid ligament flipped into the joint in a patient with a trimalleolar equivalent ankle fracture. (C through E) Unstable medial talus osteochondral lesion associated with an acute Weber C distal fibula fracture. Debridement and microfracture were performed.

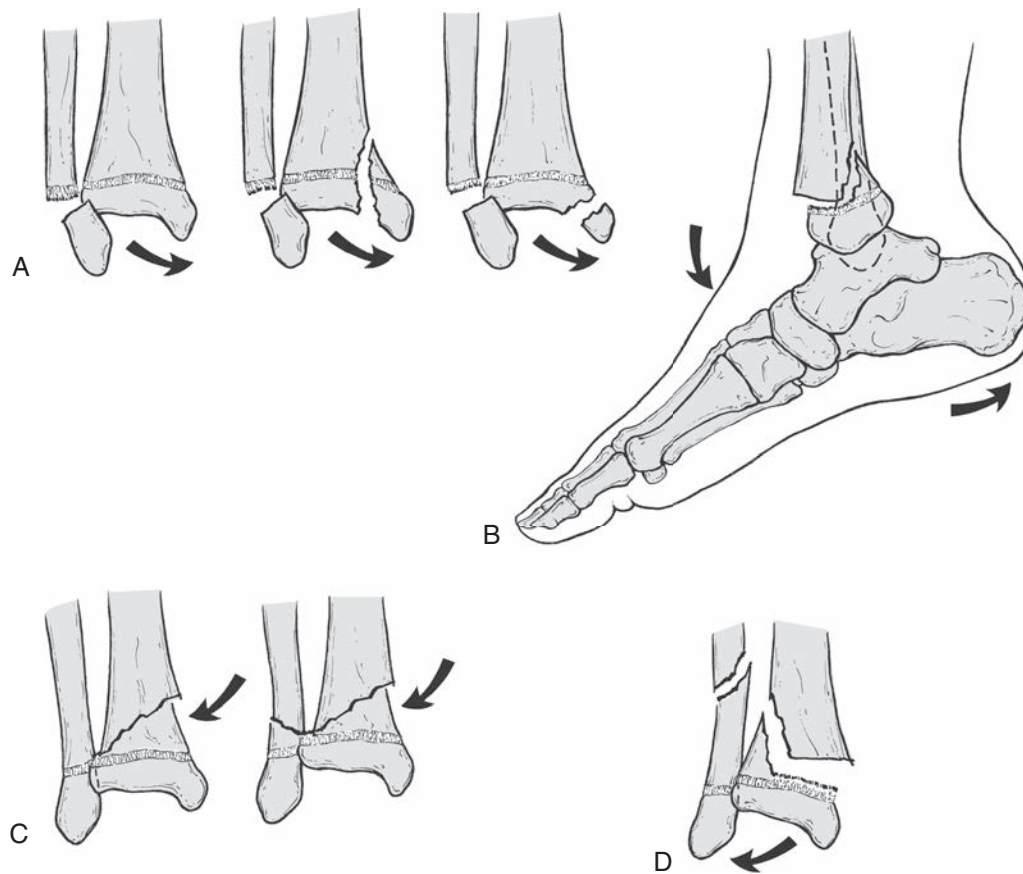


Fig. 6.11 Dias, Tachdjian modification of Salter-Harris' classification of ankle fractures in the immature skeleton. (From Green NE, Swiontkowski MF: *Skeletal trauma in children*, Philadelphia, 2002, WB Saunders.)

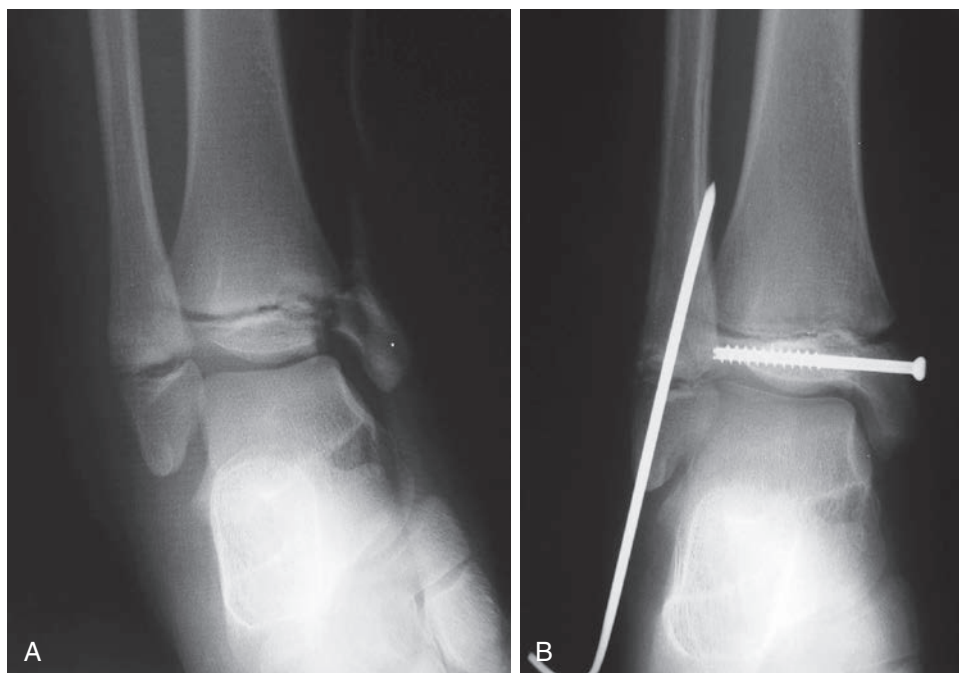


Fig. 6.12 (A and B) Supination-inversion injury of the ankle. (B) With repair. Care is taken to avoid the tibial physis and articular surface. The fibular pin is removed after 4 to 6 weeks.

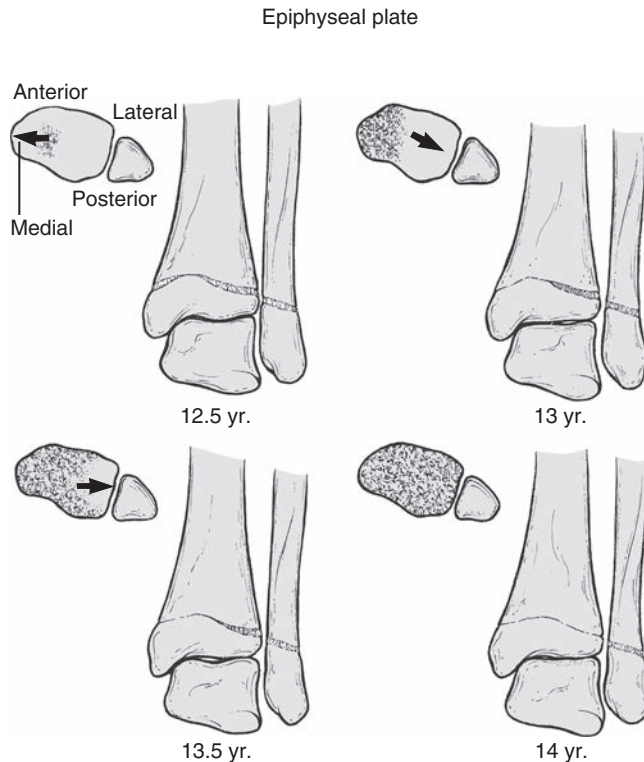


Fig. 6.13 Demonstrating the unusual closure of the distal tibial physis. First, it starts in the middle of the growth plate, then moves anteromedially, and finally laterally. (From Green NE, Swiontkowski MF: *Skeletal trauma in children*, Philadelphia, 2002, WB Saunders.)

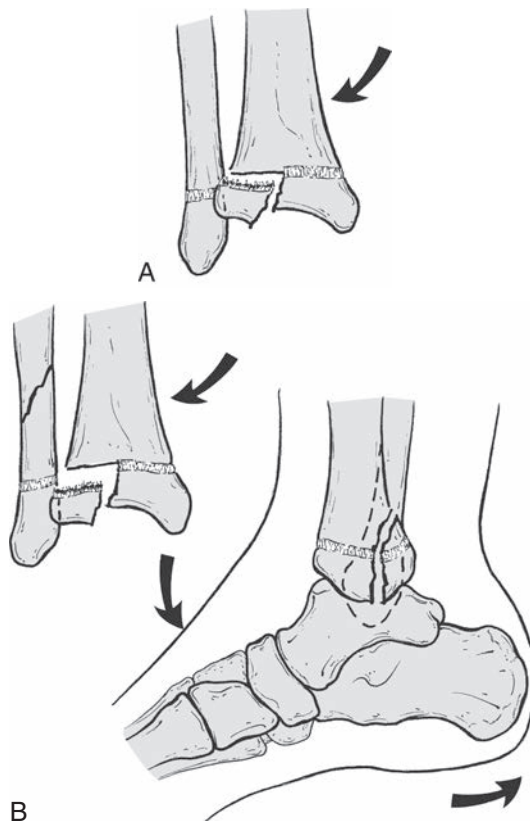


Fig. 6.14 (A and B) Tillaux and triplane ankle fracture variants in the adolescent athlete. (From Green NE, Swiontkowski MF: *Skeletal trauma in children*, Philadelphia, 2002, WB Saunders.)

LATERAL PROCESS TALAR FRACTURES

Fractures of the lateral process of the talus previously have been considered an uncommon injury. Historically, this injury was thought to occur as the result of high-energy trauma and would result from a peritalar dislocation that caused avulsion of the subtalar ligamentous attachments on loading. More recently, however, this injury has gained notoriety because of its strong predilection for presentation after snowboarding injuries. Before the advent of this relatively new winter sport, reports were infrequent. However, with the explosion of attention to this activity by a predominantly young, risk-taking population, the incidence and recognition have risen dramatically—so much so that this injury has been deemed by some as the “snowboarder’s ankle.”^{67,68} One review demonstrates 74 lateral process fractures of the talus that occurred as the result specifically of snowboarding, accounting for 2.3% of all snowboarding injuries. This is, to date, the largest series reported.⁶⁸

Lateral process fractures often are missed, commonly masquerading as chronic ankle sprains. It is easy to understand why this happens because of the relative anatomic proximity of this injury to the anterior talofibular ligament, as well as the lack of reliability of reproducible evidence of fracture on standard radiographic studies. Early diagnosis and treatment, however, are important, because studies have suggested that late recognition and failure to implement treatment routinely lead to poor outcomes such as chronic pain, stiffness, instability, and arthritis.⁶⁹⁻⁷⁶

Traditionally, lateral process fractures were purported to arise from a sudden dorsiflexion inversion force on a fixed foot. However, mechanical loading studies have demonstrated that an acute external rotation or shear force is a key element in reproducing this fracture pattern in a cadaveric model.⁶⁷

Hawkins⁷¹ has classified these fractures into three subcategories (Fig. 6.15). Type I is a simple fracture of the lateral process extending from the tibiofibular articulation down to the posterior talocalcaneal articular surface of the subtalar joint, with or without displacement of the fragment. Type II fractures involve comminution of the fibular and posterior calcaneal articular surfaces, as well as the lateral process. Type III is an avulsion or chip fracture off the anterior and inferior part of the posterior articular processes of the talus. Another classification system has been proposed by Fjeldborg,⁷⁰ who described stages of injury with type I fissuring, type II lateral process fracture with displacement, and type III lateral process fracture with subtalar dislocation. Diagnostically, this fracture pattern presents a dilemma, and a high index of suspicion is needed by the clinician. Injury pattern reports by the patient often are unreliable and inaccurate. Physical examination findings often are similar to those found with an acute, severe ankle sprain with tenderness just anterior and inferior to the tip of the fibula, along with swelling and ecchymosis.

Radiographs sometimes are helpful when large fragments or significant comminution are present but, again, are not reproducibly diagnostic because of the irregular anatomy and overlap of joints in this area.^{69,77} Special radiographic views have been proposed to help elucidate these fractures, including

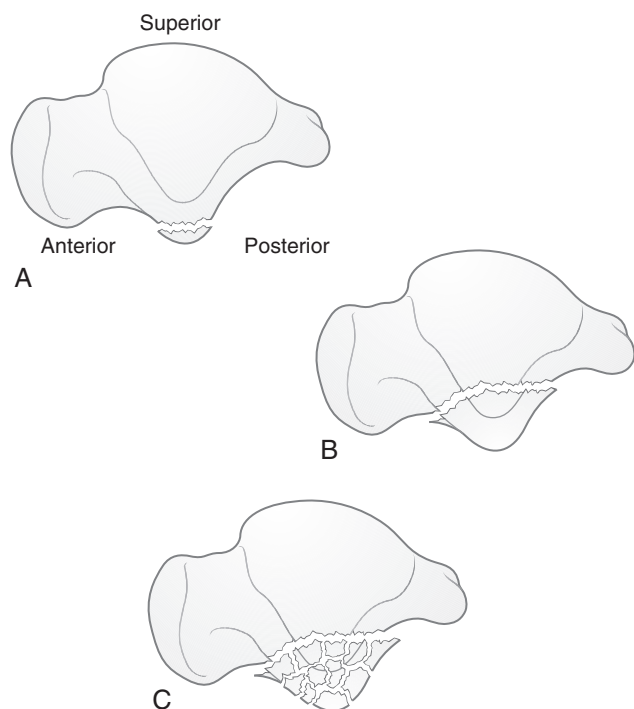


Fig. 6.15 (A through C) Hawkins classification of lateral talar process fractures. (From Boon AJ, Smith J, Zobitz ME, Amrami KM, et al: *Am J Sports Med*, 29(3):333, 2001.)

a 20-degree internal rotation view with the foot in neutral dorsiflexion.⁷⁸ Alternatively, Dimon⁶⁹ has suggested that the ankle be placed in 45-degree internal rotation and the foot plantarflexed at 30 degrees to show the posterior facet in profile. If the diagnosis is entertained, the best and most reliable study remains CT scanning. This not only provides the examiner with diagnostic evidence but also demonstrates the degree of displacement and comminution of fragments. Because of the poor outcomes obtained, all fractures must be sought and treated aggressively. Nondisplaced fractures should not be ignored but immobilized in a cast for 6 to 8 weeks with no weight bearing and then reevaluated at that time for bony union. Failure to aggressively treat larger displaced fracture fragments or comminuted fractures may and often does result in malunion, nonunion, heterotopic overgrowth, subtalar instability, and, ultimately, disabling arthritis.^{71,72,74,75,79-85} Late symptoms have been shown often to not respond to excision of the offending fragments.^{69,79,83}

Treatment is fracture type dependent. Large displaced fractures are managed with anatomic restoration of the articular surface with internal fixation (Fig. 6.16, A through E). These often are large enough to accept at least one small fragment screw for fixation (2.7 or 3.0 mm usually will suffice). This most often can be done through a typical Ollier approach to the sinus tarsi and subtalar joint region. Comminuted fracture patterns are more ominous and carry a more unpredictable outcome. These often are refractory to repair and necessitate excision for all fragments. This at least removes any potentially abrasive surfaces and areas of future impingement. If repaired, early range of motion, focusing on inversion/eversion, will promote restoration of subtalar mechanics.

Sequelae of untreated or missed fractures are well documented. Malunion, non-union, instability, overgrowth, and/or arthritis of the subtalar joint can be debilitating. Missed fractures that present late often are refractory to repair or remove fragments and will necessitate subtalar arthrodesis.^{69,79,83} Therefore it is critical that awareness of this injury pattern remain prevalent and a high index of suspicion be maintained for any patient presenting with atypical or persistently painful ankle sprains.^{7,68,79}

ANTERIOR PROCESS CALCANEAL FRACTURE

Anterior process calcaneal fractures often are missed in the acute setting. This fracture must be sought in anyone with recalcitrant lateral foot pain or ankle sprain; if untreated, it will lead to problematic sequelae. There are two types of anterior process fractures, and they occur by opposing mechanisms of injury: avulsion and compression.^{82,86}

Avulsion injuries occur as a result of a plantarflexion, inversion force (Fig. 6.17A). As such, these often are misrepresented as lateral ankle sprains.⁸⁷⁻⁸⁹ The overall presentation is similar with respect to mechanism in the onset of lateral foot or ankle pain, ecchymosis, and swelling. However, tenderness typically occurs 1 to 2 cm more distally in the region of the sinus tarsi. The fracture fragment often is small and extra-articular, occurring as a plantarflexion and inversion force tensions the bifurcate ligament, which overcomes the attachment of the distal-most calcaneus.

Alternatively, compressive injuries occur with sudden abduction forces across the foot and are much more ominous (Fig. 6.17B). These often will be intra-articular and involve variably sized fragments of joint surface, as well as causing displacement of the fragments posterior, dorsal, or lateral, sometimes leading to substantial incongruity. Because of the similarity in presentation to lateral ankle sprain, a high index of suspicion should be maintained and careful clinical inspection performed. Radiographs are helpful, but a clear and obvious fragment is not always visible. Because these often are confused with ankle sprains, it is not uncommon that only ankle x-rays are obtained. Suspicion should prompt the clinician to obtain foot radiographs, particularly obliques, to verify the diagnosis.⁹⁰ Occasionally, a small fragment or ossicle, the calcaneus secundarium, will be noted on a lateral ankle or oblique foot radiograph. This is smooth and regular in its contours and should be differentiated from the rough, irregular edges of an acute anterior process fracture. Computed tomography may be helpful to determine specific characteristics of acute fracture versus ossicle presence.^{82,90} Additionally, this is recommended for those patients presenting with compressive injuries to determine the degree of articular involvement.

Early diagnosis aids in the quality of treatment for this injury. Healing, particularly of the avulsion type of fracture, is reliable if identified early. Typically, immobilization in a walking boot or a cast for 4 weeks is sufficient. Delay in diagnosis and lack of immobilization can lead to persistent symptoms and affect the ultimate outcome. Occasionally, excision will be required to remove a non-united fragment after delayed or missed diagnosis. Compressive injuries, especially those with displacement, are more complex and carry less-predictable outcomes because

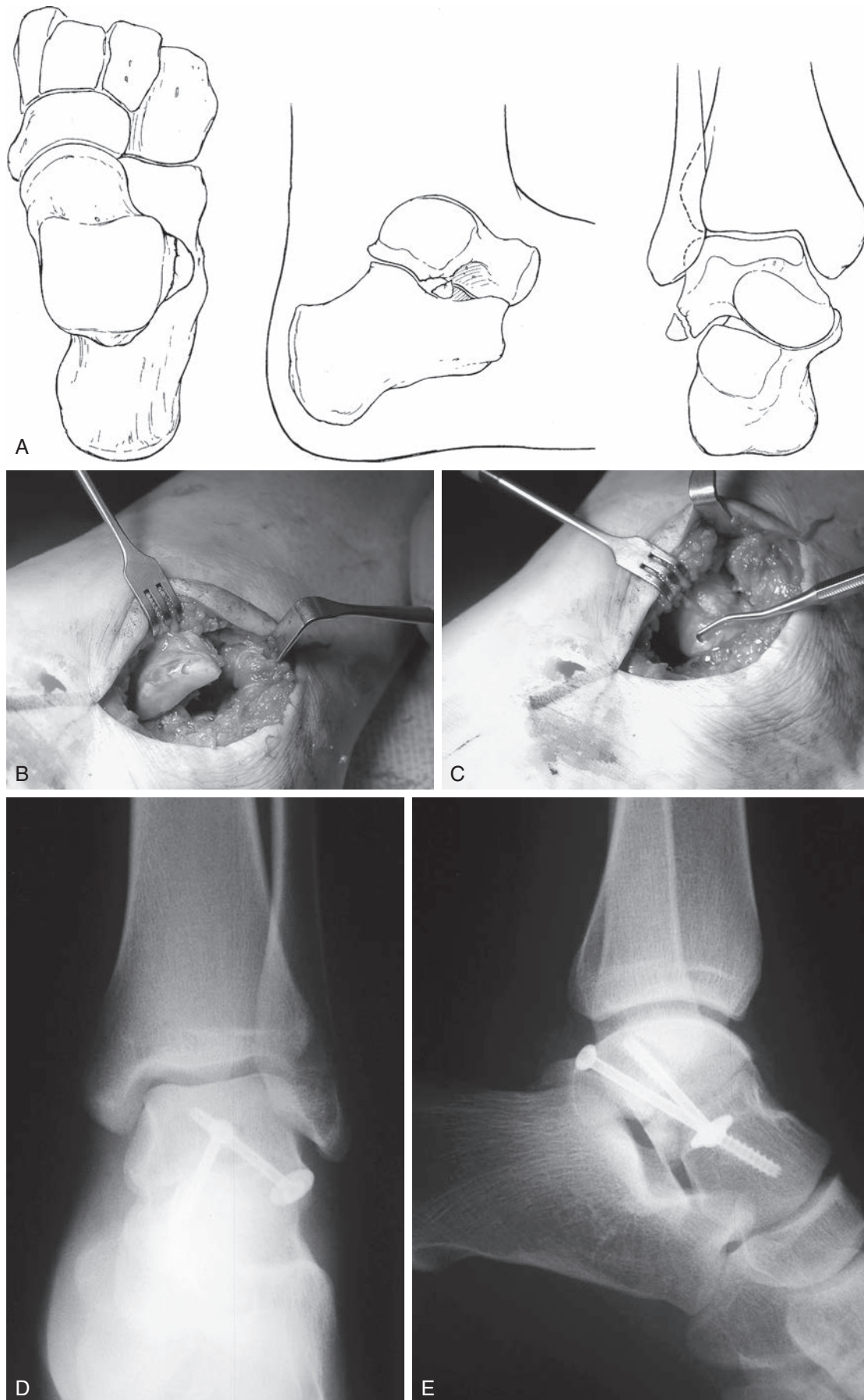


Fig. 6.16 (A) Schematic of a lateral process talus fracture. (B and C) Direct visualization of the lateral process fragment before (B) and after (C) reduction. (D and E) Fixation is achieved with a posteromedially directed screw. A talar neck fracture is fixed here, as well. ((A) from Myerson MS: *Foot and ankle disorders*, St Louis, 1999, Mosby.)

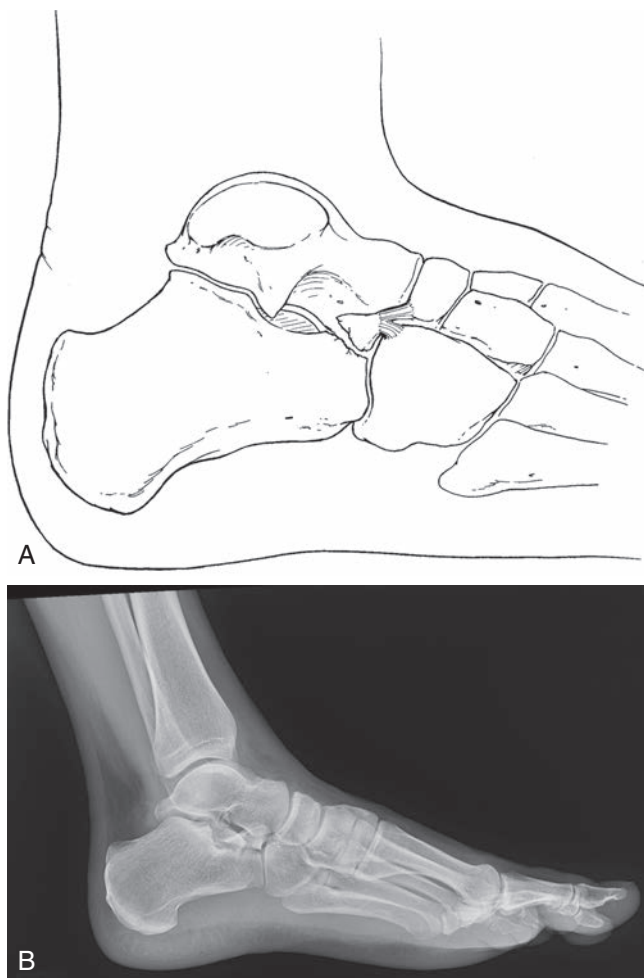


Fig. 6.17 (A) Anterior process fracture of the calcaneus. (B) Radiograph of a larger anterior process fracture in a distance runner. Fractures of this size would be considered for open reduction and internal fixation. ((A) from Myerson MS: *Foot and ankle disorders*, St Louis, 1999, Mosby.)

of the articular damage sustained at the time of the injury. Displacement of the fragments requires early reduction and fixation to restore congruity. Still, these patients may develop degenerative disease at the calcaneal cuboid articulation, depending on the energy of the injury. Results of treatment for any form of these fractures are few and anecdotal. Degan et al.⁹¹ reported on surgical treatment of seven patients who developed symptomatic nonunions after anterior process fractures. Late excision provided pain relief in five of six patients. Surgical treatment may involve a need to osteotomize the calcaneus just below the area of nonunion to excise the entire affected area. Based on location and treatment principles, these may be viewed as traumatic coalitions and addressed in the same way as a congenital calcaneo-navicular coalition. Care should be taken to immobilize the foot for 6 weeks after this procedure, because destabilization may occur as a result of removing the bifurcate ligament, which connects the hindfoot to the midfoot at the navicular and cuboid, respectively. Resumption of full athletic activity after surgical treatment may take up to 6 months; and rarely, in some patients, persistent residual degenerative joint disease symptoms may persist and limit return to sport.^{82,91}

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Midfoot Fractures and Dislocations

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INTRODUCTION

Injuries to the foot are common in the athletic population, accounting for approximately 16% of all sports injuries.¹ Trauma to the tarsometatarsal (TMT) joints is the second most common injury pattern, second only to metatarsophalangeal (MTP) joint injuries. The Lisfranc joint complex is the most commonly involved in injuries to the midfoot due to the limited static and dynamic stability of this region. Stress fractures, especially of the fifth metatarsal, continue to be a common reason for loss of time in athletes.

Misdiagnosis or maltreatment of these potentially career-ending injuries may not only prolong return to competitive play but can also lead to post-traumatic degenerative changes and pain that limit activity and quality of life in the future.²

CLINICAL DIAGNOSIS

In evaluating patients with trauma to the foot, it is essential to obtain a thorough, detailed history to direct the examiner in physical and radiographic examination. In addition, it will provide a clue to the associated degree of soft tissue injury.

Physical examination should be meticulous and systematic (Video 7.1). It is recognized that although most forefoot injuries are easily diagnosed, midfoot injuries often go undetected. Because of the high incidence of multiple fractures or fracture/dislocations in the injured foot, careful examination and palpation of points of tenderness should be performed to detect evidence of occult injury. Evaluation of range of motion of the ankle, subtalar midtarsal, and MTP joints is incorporated into every routine examination. A careful motor examination, both intrinsic and extrinsic, as well as a sensory examination are performed. Vascular examination, including Doppler studies, is essential. Radiographs are guided by the examiner's history and physical examinations. Because of the complexity of the anatomy and lack of uniform appreciation or interpretation of the foot radiographs, adjunctive studies, such as computed

tomography (CT), especially weight bearing CT, bone scan, and magnetic resonance imaging (MRI), can be of tremendous value. These also are particularly useful because of the subtle nature of many foot and ankle injuries.

Weight-bearing anteroposterior (AP), 30-degree oblique, and lateral radiographs are the first and typically most valuable imaging studies obtained for a patient with a foot injury. If the patient is too painful for a standing radiograph, an ankle block, or intra-articular local anesthetic, could be performed to obtain adequate weight-bearing radiographs. It will also allow stress x-rays to be performed (Video 7.2). A comparison weight-bearing AP radiograph of the contralateral foot should be obtained to identify subtle subluxation or translation at the Lisfranc joint complex, which appears asymmetric in the injured foot. This is particularly useful for cases with less than 2 mm of instability, as this amount of displacement is often difficult to assess on a plain radiograph.²

The oblique view, for example, is particularly useful for evaluating joints, such as the calcaneal cuboid joint and the 4-5 TMT joints, that typically are hidden or poorly examined in AP view. Specialty views, such as axial views of the heel, Broden's view of the subtalar joint, and stress views of the foot, also are helpful in certain circumstances.

Advanced imaging studies are utilized to identify or confirm a subtle injury, especially as up to 20% of Lisfranc complex injuries are missed on initial radiographs. Thin-cut CT imaging with reconstruction views will identify small fractures or possibly subluxation at the TMT joints, and may be particularly helpful if the foot can be loaded with a simulated weight-bearing study. MRI is both sensitive and specific in identifying partial and complete ligamentous injuries at the Lisfranc joint complex, but it is unnecessary for evaluation of injuries with obvious subluxation or dislocation.³ A normal appearance of the plantar Lisfranc ligament on MRI is highly suggestive of a stable midfoot whereas the best predictor for instability is a rupture or a grade-2 sprain of this ligament. MRI has demonstrated a sensitivity, specificity, and positive predictive value for detecting

injury to the plantar Lisfranc ligament of 95%, 75%, and 94%, respectively.⁴

TREATMENT

Generic goals in the treatment of fractures and dislocations of the foot are as follows:

- Avoiding stiffness and loss of mobility.
- Removing bony prominences, which may result in pressure phenomena.
- Restoring the articular surfaces.

Any fracture or dislocation of the foot or ankle that results in focal skin pressure or evidence of neurovascular compromise must be addressed immediately. Manipulation or even open reduction must be carried out to reduce the potential sequelae, including skin necrosis, neuropraxia, ischemia, and/or pressure-induced necrosis of articular surfaces, because of abnormal loading secondary to malpositioning after fracture or dislocation.

Even anatomic restoration does not guarantee optimal functional outcome, but it certainly provides the athlete with a significantly reduced risk of morbidity associated with sequelae of delayed or untreated injury. However, injuries that present without gross distortion of anatomy or imminent threat to the viability of the limb may be treated better after an appropriate “cooling down” period. This is not to say that they should be splinted and ignored, but a short period should be devoted to rest, ice, compression, and elevation (RICE) to allow the soft tissue integrity and oxygenation to reestablish itself, particularly before the clinician embarks on any invasive procedures.

The evolution of treatment of the traumatized foot and ankle of the athlete has directed more attention to aggressive intervention than to “benign neglect.” Recognition of the fact that long periods of immobilization after trauma may lead to muscular atrophy, myostatic contracture, reduction of joint mobility, associated connective tissue proliferation leading to scarring, synovial adhesion, and cartilage degeneration has prompted a more aggressive approach to foot and ankle injuries, using appropriate surgical intervention to stabilize injuries and institute earlier range of motion and weight bearing when possible. These tenets provide for the ability to institute potential prevention against previously disabling factors such as disuse osteopenia, limb atrophy, proprioceptive losses, and chronic, persistent pain. Introduction of early range of motion, physical therapy modalities, appropriate splinting, and bracing, as opposed to casting, allows for the earlier restoration of function and avoidance of complications. The static accumulation of hematoma, fluid extravasation, and resultant articular and tendinous adhesions is far less with treatment that promotes earlier rehabilitation. This type of treatment also helps to prevent disabling sequelae, such as arthrofibrosis and regional pain syndromes.

Although the realm of athletically related foot and ankle injuries is too vast to be encompassed in this chapter, the more common injury patterns encountered are addressed. Diagnostic and management controversies are discussed and elucidated for the reader. Rather than a trauma compendium, this is meant to be a guide for the treatment of frequently occurring sports

and athletic injuries to the foot and ankle for one's reference and perusal.

TARSOMETATARSAL DISLOCATIONS

The TMT joint, consisting of the bases of the five metatarsals and their articulation with the three cuneiforms and the cuboid, is named after Lisfranc, a French surgeon in the army of Napoleon, who originally described an amputation through that joint.^{5,6} These are recognized more commonly in a poly-trauma patient because of the severity,^{2,7,8} but also are increasingly recognized to occur in the athletic population.^{2,5}

Faciszewski et al.⁹ have reported on patients with “subtle” injuries to the Lisfranc joint, defined as diastasis of 2 to 5 mm between the bases of the first two metatarsals. A third of their patients' injuries were sports related. Other reports support the increasing frequency of occurrence of Lisfranc injuries in athletic events.¹⁰⁻¹⁵ Any patient diagnosed with a midfoot sprain should arouse suspicion for an undiagnosed TMT ligament disruption.

In the athletic population, the occurrence and severity vary by sport. Lisfranc injuries are reported to be the second most common athletic injury to the foot, after MTP joint injuries, presenting in 4% of football players per year, with a preponderance occurring in linemen (29.2%).¹¹ Although complete injuries resulting in diastasis of more than 5 mm are easier diagnostically and more dogmatic in treatment plan, more subtle injuries (1 to 5 mm) often are overlooked and, even when diagnosed, may lead to therapeutic dilemmas for the surgeon, as well as frustration for the injured athlete. Partial capsule tears with no diastasis, for instance, can be a compounding problem resulting in prolonged disability for the elite performer.

The TMT joint really is more of an articulating complex providing both motion and stability—much more so the latter. The osseous anatomy reveals multiple, wedge-shaped bones coalescing to form an arch in the transverse plane. The second metatarsal often has been referred to as the keystone of this Roman arch analogy, reflecting its overall importance to the integrity of the maintenance of this structure. Structural rigidity of the shape of the foot is dependent on the stability of this relationship of the midfoot bones. Because the bases of the metatarsals are wider dorsally, collapse of the arch in any plane is prevented in the face of weight-bearing load.

The second through fifth metatarsals are interconnected by a dense weave of short, broad-based ligaments and capsular ligamentous structures. These tend to be bundled together and often will move as one unit. However, there is a notorious absence of ligamentous connection between the bases of the first and second metatarsals. This is thought to account for the predominance of diastasis in this interval. Instead, there exists a dense, plantar-based, oblique ligament extending from the base of the second metatarsal to the lateral portion of the medial cuneiform—Lisfranc's ligament. This ligament anchors the lesser metatarsal complex to the medial column of the foot.

The anatomy of the Lisfranc ligament complex has been studied in detail in recent orthopedic and radiology literature, with an emphasis on clarifying the anatomy and determining

the amount of ligamentous disruption that leads to an unstable injury pattern. The dorsal ligaments are the weakest of the complex and are the first to fail in the typical indirect injury mechanism associated with subtle Lisfranc injuries. The next ligament, the C1-M2 interosseous ligament, is the largest and strongest ligament in the complex, and is referred to as “The” Lisfranc ligament. The plantar ligament attaches C1 to M2 and the third metatarsal (M3). This C1-M2/M3 ligament is considered by some to be the primary stabilizer of the Lisfranc joint.¹⁶

The tibialis anterior, with its insertion on the medial aspect of the proximal first metatarsal and the peroneus longus, which inserts into the lateral proximal first metatarsal, also contributes to the stability of the Lisfranc articulation. In certain phases of gait, these two tendons provide dynamic restraint. Plantar fascia, intrinsic musculature, and plantar TMT ligaments provide additional structural support against arch collapse and plantarward dislocation. The midfoot articulation may be divided mechanically by columns. The medial column includes the first metatarsal and medial cuneiform. The middle column consists of the second and third metatarsals, as well as the middle and lateral cuneiforms. The lateral column is formed with the fourth and fifth metatarsals, along with the cuboid bone. This column provides the greatest motion throughout the TMT joint.

Vascular structures in this region deserve mention because of their proximity to the area of potential injury. The dorsalis pedis artery and the plantar arterial arch are structures at risk, particularly when the dorsalis pedis dives down between the bases of first and second metatarsals. Disruptions here, especially with a tethered vessel, can result in kinking, vasospasm, and, ultimately, ischemia. Lisfranc dislocation derives its name, in fact, as previously stated, from the Napoleonic surgeon who so definitely amputated cavalrymen after midfoot injuries resulting in vascular catastrophes.^{2,5,7,8} Although less common compared with the high-energy version of this injury, anecdotal reports of associated vascular injuries abound and should be sought for fear of missing an ischemic sequela.

Patterns of injury to the TMT joint have been described as a result of both direct trauma to the foot and indirect violence. The majority of nonathletic traumatic midfoot injuries can occur as a result of significant direct force, usually applied to a foot in plantarflexion or abduction, and typically will accompany high-velocity or high-energy trauma, such as motor vehicle accidents or falls from heights.^{2,7,16-18} These can result in significant soft tissue compromise, neurovascular injury, and compartment syndrome.⁸

Indirect injury is more relevant to this discussion. Athletes may sustain direct violence to the foot as the result of an awkward collision or in the melee of a collision in certain sports. However, more commonly the athlete is injured because of low-velocity, indirect energy imparted to the foot. Most will describe some sort of axial longitudinal force while the foot is plantarflexed and, often, slightly rotated.¹² Two specific patterns have been described. Simple lateral dislocations result from eversion of the hind foot on a fixed plantarflexed foot, as may be seen in ballet dancers *en pointe*.^{16,19-22} Alternatively, supination or inversion of the hindfoot on a fixed plantarflexed forefoot will result in a more dissociative pattern of injury because the

medial column is disrupted, followed by the lateral dislocation of the lesser metatarsal and associated tarsal cuneiforms.²³ A third type of injury occurs when the fixed plantarflexed foot is forced into extreme equinus as a result of being struck from behind.^{9,13} This is more common in turf sports such as football. Elements of torque, rotation, and compression are all present and cumulatively lead to a dorsal capsule ligamentous disruption.

Many classification systems have been proposed to describe the multitude of injury patterns that may occur.^{2,7,10,24,25} Because of the tremendous variation, no one system has been universally accepted. These classification systems usually apply to high-energy injuries and are based on segmental patterns of metatarsal-tarsal bone displacement.

Recently a useful classification has been proposed specifically for the athletic midfoot injury, including undisplaced sprains, and is based on clinical findings, weight bearing, x-rays, and bone scan results (Fig. 7.1).¹² Stage I patients were found to have pain at the midfoot and were unable to play sports but had no radiographically visible changes. Bone scan results did demonstrate increased uptake in the area of Lisfranc joint. Pathoanatomy is thought to include dorsal capsular tear without elongation of Lisfranc's ligament. Stage II is described as clinical findings similar to those in stage I, but with diastasis of 1 to 5 mm between the bases of the medial two metatarsals present on plain AP radiographs. Most important, no loss of longitudinal arch was noted on weight-bearing lateral x-rays. Pathoanatomy here differs from stage I in that the Lisfranc ligament is elongated, but the plantar structures remain stable and prevent arch collapse. Stage III was defined as diastasis greater than 5 mm and loss of lateral arch height, defined by loss of space between the fifth metatarsal and the medial cuneiform on lateral radiograph.^{12,25} All capsuloligamentous structures are thought to be injured in stage III. Other forms of injury, such as gross disruption with fracture and/or dislocation, were defined by these authors in the method originally proposed by Myerson,²⁶ which was based on segmental instability (Fig. 7.2). The advantage of such a classification is that treatment may be predicated on the level of injury.

Up to one in five Lisfranc injuries are missed or improperly diagnosed on initial screening, whether it be in the emergency department or at practitioner's office. This often can be ascribed to the presentation of these injuries as part of a polytrauma, with other, more severe and more obvious injuries demanding the bulk of attention.^{2,3,5}

In the athlete, however, it is the subtle or complete absence of radiographic diastasis that may occur that confounds the examiner.^{9,10,12-14,27} A high index of suspicion must be maintained for athletes presenting with midfoot pain after athletic contact or activity, even without radiographic evidence of injury. Consideration should be given to stress radiographs as a means of furthering diagnostic abilities.

Physical examination is especially important with subtle injury. Gross distortion of the bony architecture of the foot is readily identified. Clinical and radiographic findings of fractures and dislocations are relatively simple to determine. The patient presenting with no overt disruption or equivocal radiographic divergence becomes a diagnostic dilemma.

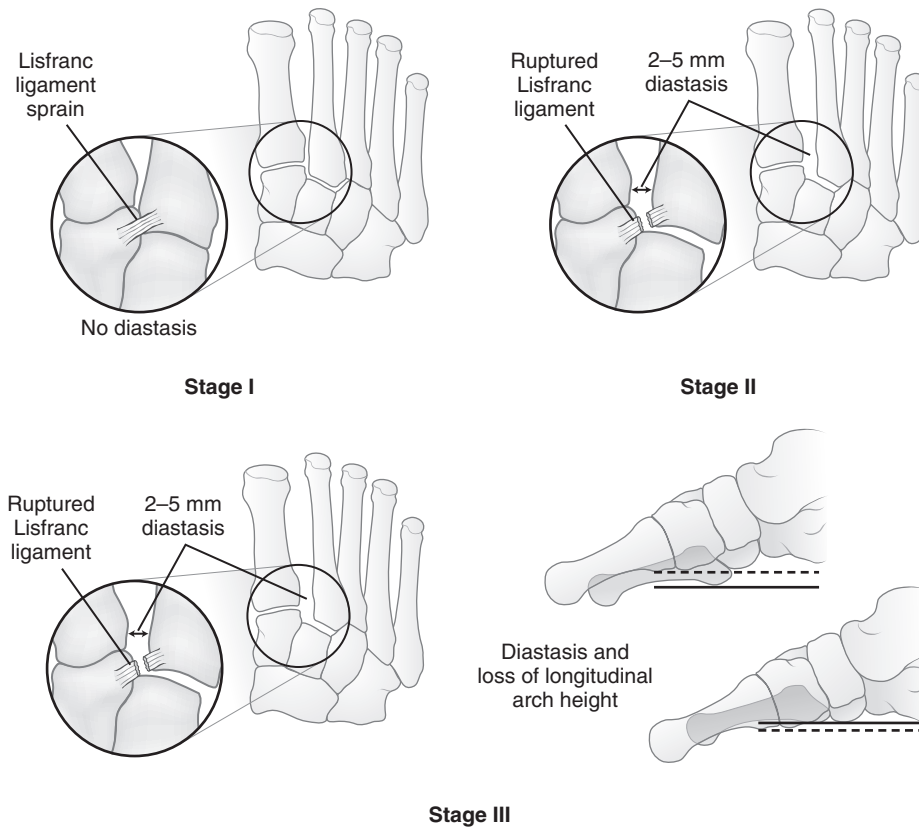


Fig. 7.1 Nunley classification of athletic Lisfranc injuries. (From Nunley JA, Vertullo CJ: *Am J Sports Med*, 2002;30(6):872, Fig. 7.1.)

Examination typically demonstrates tenderness at the midfoot that is worsened by provocative maneuvers such as pronation or abduction of the foot. Swelling is often significant, and ecchymosis is variably present. Neurovascular injuries are unusual in the lower-energy traumas, but the possibility of impending compartment syndrome always should be considered, because there often is tremendous edema accompanying these injuries.

Classic radiograph findings and markers have been well established. A minimum of three radiographic views of the foot (AP, lateral, and oblique) should be obtained. Assessment in suspicious injuries should be made of all of the following relationships:

1. Diastasis of metatarsals one and two.
2. Cuneiform diastasis, especially medial and middle.
3. Widening between the second and third metatarsals.
4. Widening between middle and lateral cuneiforms.
5. Small fracture, "fleck sign" at the medial base of the second metatarsal or medial cuneiform, representing an avulsion of Lisfranc's ligament.
6. Horizontal plane malalignment of metatarsals on lateral x-ray.
7. Relationship of medial border of the second metatarsal should be parallel to the medial edge of the middle cuneiform.
8. Relationship of the medial fourth metatarsal should be parallel to the medial edge of the cuboid.
9. General loss of parallelism of metatarsal bases with respect to one another.

10. A small compression fracture at the lateral edge of the cuboid.^{10,24-30}

Even after perusing the radiographs with these parameters in mind, the clinician may find it difficult to make a diagnosis. Weight-bearing, contralateral radiographs often are helpful in discerning any asymmetry.

In more subtle and problematic cases, multiple advanced imaging studies have been suggested, including CT, MRI, static stress radiographs, and stress fluoroscopy under anesthesia.^{2,5,10,29-33} However, the best and most reliable studies seem to be a set of standing radiographs (Fig. 7.3, A through E) and bone scan, if necessary, in the completely undisplaced metatarsal that manifests persistent pain.^{12,13} There are two advantages to weight-bearing radiographs. First, the dynamic nature of the injury can be determined in a more appropriate physiologic and mechanical state, thus determining the need for treatment. Second, prognostic value exists in determining the presence of collapse or instability.^{9,14,15}

Principles of treatment of Lisfranc injuries are universal and include providing an anatomic reduction in stabilization. Care must be taken to observe and manage the soft tissue and neurovascular consequences, as well.

Debate still exists as to how much diastasis is acceptable in the injured athlete. The literature is heavily weighted toward high-energy trauma management, and little has been proposed regarding management of the athletic midfoot sprain. Most recent literature suggests that residual diastasis may result in a poor outcome, such as persistent pain, deformity,

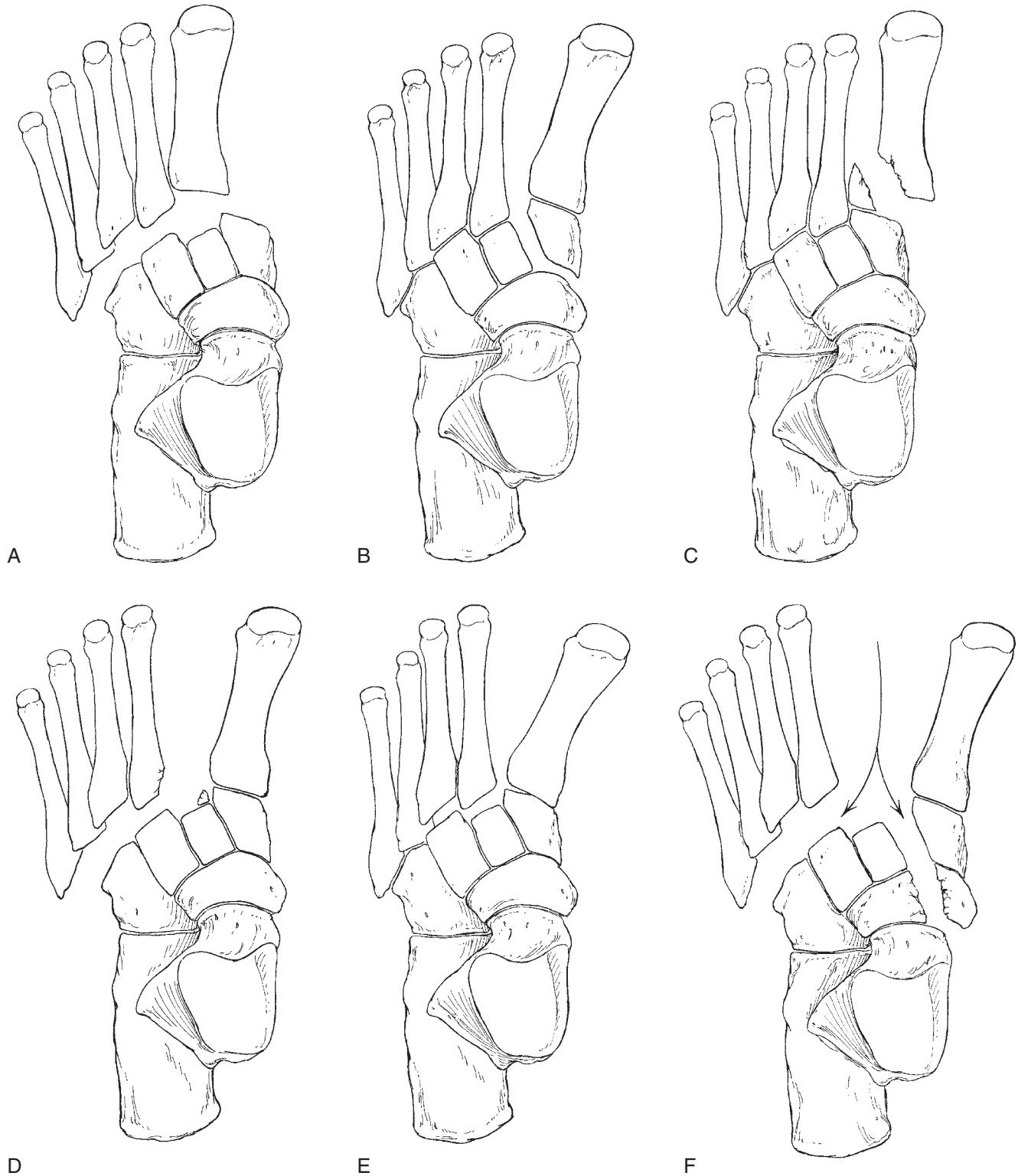


Fig. 7.2 Myerson classification of Lisfranc injuries. (From Myerson MS: *Foot and ankle disorders*, St. Louis, MO: Mosby; 1999.)

and arthrosis.^{10,12-18,26,32-35} Nonanatomic reductions have been shown to be inferior with respect to outcome and the need for secondary procedures, such as revision repairs or fusions.

Athletic injuries are sparsely documented, but the evidence that is available seems to support the conclusion that injuries resulting in diastasis will lead to poor outcomes. Curtis et al.¹⁰

reviewed 19 athletes with varying degrees of TMT injury, citing poor functional results despite “relatively nondisplaced injuries” in patients with delays in diagnosis and those not treated adequately, with three failing to return to sport. Lewis in one paper and DeOrio in another showed excellent results with treatment of Lisfranc injuries in athletic populations. A high level of return to



Fig. 7.3 (A) Radiograph of a 34-year-old professional waterski jumper with acute midfoot pain after a fall. There is a suggestion of subtle intermetatarsal diastasis. (B through D) Various advanced imaging studies confirm the Lisfranc ligament disruption by avulsion of the base of the second metatarsal. (B) Bone scan shows increased uptake about the midfoot. (C) Computed tomography demonstrates the avulsed fragment. (D) Magnetic resonance imaging reveals edema in the region of the ligament with suggestion of bony injury. (E) Plain anteroposterior weight-bearing x-rays of the injured and comparative contralateral side clearly disclose the diastasis.



Fig. 7.4 Incision placement for exposure to the diastasis of Lisfranc's joint.

play was achieved.^{14,18} Nunley and Vertullo¹² showed that 14 of 15 patients had good results when treated within the algorithm based on a classification, they proposed that guided treatment on the basis of displacement. Patients were assessed on the basis of plain x-rays and bone-scan-documented injury. Only completely non-displaced injuries (seven patients) were treated nonoperatively. All others were treated by open reduction with internal fixation. The only patient with residual pain was one treated by open reduction and internal fixation (ORIF) after 10 months of failed conservative treatment. Return to sport in the operative group averaged 14.4 weeks, which was comparable to nonoperative results.

Only one study demonstrates reasonable results with nonanatomic reductions. Shapiro et al.¹³ reported on nine athletes with diastasis between 2 to 5 mm. Eight elected for nonoperative treatment and returned to sport within 3 months, with good results reported in an average of 33 months after the injury.

On the basis of these reports and personal experience, my recommendation is for operative treatment in all but nondisplaced injuries of the TMT joint. Although percutaneous techniques have been proposed, an open approach is more reliable and eliminates the possibility of retained or interposed tissue, as well as allowing direct visualization of the joint for an anatomic reduction (Fig. 7.4). Closed or percutaneous techniques using the large Weber reduction clamp carry the risk of malreduction, especially in a horizontal plane, even in the face of an anatomic appearing anterior/posterior image (Fig. 7.5).

Open treatment affords the surgeon the opportunity to extricate any incarcerated bony fragments or soft tissue that may have been interposed, including the Lisfranc's ligament itself or, in high-energy injuries, the tibialis anterior tendon. Anatomic restoration of the arch is achieved and verified, as well as providing direct visualization for hardware placement.

Screw fixation is preferable because K-wire fixation is tenuous, at best, and not as reliable in maintaining an anatomic



Fig. 7.5 Percutaneous reduction technique with a large Weber clamp. Surgeons must be aware of the tendency for dorsal plane malalignment as a result of overtightening or improper clamp placement. Lateral fluoroscopy should always be employed to verify anatomic reduction.

reduction. In addition, especially in unstable injuries, the motion at the joint surfaces will induce pin loosening and migration with predictable loss of stability, thus requiring concurrent cast immobilization, which prevents early rehabilitation. Conversely, screw fixation is reliable and allows for early mobilization of the foot and ankle, as well as edema control after wound healing has been achieved.

Screw configuration is dependent on injury pattern and extent of ligamentous disruption. My preference is to use fully threaded, 4.0-mm or larger screws. Partially threaded screws are acceptable, but because this is a “position screw” to maintain reduction, the surgeon must guard against the tendency to compress across the TMT joints. Typically, the first screw is placed on the orientation of the disrupted Lisfranc's ligament, that is, from medial cuneiform to second metatarsal base. Additional screws are placed as needed (Fig. 7.6, A through F). Should the injury extend through the medial and middle cuneiforms, an intercuneiform screw should be placed first.

The patient is kept non-weight bearing for 6 to 8 weeks. Early motion and therapy modalities such as muscle stimulation can begin as soon as soft tissue healing allows. Partial weight bearing in a boot begins at 6 to 8 weeks and is advanced until 12 weeks. Screws are maintained for no fewer than 16 weeks and often, but not routinely, are removed. The athlete is returned to athletic activity with a molded, semirigid insole and a semirigid extended steel shank device.

There is a movement away from transarticular screw fixation for ORIF of Lisfranc injuries. The premise is that if you want to maintain/preserve the joints, it is best not to violate a substantial cross-section of the articular surface with a screw. A secondary issue is that if the screws break in the joint, it could significantly compromise the joint.



Fig. 7.6 (A through D) Running back with Lisfranc injury that was treated with a Bridge plate and home run screw. (E and F) The hardware was removed at 4 months and examination showed the joints stable.

Bridge plating has become increasingly popular, as it does not violate the joint, even if the screw breaks, and is easy to remove. Hu and coworkers showed in their study that the bridge plate patients did better than screw fixation.^{2,29}

Frank disruptions and intra-articular fractures are treated in the way that trauma guidelines dictate and are managed ORIF or primary arthrodesis.^{14,18}

Postoperative protocols are similar to those described previously, but usually require larger periods of rehabilitation, and return to activity is less predictable in these patients.

TARSAL BONE FRACTURES

Anatomic variants of Lisfranc's injuries do exist. There have been reports citing evidence of bipartite cuneiforms and anatomic variations in anatomy throughout the midfoot bones (Fig. 7.7, A through F). Should these be encountered, pursue and treat aggressively, with the same guidelines as those for the previously described injuries.³⁶⁻³⁸

Fractures or dislocations exclusive to the cuneiforms or cuboid area are unusual. These often are present in conjunction with a TMT joint injury, in which the force of the injury has disrupted further proximally through the navicula, cuneiform, or talonavicular joints, or even through the body of the cuboid. Although rare, these injuries have been identified.^{39,40} Because cuneiform fractures and dislocations often occur as part of a midfoot dislocation, treatment principles should follow those of the injured TMT joint.

Isolated cuboid injuries most often present as insignificant "chip" fractures along the lateral side. Typically, these occur as a result of an inversion injury and often are seen secondarily after the patient has been diagnosed with "sprain." Treatment requires supportive immobilization in either a walking cast or hard-soled shoe for approximately 4 weeks or until symptoms allow resumption of activity. A rigid orthosis may allow earlier return to sport. Fracture instability is not usually a concern.

Compressive cuboid injuries can occur with a sudden abduction force. So-called nutcracker injuries are far more severe. Again, this is considered a variant of the mechanism for Lisfranc injuries, and the same principles are applied. Early anatomic reduction is necessary (Fig. 7.8, A through F). Manipulation alone is often unsuccessful in restoring the length of the lateral column. Open treatment frequently is required. Placing a small plate to span the collapsed intercalary segment is necessary on occasion. If there is poor-quality bone fixation in the subarticular cuboid, a spanning plate to the distal calcaneus represents a good alternative. For severe comminution, the author prefers structural tricortical graft to reestablish the length. This may be interposed between subchondral bone proximally and distally, because the articular surfaces often are not severely comminuted. If necessary, fixation can be applied as previously stated, or a spanning external fixator from distal calcaneus to proximal metatarsals may be used to distract the lateral column.

FRACTURES OF THE FIFTH METATARSAL

Fractures of the base of the fifth metatarsal are the most common metatarsal fracture. However, there are many misconceptions

regarding the description, the understanding, and thus the treatment of these injuries throughout the literature. The classic Jones fracture was named after Sir Robert Jones,⁴¹ who originally described the fracture in his own foot in 1902. He sustained the fracture "Whilst dancing, I trod on the outer side of my foot, my heel at the moment being off the ground. Something gave way midway down my foot...the fifth metatarsal was found fractured about 3/4 inch from its base." Jones originally described the fracture of the metaphyseal diaphyseal junction without extension distal to the anterior metatarsal (4-5 intermetatarsal) junction. Currently, a Jones fracture is recognized as any fracture involving the fifth metatarsal metaphyseal-diaphyseal junction. This fracture often is confused with, although less commonly encountered than, its cohort, the avulsion of the tuberosity encountered more proximally. The significance of the true Jones fracture is that it can develop delayed or non-union. Zelko et al.,⁴² Kavanaugh et al.,⁴³ and DeLee⁴⁴ have reported difficulty treating the fractures of this region in which diagnoses initially were missed or that, in reality, were stress fractures.

Stewart⁴⁵ originally introduced a classification to help clarify fractures in this region. Type I fractures are at the junction of the base and shaft of the metatarsal. Subgroups include noncomminuted (IA) and comminuted (IB) variants. Type II fractures involve only the styloid process. Again, these are subdivided into extra-articular (IIA) and intra-articular (IIB). Stewart established a treatment plan that is based on his classification system.

Zelko et al.⁴² tried to define fractures on the basis of clinical history and initial radiographic findings. Group 1 patients reported an acute injury with no previous symptoms. Radiographs demonstrated what appeared to be acute fracture line and no evidence of any chronic change, defined as periosteal reaction or intramedullary sclerosis. Group 2 demonstrated an acute injury but also reported a prodrome of mild lateral foot pain. Radiographs in these patients evidenced a clear fracture pattern. However, there also was demonstration of some periosteal reaction. Group 3 patients were categorized as a reinjury after one or more previous injuries. Radiographs of these patients demonstrate lucent fracture line, periosteal reaction, and intramedullary sclerosis, and this group presented with chronic pain or multiple recurrent injuries with sclerotic margins bordering a lucent fracture line.

DeLee and colleagues⁴⁴ attempted to combine classifications and divided these into multiple fracture types. Type I fractures are those at the junction of the base of the shaft and the base and are subcategorized into type A for nondisplaced and type B for comminuted fractures in this area. Type II fractures occurred again at the junction of the shaft and the base but carried clinical and radiographic evidence of prior injury. To fall into this category, patients had to report prior lateral foot pain and/or an established radiographic periosteal stress reaction or frank fracture line. Type III fractures included those of the styloid process or tuberosity and again were classified into subcategories A, nonarticular, and B, articular.

The recommended current classification includes a combination of all the classifications discussed and divides the metatarsal injuries into classification that correlates to zones of

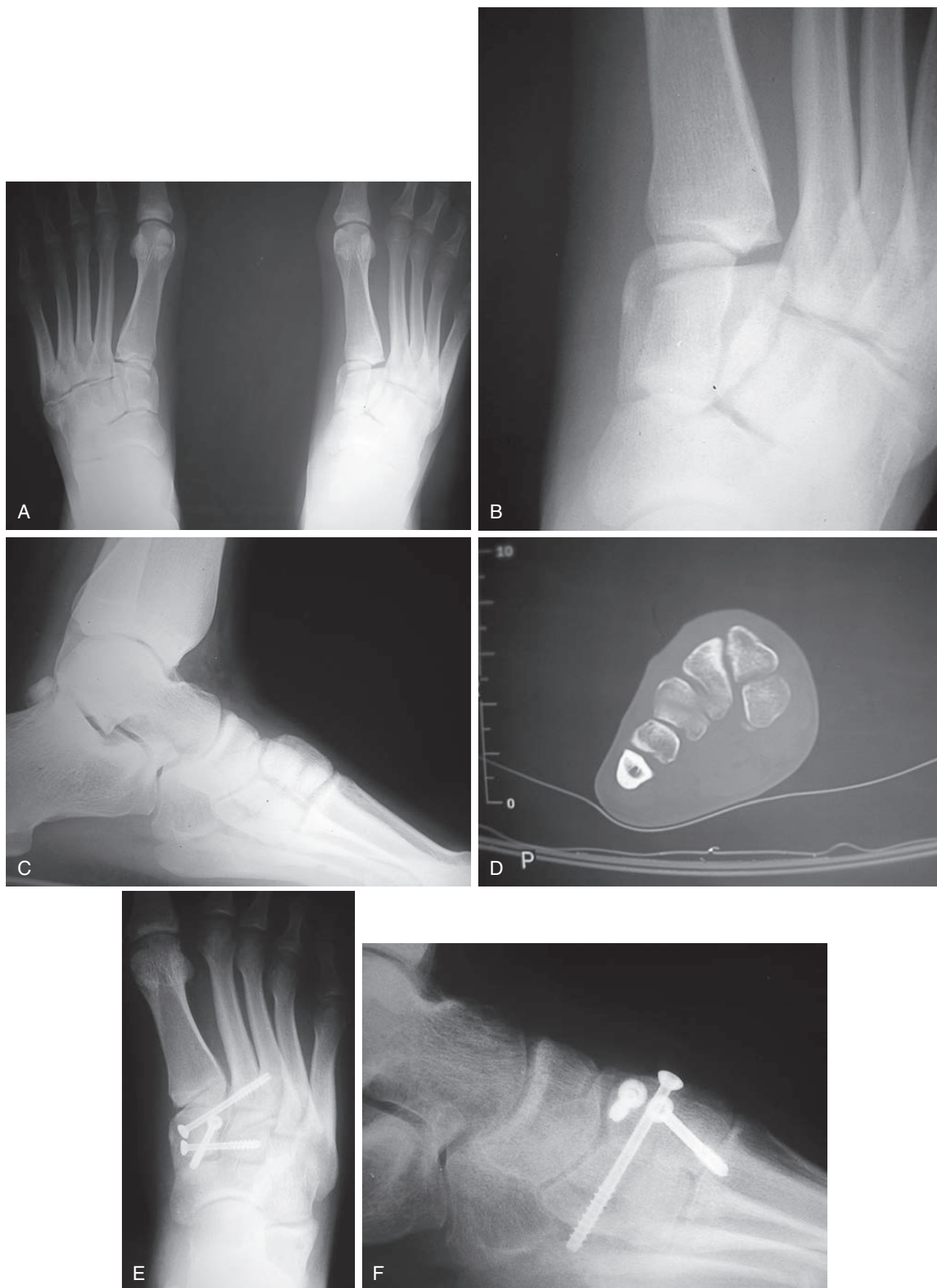


Fig. 7.7 (A) Weight-bearing anteroposterior radiograph, with comparison, of a high school quarterback with acute midfoot injury. (B) Close-up suggests unusual arrangement in the area of medial cuneiform. (C) Lateral radiograph demonstrates separation of dorsal and plantar halves of medial cuneiform. (D) Computed tomography confirms bipartite tarsal bone. (E and F) Open repair requires attention to both the separated bipartite cuneiform with removal of synchondrosis and closure of the intermetatarsal diastasis, as well.

vascular anatomy (Fig. 7.9). Currently, preferred classification uses three separate zones. Zone 1, or the most proximal zone, includes the cancellous fifth metatarsal, the so-called tuberosity fragment. It includes the insertion of the peroneus brevis tendon and calcaneometatarsal ligamentous branch of the plantar fascia. Fractures in this zone typically extend into the fifth metatarsal cuboid joint but may be extra-articular. Zone 2 injuries involve the metaphyseal-diaphyseal junction. This encompasses the articulation of the proximal fourth and fifth metatarsals. The ligaments holding the fourth and fifth metatarsals together

proximally are secure both dorsally and plantarly and provide tremendous stability. Finally, zone 3 injuries are fractures of the fifth metatarsal shaft. This zone begins just distal to the fourth and fifth intermetatarsal ligaments and extends distally into the tubular portion of the diaphysis approximately 1.5 to 2.0 cm. Most current management protocols use some form of zone concept in classifying and reporting fractures. Therefore, the bulk of the discussion regarding treatment will reflect this trend and be focused on management of fractures by type and location.⁴⁶⁻⁴⁸



Fig. 7.8 (A and B) Shows a severely comminuted fracture dislocation of the talonavicular (TN), calcaneo-cuboid, and cuboid/metatarsal joints. Also fracture dislocations of the second, third, and fourth metatarsophalangeal joints. This patient was involved in a high-speed bicycle crash. (C and D) shows initial open reduction and internal fixation of the fractures and a primary talonavicular arthrodesis. (E and F) Four months after with the fractures and TN arthrodesis healed. The TN arthrodesis did not limit her as a cyclist, and she was able to return to her sport.

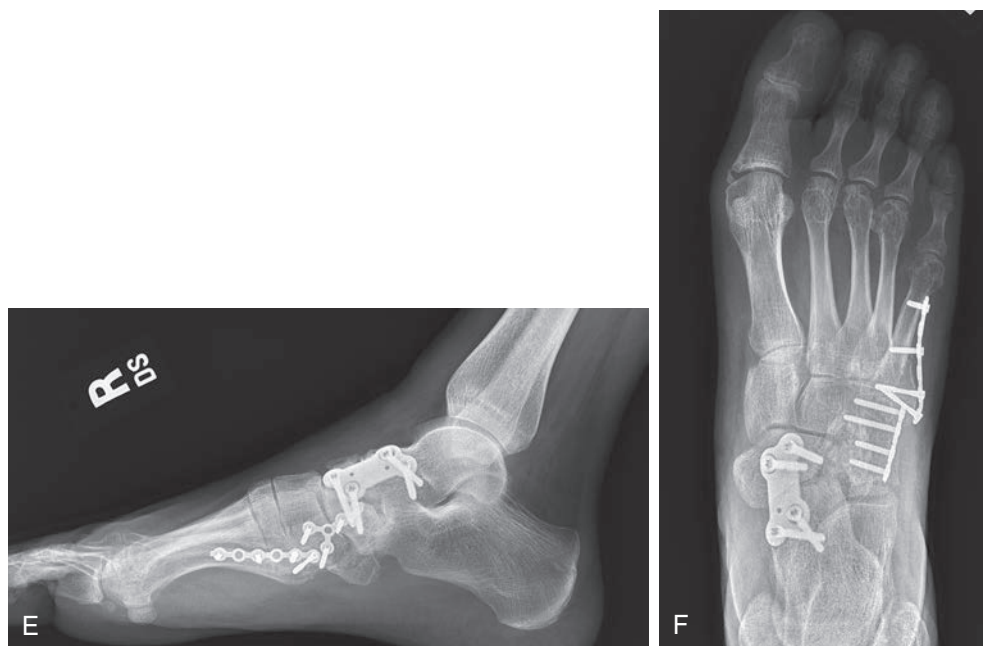


Fig. 7.8, cont'd

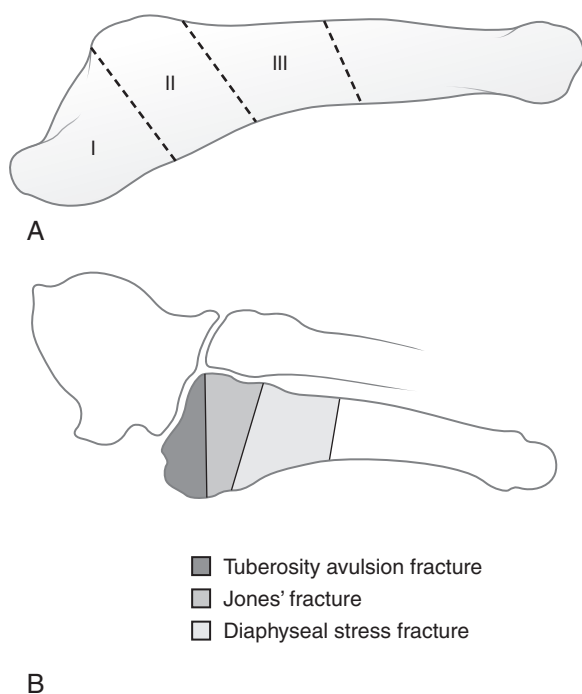


Fig. 7.9 Fracture zone classification at the base of the fifth metatarsal. (From Lawrence SJ, Botte M: *Foot Ankle* 1993;14:360.)

The fifth metatarsal itself has been subdivided into main segments, including the head, neck, shaft, base, and tuberosity or styloid process. The metaphyseal portion of the bone tapers into a tubular diaphyseal segment. The size and the shape of this bone vary somewhat but typically demonstrate that a larger, wider, more triangular proximal portion becomes a fairly narrow tubular structure that has a slightly lateral curve as it traverses distally. The radius of curvature as the bone proceeds distally is highly variable and can lead to tremendous distortions in the shape and stress applied to the distal end of this structure.⁴⁶

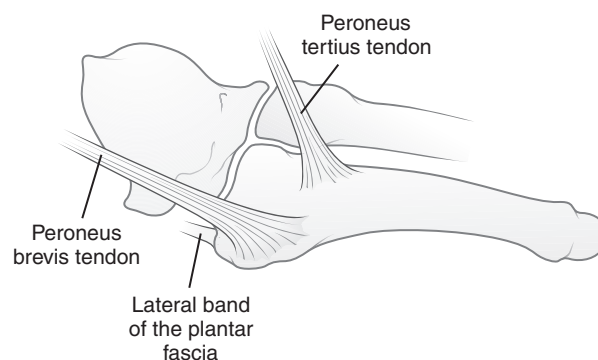


Fig. 7.10 Anatomy of tendon attachments at the base of the fifth metatarsal. (From Lawrence SJ, Botte M: *Foot Ankle* 1993;14:360.)

The proximal end of the fifth metatarsal not only articulates with the cuboid at the TMT joint but also has an intermetatarsal articulation with the base of the fourth metatarsal. This is a true joint. The bases of the fourth and fifth metatarsals are bound closely to the cuboid by dense, ligamentous structures on every side. The stability of the TMT complex is provided by capsular ligamentous structures, the dorsal and plantar cubometatarsal ligaments, the lateral band of the plantar aponeurosis, and the broad insertion of the peroneus brevis tendon (Fig. 7.10). It is believed that these capsular ligamentous structures contribute greatly to the genesis of a true Jones fracture,^{3,43} because the proximal portion of the fifth metatarsal and its articulation with the cuboid is held fast while torsional forces produce stress that is relieved through the fracture line just distal to these structures, approximately 0.5 cm distal to the insertion of the peroneus brevis and just distal to the joint between the fifth and fourth metatarsals. The base of the fifth metatarsal proceeds laterally and inferiorly beneath the inferior edge of the cuboid on the lateral radiograph. There is a tremendous variability in size and shape of this prominence, accounting for its variable susceptibility to injury.

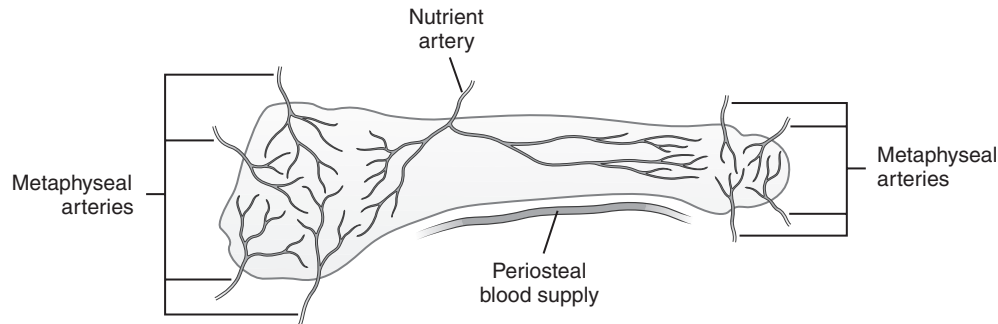


Fig. 7.11 Vascularity of the fifth metatarsal. (From Smith J, Arnoczky SP, Hersh A: *Foot Ankle* 1992;13:144.)

The vascular anatomy in this region also is relatively important (Fig. 7.11). This has been thought to be a fairly tenuous vascular supply, particularly at the proximal diaphysis. The arterial plexus at this level has been well established by Shereff et al.⁴⁷ and Smith et al.,⁴⁸ demonstrating only a small nutrient vessel in the so-called watershed area. This is unique contradistinction to the fairly abundant blood supply more proximal to this watershed area.

Direct and indirect mechanisms have been implicated in the genesis of the fifth metatarsal fracture. Certainly, the prominence of the tuberosity makes it particularly at risk for a more direct mechanism of injury when discussing this version of the fracture.⁴⁶ Jones himself alluded to the indirect nature of injuries, describing a “cross-breaking strain directed anteriorly to the metatarsal base and caused by body pressure on an inverted foot while the heel is raised.”⁴¹ Presumably, he is describing the commonly accepted foot in fixed equinus sustaining rotatory and/or tensile forces overcoming the thinning cortical bone in the proximal metaphyseal-diaphyseal junction.

Fractures of the tuberosity occurring indirectly are more common because of the number of structures that attach to the prominence.⁴⁶ These structures have been identified previously. The importance of the pull of the peroneus brevis has been emphasized in the creation of a separation stress that forces the proximal fragment of the metatarsal away from the shaft. Because of the strong peroneus brevis contraction in stance phase, the tendon already is contracted when an inversion stress is applied to a weight-bearing, plantarflexed foot. This tendon holds fast while the force causes the shaft to be pulled away from it. Avulsion of the base away from the shaft is the result.³ Kavanaugh et al.⁴³ used high-speed cinematography and force platform analysis in an attempt to recreate the position of the foot at the time of the index injury. Conclusions of this study suggested either an axial or mediolateral force or a combination of these acting on the fixed base of the fifth metatarsal. This would bring the patient up on the metatarsal heads, concentrating the axial and mediolateral forces on the lateral metatarsal. It was postulated that failure to invert the foot would produce a tremendous axial and mediolateral ground force culminating in fracture.

Other factors also have been implicated in the genesis of the injury here, including repetitive use, such as prolonged running or jumping activities; vascular contribution, particularly at the avascular or watershed zone; and certain morphologies of foot

shape. Individuals with more cavus foot alignment have been shown to be more likely to develop this injury pattern because of the increased rigidity of the foot, as well as the propensity to have a stress transfer to the lateral foot.^{3,42-44,47-49} Individuals with planovalgus foot also have been suggested to be predisposed to this injury because of increased loads forced along the lateral border of the foot during the latter part of stance, phase, and gait. These relationships have not been demonstrated in any formal mechanical studies.

Clinical diagnosis of the Jones fracture is dependent on making an appropriate diagnosis and localizing the specific type of injury with respect to zone as well as acuity. History may be vague but typically involves an aching sensation on the lateral aspect of the foot related to some sort of push-off or inversion-type injury. Prodromal symptoms may be reported for up to several weeks before any evidence of the actual documented injury suggestive of a prefracture state or impending fracture.^{42,44,46}

Physical examination findings are fairly reproducible and include an improved tenderness, specifically over the base of the fifth metatarsal. Ecchymosis and swelling are present to variable degrees and, again, depend on the acuity of the injury. There is typically an accentuation of pain by inversion of the foot. However, there is little motion at the fracture site, and therefore no crepitus or palpable mobility of the fracture site on manipulation.

Radiographs often will confirm the diagnosis, although in some instances some fractures may present as occult or incomplete. Careful radiographic assessment is important to determine the presence of a fracture line because this may be particularly subtle. If the diagnosis is in question, studies such as MRI or bone scintigraphy tend to be particularly helpful.⁴³⁻⁴⁵

Diagnosis of fractures can be especially confounding in the adolescent athlete because secondary centers of ossification at the base of the fifth metatarsal are present and sometimes are confused with acute fractures. The ossification center typically occurs between 8 and 12 years of age and usually is united by 12 years in girls and by 15 years in boys. A secondary ossification center occurs in approximately one-fourth of all children.⁴⁶

Distinction between these secondary centers of ossification and acute fracture is relatively straightforward. Distinguishing characteristics include the orientation of the apophyseal line, which reproducibly traverses the tubercle parallel to the long

axis of the shaft. Additionally, the apophysis occurs lateral to and does not extend into the TMT joint.⁴⁶ Ossification centers also tend to have smooth, regular edges, as opposed to a more irregular appearance of fracture.

Two other ossicles often will occur in this region. The os peroneum is present in approximately 10% to 15% of all radiographs. The os vesalianum is variably present as well. Again, a smooth, sclerotic, appositional surface often is present and differentiates this from fracture. These ossicles, which are independent, sesamoidal-type bones, should be distinguished easily from acute fracture situations.

Treatment is injury specific and fracture type dependent. Treatments vary and range from weight bearing in a protective shoe as soon as pain allows to various forms of ORIF and, sometimes, bone grafting. The literature is replete with information to support just about any stance one may want to take. It is crucial that a clear understanding of the injury pattern, the outcomes of nonoperative versus operative treatment, and the potential complications be understood by the surgeon before embarking on a treatment plan.^{50,51} Various forms of surgical treatment have been described and are addressed independently by procedure.

First, the technique of medullary curettage and inlay bone grafting has been well established.^{49,52} At present this method is essentially of historical value and is seldom used. The base of the fifth is approached via a curvilinear dorsolateral incision. The fracture site is exposed subperiosteally. A rectangular section of bone measuring 0.7×2.0 cm centered over the fracture is outlined by four drill holes and removed with a sharp osteotome. The medullary canal is curetted free of all sclerotic bone, and the continuity of this cavity is reestablished. The original description includes a tibial corticocancellous graft that is fashioned and replaced into the fracture defect. No fixation typically is applied because the graft often is slightly oversized, yielding a tight fit. The postoperative protocol includes non-weight-bearing cast applied for 6 weeks, with gradual resumption of activities determined on the basis of pain tolerance after that.

Percutaneous intramedullary screw fixation also has been described.^{3,5,42,44,50,52-58} This is performed through a small incision initially at the base of the fifth metatarsal between the peroneus brevis tendon and the lateral band of the plantar fascia. The interval is developed, and a guidewire for a cannulated screw is inserted under fluoroscopic guidance. The key point to remember about placement of the screw is that, on the basis of the anatomy, the wire should be initiated “high and inside.” This suggests that the guidewire should be started on the dorsal and medial aspect of the bone just inside and superior to the edge of the tuberosity. Once the guidewire is positioned appropriately and verified under fluoroscopic guidance, a canal is drilled and an appropriate-length screw is placed. Choices for the size of the screw typically are based on the size of the bone, and it is well accepted that the largest screw that the canal can accommodate should be placed. One technique tip is to overdrill using the cannulated guidewire system and then to remove the guidewire and place a solid screw to provide greater tensile strength to the bone. It is crucial to avoid fracturing the metatarsal, and thus maintenance of the intramedullary position is of utmost importance. No cortex should be violated on passage of the screw. Postoperatively,

the patient is placed in a splint for approximately 1 week, and a short leg, non-weight-bearing cast is applied for an additional 2 to 3 weeks. At 3 weeks, stationary bicycling, swimming, and stair climbing are allowed in a protective boot, with weight bearing progressed as tolerated, depending on pain. Running is encouraged only when evidence of significant fracture healing is present radiographically, and typically this takes 5 to 7 weeks. Return to sports-specific activity is prohibited until the patient can run and cut painlessly. Caveats with respect to this procedure involve injury to the sural nerve, which is as close as 2 to 3 mm from the position of the screw head.⁵⁴

Lastly, a combination of the previous two procedures mentioned has been applied.⁵ The technique for screw placement is as previously stated. However, this is done with a larger incision, and access is gained through the canal before placement of the screw. Bone graft should be placed dorsally, medially, and plantarly before insertion of the screw. Once bone graft is placed, the screw is inserted and the wound is closed. An alternative to this method is a so-called strain-relieving cancellous bone graft, which can be placed in similar fashion but specifically in a dorsomedial trough spanning the fracture site. Once the screw has been placed, additional bone graft can be packed in and around the fracture site. Return to activity is similar to that as previously stated for screw fixation alone.

As previously stated, literature abounds regarding multiple forms of fractures. It is somewhat confusing because, in some of the earlier literature, either specific type of fracture is not specified or uniform treatment is applied to all fracture types. An attempt will be made to dissect the literature and apply it in a relatively simple yet appropriate fashion.^{5,42,49,50,51,54-59}

Extra-articular tuberosity fractures typically require no more than supportive therapy and weight bearing as tolerated as soon as the patient is able to manage pain and swelling appropriately. Multiple forms of “benign neglect” have been described, including suggestions for compressive dressings, adhesive taping, supportive footwear with padding around the prominence, and even short leg casting.^{45,50,51} There has been no consensus on the type of protective device necessary. However, it has been reported that even short leg walking casts probably are overprotective in the management of this fracture.^{3,29} The pain usually has subsided significantly by the second week to allow reasonably functional walking and transition into a more sports-specific shoe and resumption of activity, again, as pain would allow. It also is important to note that radiographic union may not be present for a minimum of 4 to 6 weeks, and often longer. However, this should not preclude an athlete’s returning to sport should symptoms subside appropriately. It also has been suggested that, on occasion, the fracture will heal with fibrous union, and that typically this also is not symptomatic and, again, will allow the athlete to return to activity appropriately.⁵⁰

Indications for surgery in this region are reserved for those patients that have either significantly displaced tuberosity fractures or intra-articular involvement with displacement.^{45,60} Open reduction need not require an intramedullary screw as previously described, but only a small interfragmentary screw. Recognition and treatment after delayed presentation may require that excision of the fragment be performed, as opposed to standard open

reduction. The author's experience with this fracture, even with intra-articular, nondisplaced varieties, suggests that the non-operative treatment is and continues to be the standard of care. However, if there is any question regarding management, a more aggressive approach should be instituted. Poor results with tuberosity fractures are largely anecdotal⁶⁰ and may be a result of a painful fibrous union, because lack of bony consolidation can approach 19%.⁶¹ Other factors involved in poor outcomes would be articular incongruity or sural nerve entrapment in the fracture after healing ensues, necessitating surgical management.

Treatment of the true acute Jones fracture has evolved. Initially, universal treatment was considered to be the application of short-leg walking cast.^{3,51} However, even in reports advocating this form of treatment, there were found to be non-unions occurring that

required subsequent surgical treatment. Review of the literature demonstrates a rate of delayed union as high as 38% and a definite non-union rate of 14% with nonoperative treatment of these fractures.⁵¹ It was additionally noted by Zelko et al. that, even after an extended period of non-weight-bearing, short leg casting for a period of 10 to 12 weeks, refracture was possible, and surgical treatment would be indicated for these patients.⁴² Still, there exists a fairly large and reputable group of surgeons who suggest that only in circumstances in which previous conservative treatment has failed should surgical treatment be implemented. These authors suggest that fractures that occur with no intramedullary sclerosis or no prior attempts at treatment not only will heal, but will allow athletes to return to weight bearing within 6 weeks and to activity by 12 weeks (Fig. 7.12, A through D).^{51,61-63}



Fig. 7.12 (A and B) Acute fifth metatarsal or "Jones fracture." (C and D) The patient elected for conservative treatment and healed uneventfully after 6 weeks of non-weight-bearing casting.

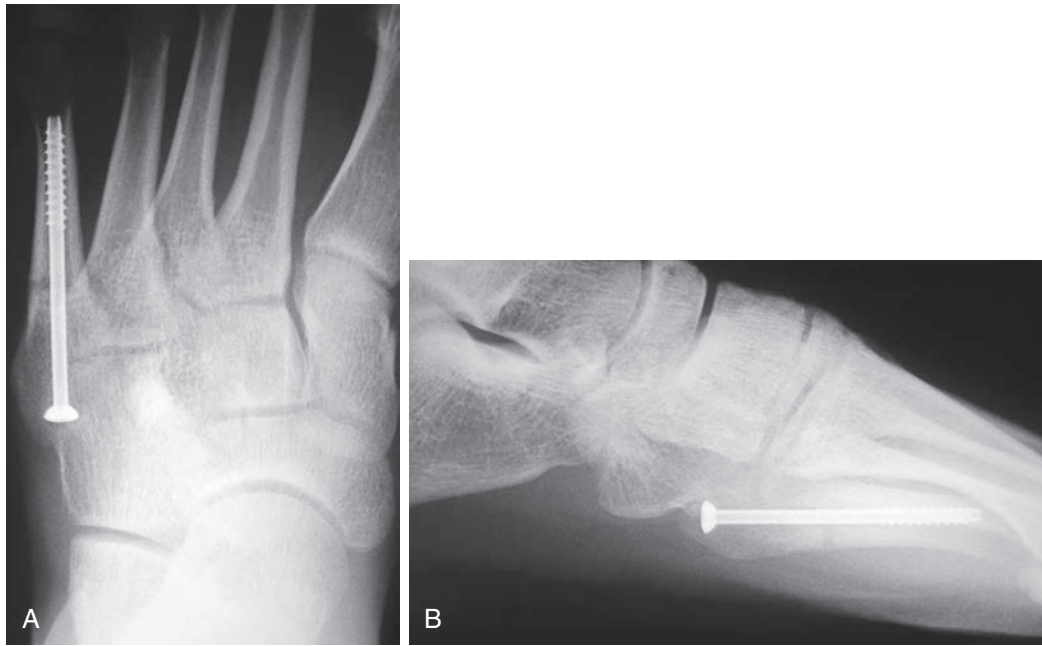


Fig. 7.13 (A and B) Percutaneous fracture reduction and treatment with intramedullary screw fixation.



Fig. 7.14 (A and B) Shows a well-healed fifth metatarsal fracture in a professional soccer player. Six months after return to full activities he came back with acute increase in pain and swelling. (C) Shows refracture as well as fracture of the screw. (D) Three months after removal of the screw and plantar plate fixation with bone marrow aspirate. Complete healing, and athlete returned to play.

In general, however, most authors agree that because of the potential for refracture, the cited delayed union rate, and the incapacitation required from nonweight bearing and immobilization as a result of casting, high-performance athletes and high-demand individuals be given the option for and be treated with some form of surgical management.^{42,44,55,58,64,65}

Paired comparisons of operative versus nonoperative treatments have been analyzed. Josefsson et al.⁵⁰ described 63 patients in which one-third of the patients were treated operatively and two-thirds conservatively. Average follow-up was 5 years, and, on the basis of delayed union or refracture, in almost 25% of the nonsurgically treated patients, subsequent surgical treatment was required. Late surgery was required in 12% of the acute fractures and 50% of chronic fractures. Clapper indirectly supported operative intervention, based on a review of 100 patients treated for acute Jones fractures with 8 weeks of non-weight-bearing casting. Results demonstrated only a 72% success rate with this form of treatment and average time to union of 21 weeks.⁵⁹

On the basis of the historical literature and currently available techniques and prevailing opinions, a protocol has been established that is my preference for the approach to the fifth metatarsal-based fracture. This should be fractures of zone 1, acute fractures of the tuberosity portion that are nondisplaced, typically are treated with a removable boot, and typically require 6 to 8 weeks for full healing. Surgery is virtually never indicated in this type of patient unless a painful non-union develops. Should the fracture be displaced or comminuted, the activity level of the patient must be assessed. In a younger, high-performance athlete, surgical management certainly is offered and may be helpful to reduce the risk of late complications and speed to recovery.

In zone 2 injuries, the classic acute Jones-type fracture, completely nondisplaced fractures may be treated in a non-weight-bearing cast for 6 to 8 weeks in a moderate-demand to low-demand type of patient. High-performance athletes should be offered intramedullary percutaneous screw fixation in a technique as previously described (Fig. 7.13, A and B).

Granata et al. showed a refracture rate of 7.3% with intramedullary screw fixation, which was felt to be mainly due to too small-diameter fixation. Average time to refracture was 8 months.⁶⁵

Lareau et al. reported on acute fixation of Jones fractures in NFL players, followed by an aggressive rehabilitation program. All players returned to play. Average return to play was between 8 to 10 weeks. The refracture rate was 12%.⁵³

O'Malley et al. reported on operative treatment of fifth metatarsal fractures in NBA players who were treated with a screw and bone marrow aspirate concentrate. Radiographic healing happened in 7.5 weeks, and return to play was 9.8 weeks. There was a 30% refracture rate.⁵²

Most of the studies in athletes show a significant refracture rate, and it brings into question if one should look at additional or alternative fixation options.

Varner treated eight Jones fractures with a plantar plate, four of which were for refractures. All eight fractures healed in 6.5 weeks, and athletes returned to play in 12 weeks. There were no refractures in the 15 months of follow-up (Fig. 7.14, A through D).⁶⁶

A zone 3 injury, a true shaft fracture, usually involves a distraction-type force and typically behaves differently from a Jones fracture. These injuries often will present in a delayed fashion and may in fact even be stress fractures. An acute fracture in this region typically will heal with a non-weight-bearing cast in a lower-demand individual, but again, operative treatment as described for the Jones fracture should be offered to a high-demand or high-performance athlete.

In a more delayed or recurrent injury at this level with prodromal symptoms, these patients should be treated with surgical management with intramedullary screw, with or without application of bone graft. Due to the variable curvature of the bone more distally, these injuries may not be as amenable to intramedullary screw stabilization and plate fixation, should be considered a viable and more reasonable alternative. Either plantar or dorsolateral plating has been effective and does not seem to provide much irritation despite the relatively thin soft tissue envelope here.

Frank non-unions and chronic injuries should be treated with internal fixation and bone grafting. The author leans towards plantar plate fixation and bone grafting in these situations.⁶⁶


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Video Legends - <https://www.kollaborate.tv/link?id=5c925cadde934>

 **Video 7.1** Title: Clinical evaluation for midfoot and Lisfranc instability. Legend: This video demonstrates clinical evaluation for Lisfranc midfoot instability. The hindfoot is stabilized by the right hand and the left hand pushes the foot into abduction.

 **Video 7.2** Title: Radiographic demonstration of midfoot instability. Legend: Abduction stress is placed across the midfoot while examining the foot radiographically with a physician controlled miniature C-arm x-ray machine. Instability of the Lisfranc midfoot ligaments is demonstrated on this radiographic video.

Rupture of the Anterior Tibial and Peroneal Tendons

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RUPTURE OF THE ANTERIOR TIBIAL TENDON

Anatomy

The anatomy of the tendon makes it susceptible to degenerative rupture due to attrition under the extensor retinaculum.^{1,2} The healthy tendon passes under the medial compartment of the transverse and cruciate crural ligament, and inserts onto the medial cuneiform dorsally and medially, with an extension of the attachment to the more inferomedial aspect of the first tarsometatarsal joint and first metatarsal. The tendon frequently divides into two slips, equal in size, one on the medial cuneiform and the other on the medial base of the first metatarsal, and only occasionally does the tendon attach only onto the medial cuneiform.

Rupture of the anterior tibial tendon (ATT) usually occurs in older individuals due to degeneration and friction underneath the extensor retinaculum.^{1,3-6} There is always a retraction of the proximal stump of the tendon ranging from 1 cm to 10 cm, and not infrequently there is only a small stump left distally at its attachment. Because the rupture rarely occurs as an acute precipitous episode, most patients do not present for some time, and a delay in diagnosis with further retraction of the tendon is common.

Diagnosis

The diagnosis of rupture of the ATT is made clinically by virtue of weakness of dorsiflexion and a foot drop, and rarely are

imaging studies necessary. Occasionally if there is focal pain, and one suspects inflammation or early degeneration, then either ultrasound or magnetic resonance imaging (MRI) are useful to delineate the extent of disease.³⁻⁵ In the presence of complete rupture, MRI of the tendon rupture does not add anything to planning treatment, since it is impossible to determine the quality of the ruptured tendon, although some surgeons may still use MRI to determine the extent of tendon retraction into the leg. Ultrasound may be used as a more dynamic evaluation of the tendon to determine the excursion of the ruptured tendon, which may have some impact on planning treatment. If the tendon has retracted and one is faced with a chronic rupture, then an MRI has a role, but here we do not focus on the ankle, but rather on the leg. This is relevant particularly in chronic ruptures where the tendon repair will require a graft, but this treatment cannot work if the muscle is not healthy. We therefore look for atrophy or fatty infiltration into the muscle prior to surgery to determine if a graft is possible or a tendon transfer is necessary.

Methods of Repair

We have three methods of repair available: an end-to-end repair, the use of an adjacent tendon such as the extensor hallucis longus (EHL), or a tendon graft.⁶⁻¹⁰ The repair chosen depends on

the size of the defect and the quality of the anterior tibial muscle noted on MRI. It does not help with treatment planning to obtain an MRI of the ankle, since this does not demonstrate the quality of the muscle, i.e., with fatty infiltration or muscle atrophy, which would contraindicate the use of a tendon graft. If a graft is not available or if the quality of the muscle is poor, then a tendon transfer with either the EHL or the EHL and the extensor digitorum longus (EDL) combined is used. For a rupture with a small gap less than 1 cm with good quality of the tendon stumps on both sides, an end-to-end repair is appropriate and easy to perform.¹¹

A patient rarely presents early enough such that an end-to-end repair or reattachment of the tendon can be easily performed. An equinus contracture or fixed claw toe deformities will often present as a result of the rupture of the ATT and the attempt to compensate by using additional muscles to extend the foot. In these cases, a transfer of the EHL and/or the EDL is indicated in combination with an arthrodesis of the interphalangeal (IP) joints of the toes.

If the ATT is short but has enough length to be attached to the navicular, this can be considered. This repair is very effective in cases in which retraction of the tendon is minimal but not enough length is available to reattach it distally into the

cuneiform. It is important to insert or attach the ATT to the navicular in the correct position under fluoroscopy over the dorsomedial navicular using a drill hole, and passing the tendon through the hole and securing it with either an interference screw or a suture anchor (Fig. 8.1A–C).

Skin Incision and Approach to the Tendon

Regardless of the type of repair or reconstruction used, the skin incision must always be more lateral than the underlying repair so that with skin closure, it is not directly over the repair, thus reducing the pressure from the tendon on the actual incision. This is less likely to occur if the tendon repair, transfer, or graft is passed under the retinaculum, but the latter is frequently scarred and this passing under the retinaculum is difficult to do. Always therefore make the incision along the central aspect of the foot at least 1 cm lateral to the position of the ATT. The skin is retracted medially, the extensor retinaculum is incised longitudinally, and the tendon ends are visualized. Before incising the retinaculum, pass a clamp under the retinaculum to see if passage of the tendon will occur to avoid cutting the retinaculum. Frequently, however, the extensor retinaculum has to be incised distally to be able to identify the stump of the tendon. The proximal tendon is sutured with a



Fig. 8.1 This patient sustained a rupture 8 weeks prior to diagnosis. (A) Note the slight hemorrhage in the distal tendon with fraying of the tendon margins distally. One can see that the tendon has been avulsed under the retinaculum; minor splits are still present in the distal tendon. (B) This is commonly associated with early ruptures. One can attempt to pull on the tendon for 10 minutes to determine if any relaxation of the scarring, fibrosis, and contracture occurs. (C) In this case, no further mobility of the tendon was noted and it was secured into the navicular shown here with an anchor inserted under fluoroscopy.

No. 2 suture, and the tendon is then pulled distally. Maximal tension is applied to the tendon for 10 minutes to determine the mobility of the muscle and then obtain some relaxation with elongation of the tendon at the musculotendinous junction. The biggest challenge with this repair is to obtain the correct tension; unless constant tension is applied at this time, elongation of the muscle develops later on, along with dorsiflexion weakness and a partial foot drop.

The other problem that occurs with all repairs is slight over-supination of the foot—an inevitable consequence of tightening the repair. This should not be of concern initially, although the foot position must be monitored during the recovery and rehabilitation phase. An Achilles tendon lengthening may be necessary to regain adequate dorsiflexion and to correct the position of the foot during the repair. The repair should be performed with minimal tension on the tendon, and the position of the foot *must* be passively correctable to at least 10 degrees of dorsiflexion without much resistance. Immediately postoperatively, use of a cast, rather than a splint, is preferable to hold the foot in dorsiflexion.

Tendon Graft

If the tendon is chronically ruptured and retraction of the tendon is greater than 3 cm, a tendon graft is useful and a hamstring allograft or autograft is used (Fig. 8.2A–G). A tunnel is created under the retinaculum with a large clamp and exits at the medial cuneiform where a small incision is made. We find that it is easier to attach the graft to the proximal ATT first before the distal attachment, which is far more difficult to gain the correct tension if done the other way around. The tendon graft is attached to the ATT using a weave through and through the ATT, which means that the graft has to be tapered to half its diameter to facilitate passage of the tendon weave. It is then passed under the retinaculum and attached to the cuneiform. The graft is always long enough to be able to secure it to the cuneiform using a double drill hole technique. This will give maximum bone tendon bone healing and more importantly allow one to tension the tendon adequately. This is more difficult when using a suture anchor technique. Here, with the latter technique, we recommend starting with the distal attachment, creating a soft tissue flap, suturing the tendon graft using the



Fig. 8.2 A chronic rupture was present in this patient who presented 6 months following the initial rupture episode. (A,B) The skin incisions are marked out. Note that the proximal incision is anterolateral, lateral to the passage of the anterior tibial tendon. (C) The allograft is now attached to the medial cuneiform with a double drill hole technique supplemented by a suture anchor. (D) The distal stump of the anterior tibial tendon is now identified and pulled with a hemostat clamp until it is quite free in the incision.

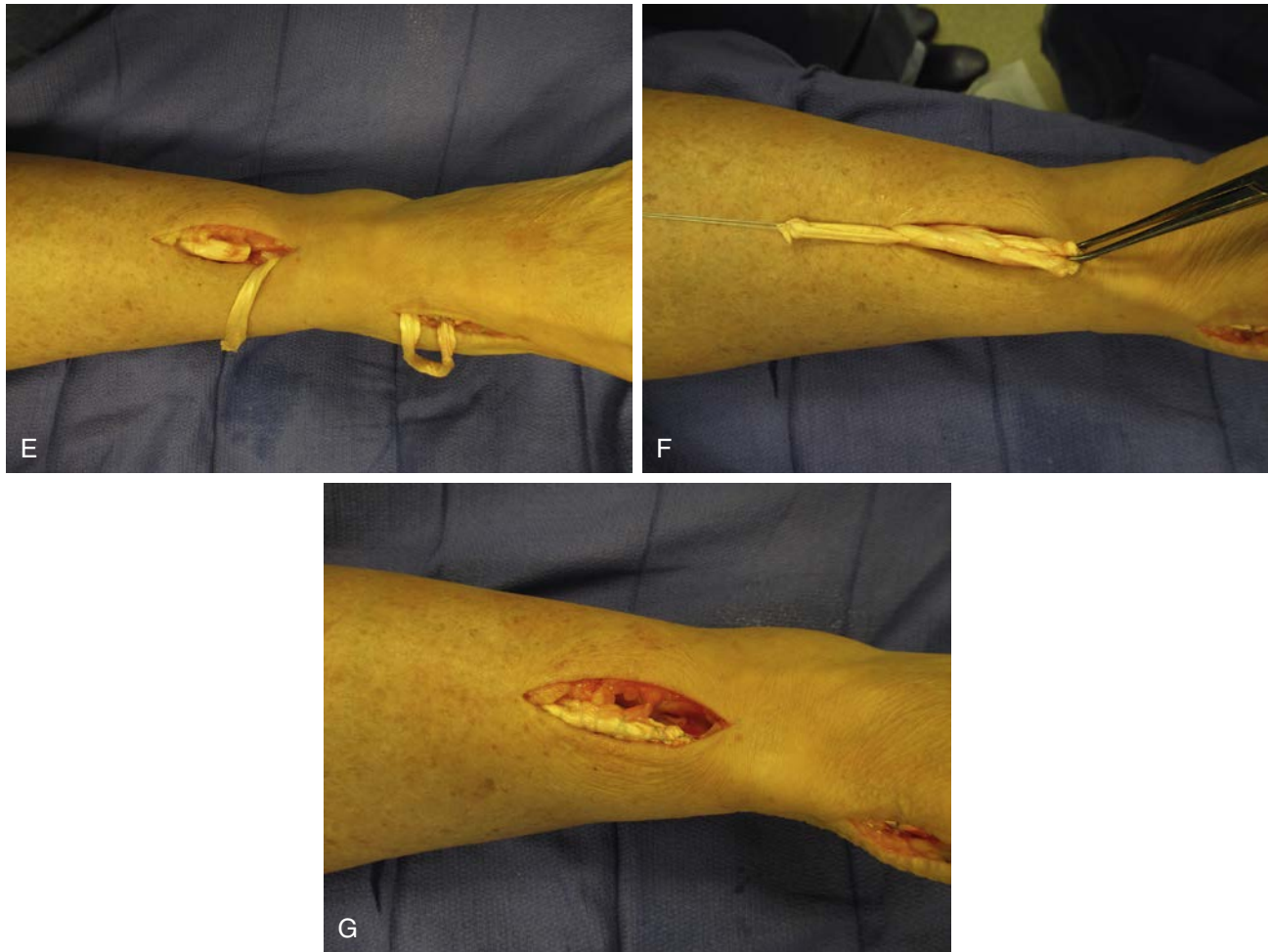


Fig. 8.2, cont'd (E) The allograft is passed deep to the retinaculum and the distal anterior tibial tendon cut sharply proximal to the zone of tendon injury. The allograft is now slightly thinned to facilitate passage through the anterior tibial tendon using a weave type attachment. (F) It is important to maintain the tension at each passage of the tendon. (G) The tendon graft is finally sutured into the anterior tibial tendon using a 2-0 slowly resorbable suture.

anchor and then overlapping the flap over the tendon for further fixation (**Fig. 8.3A–E**).

Extensor Hallucis Longus Tendon Transfer

If an EHL tendon transfer is to be performed in conjunction with an IP joint arthrodesis, the arthrodesis is performed first. The EHL tendon is then cut distally and pulled proximally to lie adjacent to the insertion point of the ruptured tendon. The length usually is sufficient for a double strand of the EHL tendon. The tendon is pulled distally and then secured over the distal stump or the medial cuneiform with a suture or suture anchor. Alternatively, one can drill two perpendicular 4.5-mm holes into the dorsal and medial cuneiform and pass the EHL tendon through the tunnel. There will be sufficient tendon length to pass the distal portion of the EHL tendon proximally as a second strand and sutured down onto the proximal ATT under tension (**Fig. 8.4A–D**).

Regardless of the type of suture repair, the foot must be immobilized in dorsiflexion of at least 10 degrees and preferably 20 degrees during recovery. As noted above, a percutaneous Achilles tendon lengthening is the rule rather than not for these cases. We use a cast that is split immediately postoperatively to

prevent anurey equinus. Patients can start weight bearing in the cast at approximately 2 weeks once wound healing is apparent. Any plantar flexion beyond neutral must be avoided during the recovery process for the first 8 weeks. Aggressive physical therapy with rehabilitation is important once the cast is removed, for the muscle to regain strength.

Outcomes of Treatment

The outcomes of both nonoperative and operative treatment of ruptures of the ATT are quite satisfying, provided that the treatment meets the expectations of the patient.^{1,7,12,13} Many elderly patients do not object to the use of an ankle foot orthosis (AFO), and this can be supplemented with a dorsiflexion assist on the AFO, if the foot is catching on the ground in the swing phase of gait. The problem with the AFO is that it is not universally accepted, and if it is not worn regularly, the foot will catch as a result of the foot drop as it lags behind in the final swing phase of gait. Surgery will always restore the ability to dorsiflex the foot to at least minus 5 degrees, and although in the worst-case scenario full active dorsiflexion is not achieved, these patients function well. Once the skin has healed, we commence with rehabilitation as noted above. This



Fig. 8.3 This is a complex chronic rupture with scar and multiple strands of apparent tendon that are actually only tendon scar. (A) The healthy extensor hallucis longus (EHL) tendon can be noted deep in the incision. (D) The scarred and ruptured anterior tibial tendon (ATT) is now mobilized under tension with a hemostat. (E) At the completion of the graft interposition, the remaining scar tissue is used to reinforce the tendon, and the retinaculum repaired.

is very important, particularly in this age group who are prone to develop muscle atrophy that at times cannot be recovered. We have not identified any patients in our experience with a recurrent rupture following treatment, but weakness in active dorsiflexion most definitely does persist in about 15% of patients.

RUPTURE OF THE PERONEAL TENDONS

Introduction and Tenosynovitis

Rupture of the peroneal leads to unpredictable results even with what appears to be optimal surgical treatment. We used to believe that a rupture that involved more than 50% of the

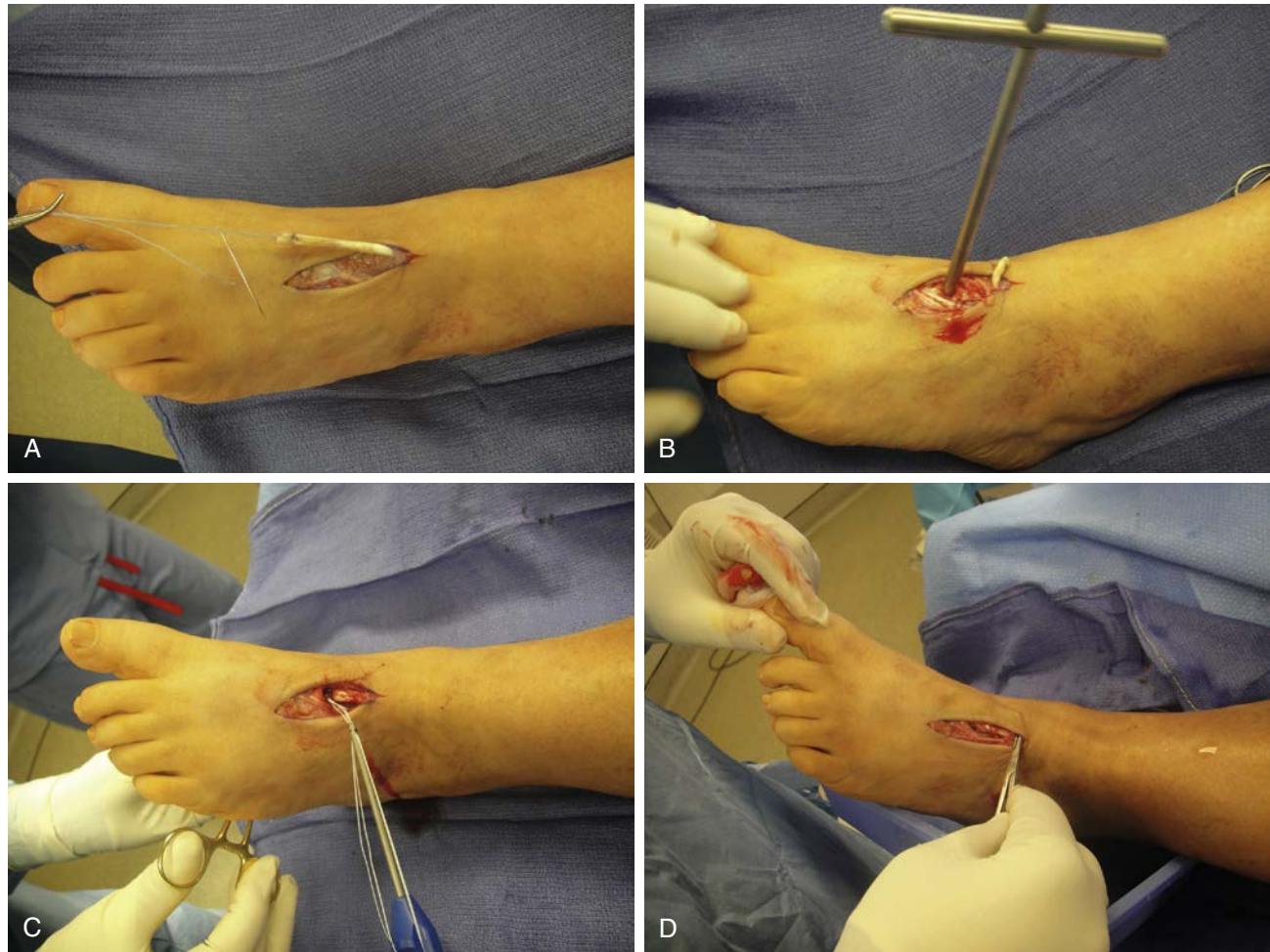


Fig. 8.4 In this case, the anterior tibial tendon (ATT) was so severely scarred and associated with fatty infiltration of the proximal muscle on magnetic resonance imaging (MRI) that we went directly to the extensor hallucis longus (EHL) transfer without opening the ATT sheath. (A) The EHL is cut in the midfoot leaving sufficient length to pass through a drill hole in the middle cuneiform. (B) A tunnel is made in the middle cuneiform using a hand reamer that removes a 7-mm diameter of bone. (C) A needle is attached to the suture on the EHL and passed through the cored hole, into which a suture anchor has been inserted prior to passing the EHL tendon. (D) The tendon is then pulled into the hole and tensioned with the foot in 10 degrees of dorsiflexion and sutured.

tendon, a repair would not be successful, but it is apparent that this is not the case in recent studies where as little as 30% of a viable tendon may be sufficient for tendon function.¹⁴⁻¹⁶ Perhaps the greater issue is that we do not understand what is the capacity for tendon healing to occur. The issue has to do with chronic fibrillation of the tendon during rupture, and it is rare that one has a clean tear of the tendon. More likely with both tendons, but more commonly with the brevis, multiple longitudinal splits of the tendon are present, and it is difficult to know what the potential is for one or the other strand to heal with suture repair.¹⁶⁻¹⁸

We are not certain if a true form of tenosynovitis actually occurs, or whether these are all prerule conditions, since progression to more extensive tears seems to be common.¹⁹⁻²³ Regardless of the pathology, it is our opinion that the earlier these conditions are treated, the better (whether tenosynovitis, tendon fibrillation, or complete tendon tears), given we see that pathologic changes will progress, leading to a far worse

clinical condition. As the tearing worsens, deformity begins to occur (in particular, varus deformity of the heel). A common example of early disease occurs with stenosis at the level of the peroneal tubercle, which enlarges when associated with heel varus deformity, and as the peroneal tubercle enlarges, it will lead to a complete rupture.^{15,22} Although the tubercle may enlarge for no particular reason, enlargement is more common in patients with heel varus or a cavus foot deformity. This leads to chronic friction and pressure of the tendons on the tubercle, causing what appears to be an hourglass shape deformity under the retinaculum over the tubercle, and this will cause thinning and narrowing of the tendons. The tendon changes will in turn be worsened by the increased stress on the tendons if the heel is in varus. If a patient presents therefore with chronic pain directly over the tubercle, one has to consider stenosis of the tendon(s) (Fig. 8.5A–D). The tubercle presses from below and the retinaculum from above, leading to the stenosis. If treated early enough, simple arthroscopic

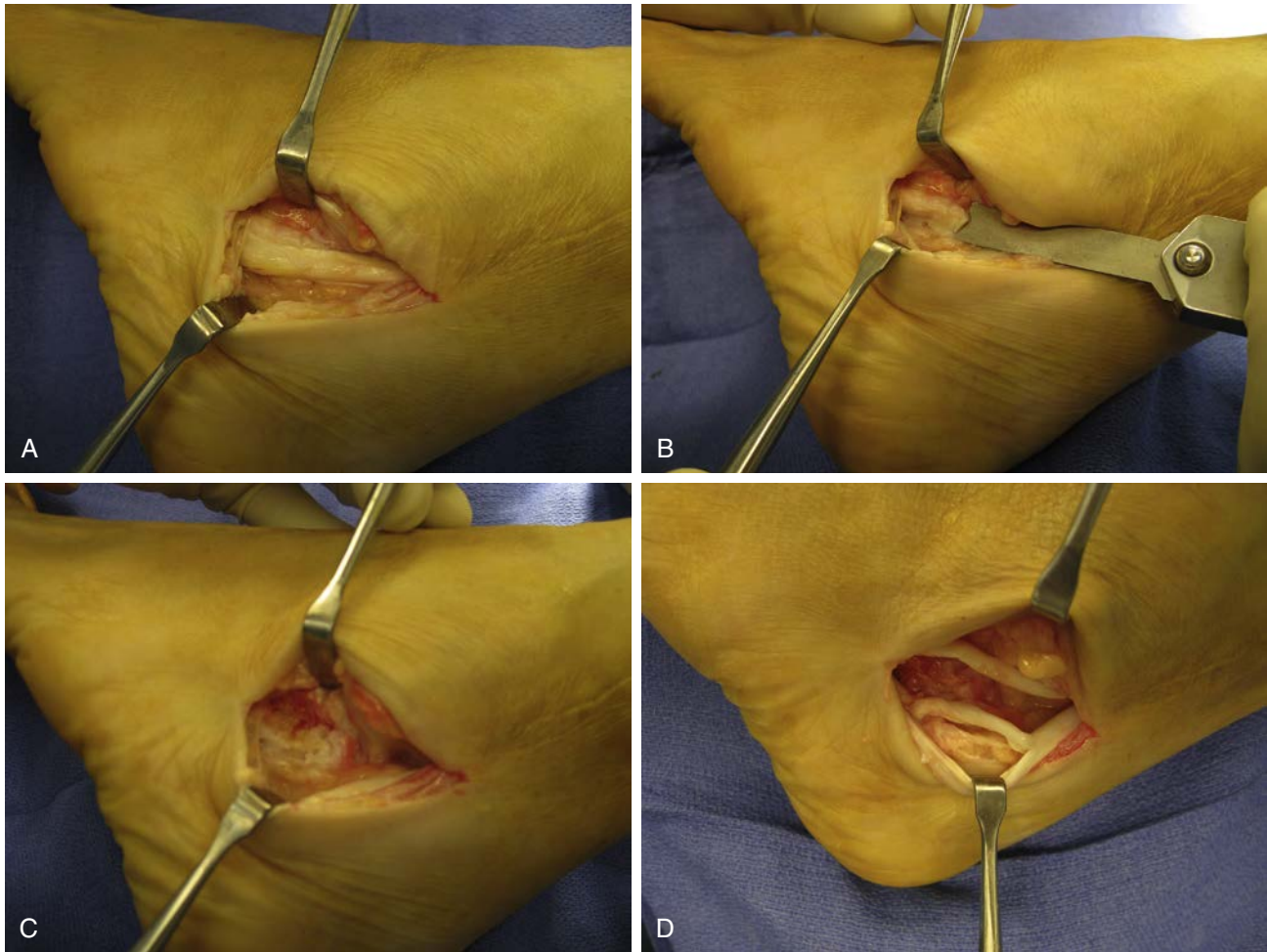


Fig. 8.5 This patient had experienced 1 year of pain over the mid-lateral calcaneus, and the pain was directly over the peroneal tubercle. (A) When opened, a normal brevis tendon was present, but the longus was severely hypertrophied and partly torn. (B) Both tendons were retracted and the tubercle removed with a sharp chisel. (C) Bone wax was then applied to the surface of the bone. One must always inspect both tendons carefully, since this tear of the longus was not initially apparent. (D) The tear was excised and the tendon left open without a tubular repair of the tendon.

decompression and debridement of the tubercle, or an open release of the tendon sheaths and removal of the tubercle, is a very effective early treatment.^{21,24} Left untreated, however, the stenosis will cause rupture of one or both tendons. The longus and brevis tendons distal to the fibula have a separate sheath, and both tendons should be opened and the split in the tendon(s) identified under the separate retinaculum, and the peroneal tubercle is debrided if it is seen to be enlarged. I use bone wax on the raw abraded surface under the peroneal tubercle once the repair is done. MRI might be expected to be a useful diagnostic modality in the management of acute tears or inflammation, but we have found that there is not a good correlation between the clinical and pathologic findings and MRI (Fig. 8.6). This is not, however, the case with chronic ruptures, where an MRI of the leg will yield useful information, particularly if one is considering whether or not a tendon transfer or a tendon graft is required for treatment. If there is muscle atrophy, or fatty infiltration of the peroneal muscles, then it is not possible to use these tendons for reconstruction, and one has to rely on a tendon transfer.¹⁴

Another common cause of retro-fibula pain that is worsened by passive dorsiflexion of the ankle is a low-lying peroneus brevis muscle.²⁵⁻²⁸ This is not associated with a tear of the tendon, but the patient presents with the same symptoms. Generally, at the level of the distal fibula, there is no muscle present, so that gliding of the tendons is present with passive plantar and dorsiflexion of the ankle. When there is a low-lying brevis muscle the tendons do not subluxate, although the patient will report that it feels as if the tendon is going to pop, but is more associated with pain than a feeling of subluxation. The pain is caused by the volume effect of the increased mass of the tendons and muscle under the retinaculum. As the foot dorsiflexes, the tendons are sucked into the fibula groove, and if the muscle volume is increased, pain will develop from impingement. This is very simple to treat. Usually, we have not made the diagnosis preoperatively unless an MRI had been obtained, but the diagnosis becomes immediately obvious when opening the retinaculum. The muscle is peeled off the tendon, and it is rare to find an associated tear of the tendon present. A simple repair of the retinaculum is enough to contain the tendons.



Fig. 8.6 The magnetic resonance imaging (MRI) of the tendons demonstrates edema around the longus tendon, but it is not possible to determine with any accuracy the extent of the tear. In our experience, there is no correlation between the MRI and the pathologic findings intraoperatively.

Tears of the Peroneus Longus

Tears of the peroneus longus occur more commonly at the level of either the peroneal tubercle or in association with a pathologic os peroneum as the tendon winds under the cuboid.²⁰ Rarely, the os peroneum is visible radiographically but not under the cuboid in its normal location, but more proximally along the margin of the calcaneal tuberosity. This is not always easy to see because of the shadows of the calcaneus, but efforts to observe should be made (**Fig. 8.7A–C**). A common X-ray finding of rupture of the peroneus longus is the position of the os peroneum on an oblique view of the foot. The os peroneum normally lies under the cuboid, and if it is more proximally located, particularly if one has prior radiographs for comparison, then an acute rupture is suspected. Acute ruptures of the longus occur under the cuboid, and one has to decide how the repair is performed. If the os peroneum is still present, and the tendon is fibrillated and torn at both ends of the os, then one can still try to perform an end-to-end repair with excision of the os peroneum. Excision of the os peroneum may create a defect, and if this is greater than 1 cm, it becomes difficult to repair end-to-end. Often, however, with careful excision of the os peroneum, the shell of the remaining longus tendon can be repaired with multiple running weave sutures as the tendon passes under the cuboid. If one attempts this kind of repair under the cuboid, it is necessary to extend the skin incision



Fig. 8.7 (A) The normal appearance of the os peroneum is demonstrated here. (B,C) In contrast to the normal os, note the pathologic changes in the next illustration associated with hypertrophy and irregularity of the bone against the cuboid.

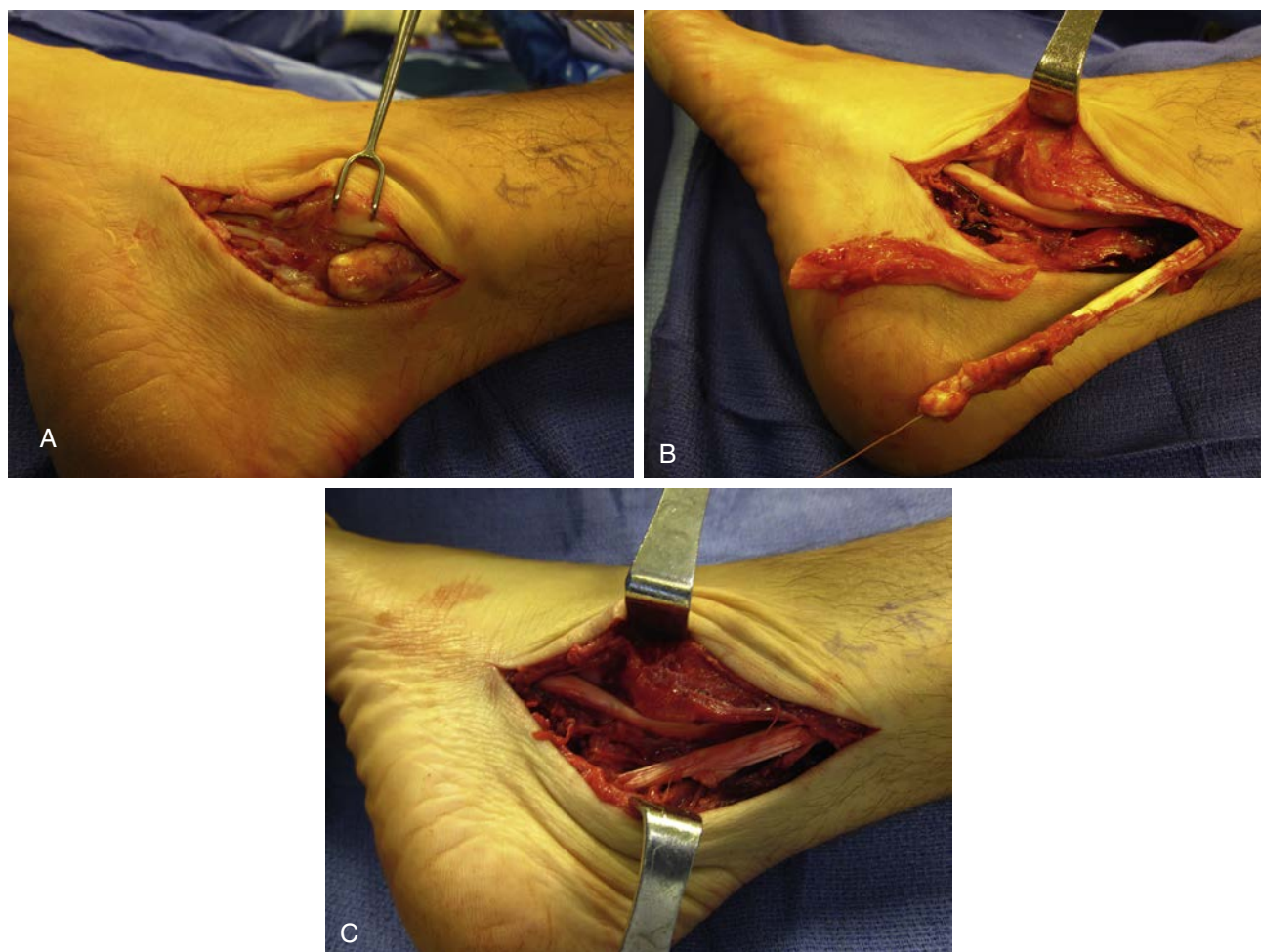


Fig. 8.8 (A) This rupture of the longus tendon occurred 8 weeks previously and the tendon has organized the stump and retracted almost to the level of the tip of the fibula. The scar has been excised and is lying free on the side of the foot, and the longus sutured and placed on tension. (B) Now one can appreciate the extent of proximal retraction. (C) As an alternative to suture of the ruptured tendon into the brevis, it was inserted into the calcaneus with an interference screw. This prevents potential scarring of the tenodesis.

along the base of the fifth metatarsal so as to gain more visualization under the cuboid by reflecting the abductor digiti minimi muscle dorsally.

If the tear is more chronic, it cannot be repaired in an end-to-end fashion because the retracted portion of the tendon is impossible to reattach distally under the cuboid (**Fig. 8.8A–C**). In this case, one has to decide what to do with the torn tendon. There are two options, one to transfer it into the peroneus brevis, and the other using a drill and interference screw into either the cuboid or calcaneus depending on the length of the tendon. We are not generally in favor of a tenodesis of the longus to the brevis, particularly in the athlete. The side-to-side tenodesis of the peroneus longus or peroneus brevis tendon is an acceptable procedure; however, insertion of the peroneus longus into the cuboid is preferable (**Fig. 8.9A–B**). Although transfer of the longus to the brevis is commonly performed to realign a cavovarus foot deformity, we worry that scarring of the tendon may cause symptoms in what was otherwise a normal brevis tendon. We have seen too many recurrent peroneal tendon problems with aggravated tears following side-to-side tenodesis of the abnormal

or torn tendon, such that wherever possible, it should be avoided. Generally, therefore, unless we specifically want to rebalance the hindfoot in a cavovarus deformity, a ruptured peroneus longus is inserted into the cuboid or calcaneus with an interference screw. Regardless of the treatment, unless one is able to repair the longus tendon underneath the cuboid, its function on the first metatarsal will be lost, but not its eversion function.

Tears of the Brevis Tendon

For repair of isolated tears of the peroneus brevis, an incision is made along the length of the posterolateral ankle extending along the course of the tendon behind the fibula. The proximal and distal extent of the incision is determined once the disease is identified after the retinaculum is opened. It may not be possible to preserve the extensor retinaculum by leaving a slip of the retinaculum intact, but make sure that there is a good cuff of tissue for repair at the completion of the repair, particularly at the distal fibula. The brevis tendon usually has multiple longitudinal splits or tears. It is important to check the ankle for instability, since there is a high incidence of tears of the peroneus

brevis associated with ankle instability.²⁹ There are a few ways to consider the repair. Either one excises the split tendon leaving the larger portion behind, or attempts a repair. Then one has to decide what to do with the remaining piece of the tendon that is left behind. We used to use a running slowly resorbable suture for repair of the remaining tendon, but found that this was not always successful in our hands or in those patients we treated who had already undergone debridement with repair (Fig. 8.10). A few years ago, we began to debride the major splits, leaving behind as much of one section of the tendon as possible and then just left it alone without a tubular repair of the tendon (Fig. 8.11). This treatment was far easier, quicker to recover from, since there was no tendon healing to recover from, and rehabilitation could begin earlier. The midterm (2–5 years) results with this technique seemed to be better than those tendons that we had repaired, but we never performed a definitive comparison of the two patient groups. We will leave the tendon behind even if the largest of the split pieces is about 30%–40% of the diameter of the normal tendon. Considerations in this decision include the size, length, and extent of the split.

If ankle instability and peroneal splits are associated with varus deformity of the heel, then all three components of the deformity can be addressed simultaneously.^{15,30} We prefer to use one and not two incisions for this procedure, through which the tendon repair, the ankle ligament reconstruction, and the peroneal debridement or repair can be performed. As for the type of ankle ligament reconstruction, one can perform an anatomic procedure, i.e., a Brostrom type procedure, supplemented by fiber tape or fiber wire sutures, but this depends on the quality of the anterior talofibular ligament. One cannot augment the repair with fiber tape unless there is sufficient ligament beneath it, since the fiber wire or tape will eventually fail unless there is biologically viable tissue beneath it. If there is any question as to the viability of the ligament or if the tear of the brevis tendon is extensive, reaching both proximal and distal to the fibula, a portion of the tendon may still be excised or even used as part of a nonanatomic ankle ligament reconstruction. The latter procedure is very useful when there is marked ankle instability and an anatomic repair cannot be performed, or where the patient is a heavy athlete for whom the Brostrom type repair will

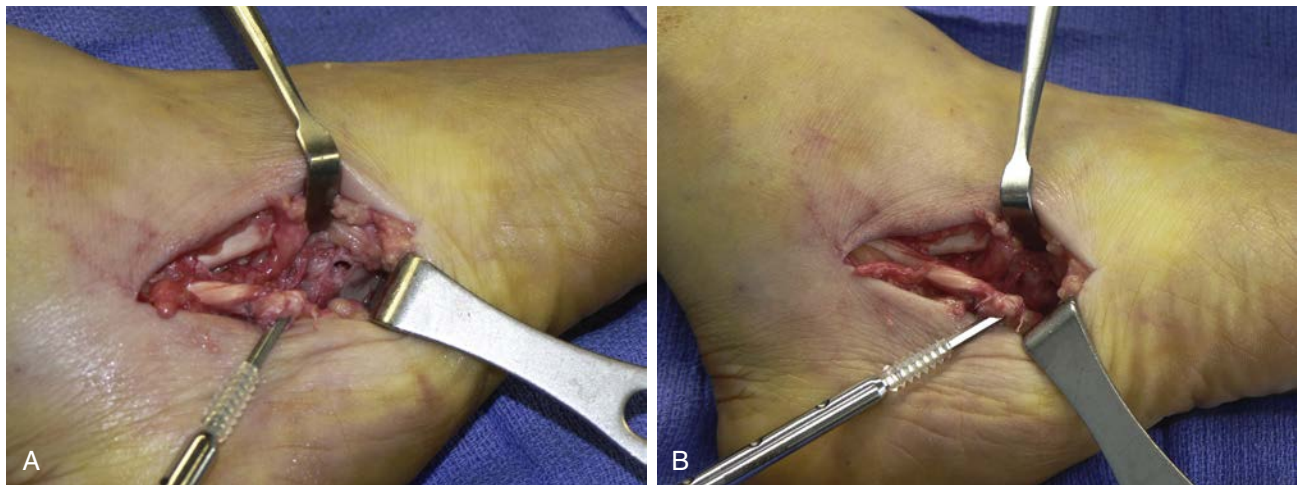


Fig. 8.9 (A,B) The rupture of the longus occurred more distally at the level of the os peroneum, but could not be repaired, and a drill hole was made into the cuboid and the tendon inserted with an interference screw.



Fig. 8.10 This isolated rupture of the brevis tendon included 30% of the tendon diameter, and the 70% remnant was then sutured by tubularizing the tendon.



Fig. 8.11 The rupture of the brevis tendon included 50% of its diameter, and the torn tendon was excised and not repaired. Once the tear was excised, no suture repair was performed.

not be sufficient. For these cases, in addition to the attempted Brostrum procedure, any number of different types of nonanatomic procedures can be considered such as the Evans or the Chrisman Snook procedure. In this type of case, a portion of one of the splits of the tendon may be used to reinforce the ankle ligament reconstruction to perform a Brostrum-Evans procedure. In cases where there is more deformity or severe instability, which may include the subtalar joint, a Chrisman Snook procedure should be considered. Any chronic isolated tear of the peroneus brevis may be associated with a varus deformity of the calcaneus. This is quite a common scenario in a cavus foot with heel varus, which is often associated with a tear of the peroneal tendons (**Fig. 8.12A–G**).

If the brevis tendon is irreparable, then one has to consider either a tendon transfer or a tendon graft.³¹ If a graft is used, the brevis muscle must be healthy on MRI and also demonstrate good excursion proximally. We note that an MRI of the leg is obtained in contrast to the usual technique of MRI of the ankle to evaluate the tendons only. The latter is of no value in managing a chronic rupture, since we are more interested in the quality of the muscle in order to perform a tendon graft. If the musculotendinous junction is scarred and has no mobility, then adding

a tendon graft to a nonfunctioning muscle does not make sense. In patients in whom both tendons are torn, but the muscle is healthy, with good excursion elicited by pulling on the musculotendinous junction, a tendon graft can be performed. The free graft is first attached proximally to the healthy tendon or at the musculotendinous junction. When it is attached distally, the correct tension on the graft must be applied. The optimal degree of tension may be difficult to determine because no retinaculum is present, and the tendon graft may have a tendency to sublux from behind the fibula. Once the suture attachment is performed distally, the retinaculum must be repaired anatomically to prevent dislocation.

If a tendon graft is not available, nor possible because of the quality of the peroneal muscle or significant scarring of the proximal tendon, then a transfer of either the flexor digitorum longus (FDL) or the flexor hallucis longus (FHL) tendon is performed. There are advantages and disadvantages of using either the FDL or the FHL tendon. All patients are aware of weakness following the use of the FHL, and it would be incorrect to assume that removal of the FHL does not cause functional losses. If one uses a short FHL tendon for transfer, then a limited length of the tendon is available, but there are

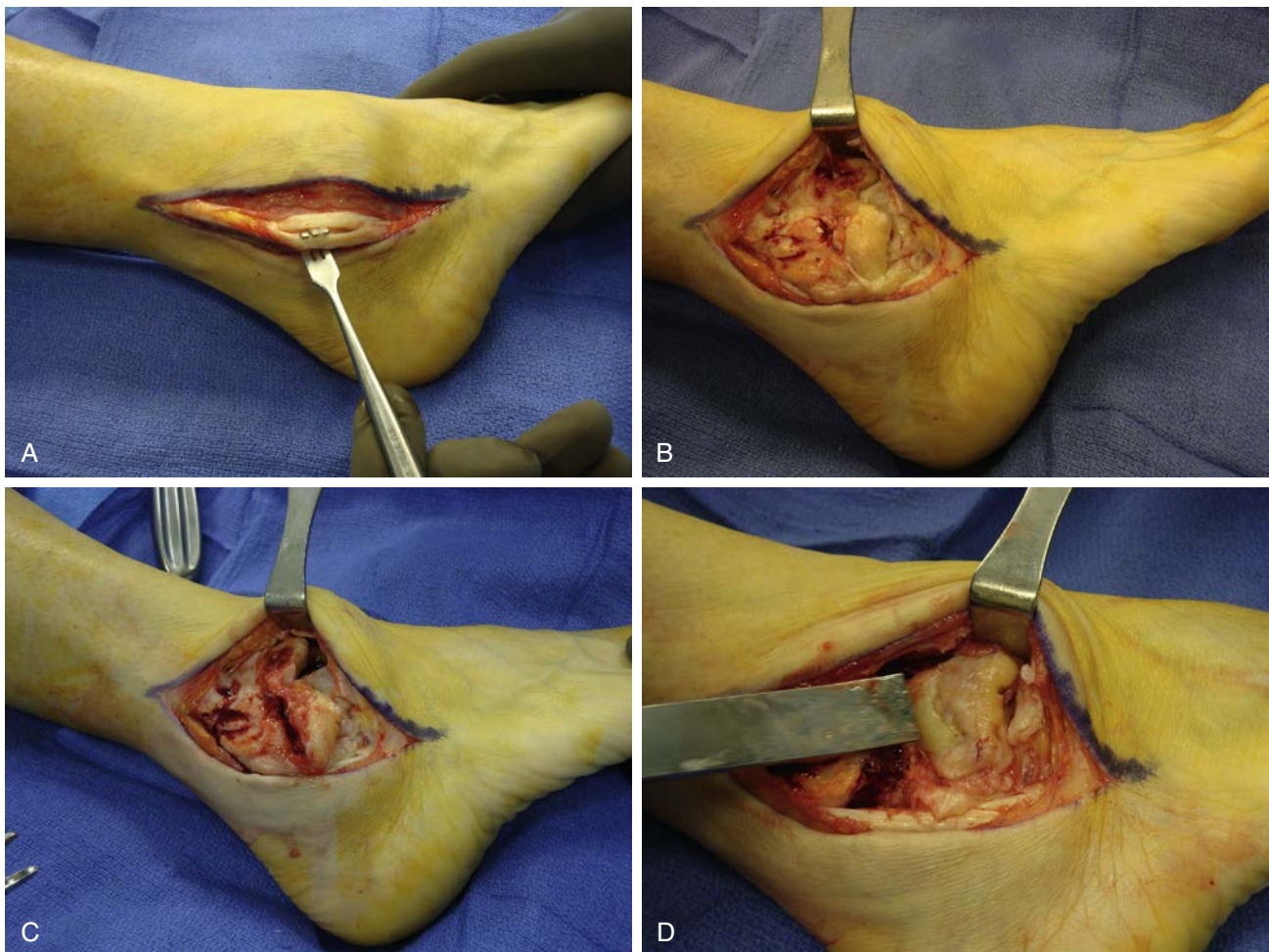


Fig. 8.12 This patient suffered from chronic retrofibular pain associated with ankle instability. (A) There was a massive osteophyte on the distal fibula and a tear of the brevis tendon noted. (B through D) The osteophyte was first removed with an osteotome from the distal fibula.

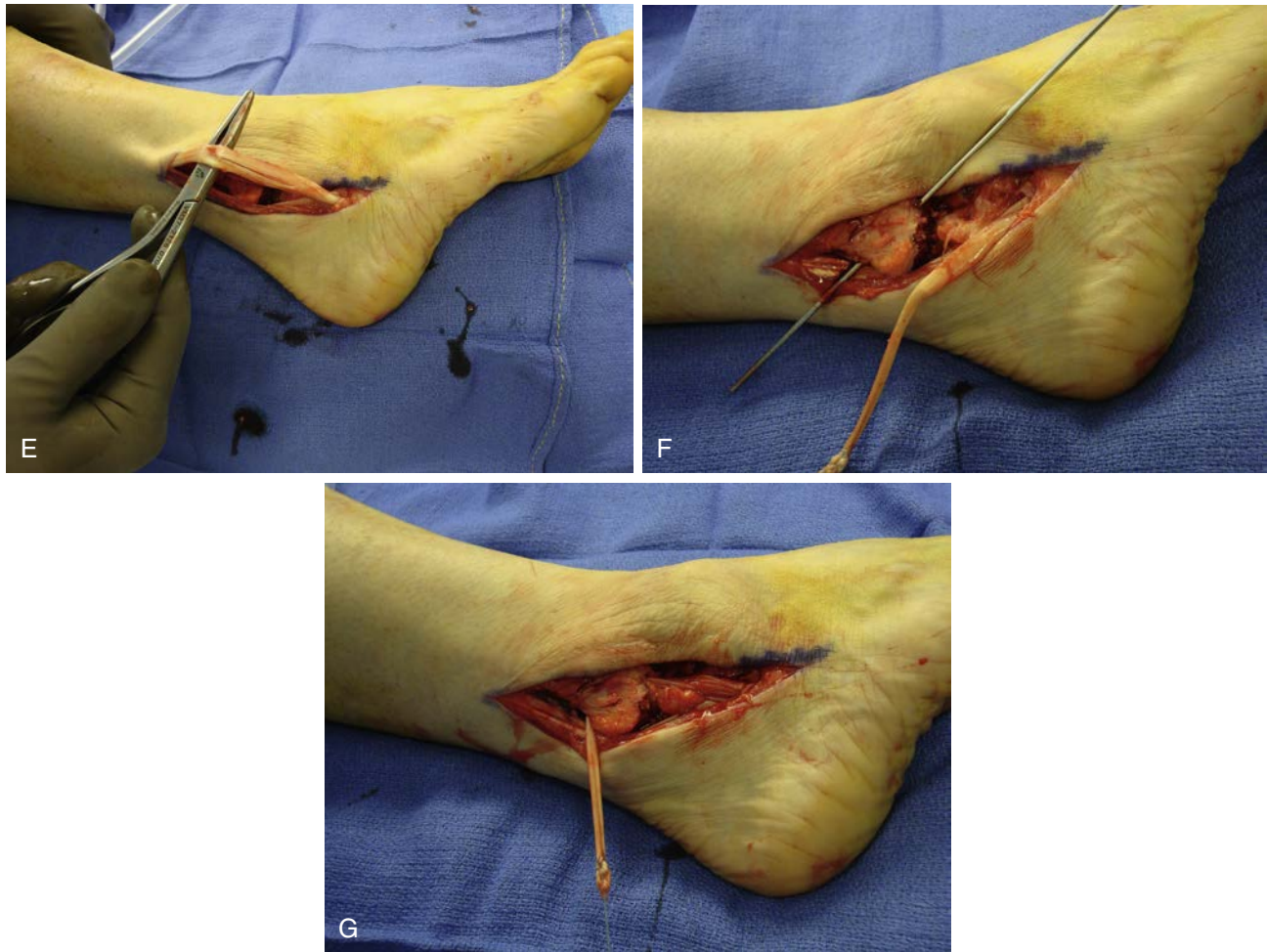


Fig. 8.12, cont'd (E through F) The split tear of the peroneus brevis was then extended more distally and the anterior half of the tendon used to perform a modified Chrisman Snook procedure.

cross connections between the FHL and the FDL just distal to the master knot of Henry, and if the FHL is cut proximal to this, there may still be adequate function of the hallux IP joint. The length of the tendon harvested in this manner is not very long, and is sufficient to pass around the back of the ankle and into the stump of the peroneus brevis. If, however, there is no peroneus brevis stump or if one desires better bone fixation, then the entire FHL tendon should be harvested with a tenotomy under the hallux IP flexion crease, and this will provide a very long strip of tendon to pass through a drill hole in the base of the fifth metatarsal and then sutured back on itself for a very secure fixation that permits early weight bearing. For the approach to harvest the FHL, we use an incision along the medial arch of the foot just between the plantar fascia and the abductor hallucis muscle. We then palpate the FHL by manipulating the hallux. Deeper dissection is performed until both the FHL and FDL tendons are visible. It can then be cut at the level proximal to the master knot of Henry or at the IP joint of the hallux depending on the length of the tendon required. Once the tendon is cut, there are cross connections between the tendon and adjacent tissue including the retinaculum, and the tendon often has to be more completely

released proximally so that it can be pulled out of the incision just behind the posteromedial ankle.

Tears of Both Tendons

When both tendons are ruptured, one should try to determine if the remaining strand of either one is viable to use, since one of the tendons (the longus) can be transferred to the distal stump of the brevis.^{16,32} This obviously depends on the percentage of the torn tendon that is still healthy. Alternatively, repair of one of the two tendons can be attempted, if the other is not salvageable. If both tendons are torn, there are generally only two options, either a tendon transfer or a tendon graft (**Fig. 8.13**). The gracilis/semiotendinosus graft is indicated provided it is available as an allograft or if the surgeon has the necessary skill to harvest an autograft hamstring tendon.³³ It can only be performed if the muscle remains healthy, with good excursion elicited by pulling on the tendon and noting mobility at the musculotendinous junction. The free graft is first attached proximally to the healthy tendon and then distally where it is easier to apply the correct tension to the graft. It is always very important to have the correct tension maintained, since



Fig. 8.13 Multiple tears of both tendons were present in this young patient who had undergone two prior attempts at repair. There are multiple splits in the brevis tendon, and the clamp is holding the longus tendon. The remnant of the longus tendon is severely scarred under the hemostat clamp.

there may be inevitable stretching out of the graft or the suture margins. The optimal degree of tension may be difficult to determine because no retinaculum is present, and the tendon graft may have a tendency to sublux from behind the fibula. Once the suture attachment is performed distally, the retinaculum must be repaired to prevent dislocation of the tendon. We do not perform a tendon graft for the peroneus longus, only for the brevis tendon.

Peroneal muscle weakness often is associated with a cavovarus foot, and injury to both tendons in a varus hindfoot is therefore common.^{14,15} A tear of both tendons leads to net inversion of the hindfoot by the tibialis posterior muscle, and this results in a fixed varus hindfoot deformity, which must be corrected in addition to the correction of the dynamic balance of the muscle imbalance. Ankle ligament instability needs to be corrected, and if a varus heel is present, a calcaneal osteotomy (Dwyer or lateral slide osteotomy) should be performed to decrease the force on the heel and protect the repair. It does not make any sense to perform a repair of the peroneal tendon in the setting of heel varus, which could lead to recurrent hindfoot instability and recurrent tearing of the tendon.

If a tendon transfer is indicated, we perform either an FDL or FHL tendon transfer to the stump of the peroneus brevis tendon or the base of the fifth metatarsal. This transfer is preferable if there is no proximal muscle function or excursion. If, however, excursion of the proximal muscle is present, use of a tendon graft (with a hamstring graft) seems more logical, but tendon grafting is contraindicated in the presence of muscle fibrosis or scarring of the tendons to the lateral compartment of the distal leg. With either repair, it is important to determine the condition of the tissue bed. If active inflammation or fibrosis exists, then a nonfunctional result is likely as a consequence of scarring and limited tendon excursion (**Fig. 8.14A–D**).

Correcting any deformity of the hindfoot or ankle that could lead to recurrent tendon injury is important. Repair of an unstable ankle must be performed as a planned procedure that is based on preoperative symptoms of instability of the ankle confirmed with stress radiographs. If present, associated hindfoot varus is corrected with a closing wedge biplanar calcaneal osteotomy, and the same incision is used for the peroneal tendon repair as for the calcaneal osteotomy.

PERONEAL TENDON DISLOCATION AND SUBLUXATION

Introduction

Peroneal tendon dislocation is often associated with athletic activity. It was first popularized as a diagnosis in the sport of downhill skiing.³⁴ It was surmised that the tip of the ski itself could get caught in the snow causing the athlete to dorsiflex and evert the ankle while the momentum carried the athlete down the hill. The tip of the ski would remain lodged in the snow while the peroneal tendon would seek a more direct path from the lateral foot to the retro-fibular area and dislocate anterior and laterally around the tip fibula. This dislocation has now been noted in most all sports and can be associated with similar activity to an ankle sprain. The association between lateral ankle instability and peroneal tendon dislocation is actually somewhat uncommon, and is probably around 10% (personal observation). It is speculated that a smooth or convex posterior fibula contribute to instability of the peroneal tendons. Nonoperative treatment for peroneal tendon dislocation has historically proven unsuccessful. However, surgical procedures for stabilization have historically shown excellent outcomes. With appropriate rehabilitation and avoidance of some common pitfalls, such as extensive immobilization that leads to scarring, a successful return to recreational or competitive athletics is typically attainable.

Historical Perspective

Peroneal tendon injury is a common problem in both the athletic and nonathletic population. Most peroneal tendon injuries occur in the middle-aged patient. Peroneal tendon tear and tendinopathy is the most common injury to the peroneal tendon complex. The degenerative tearing of the peroneal tendon again occurs in the older population, while the younger athletic population has a much less common incidence of peroneal tendon injury. However, in the younger population, and especially the athletic population, peroneal tendon dislocation is much more common.^{35,36} In fact, peroneal tendon dislocation may be the most common presentation for peroneal tendon complex injury in the teenage and young adult athlete.³⁷ A great deal of attention has been given to instability of the peroneal tendons, but true dislocation remains uncommon. Pseudo-subluxation or intrasheath peroneal tendon instability is also another cause of peroneal tendon pain.^{38–40} This involves the peroneal tendons subluxing within the sheath around one another. Intrasheath subluxation involves the peroneus longus tendon displacing lateral to the brevis within a stretched-out sheath and “loose” superior peroneal retinaculum.

The Anatomy of the Tissue (Peroneals, Retinaculum, and Posterior Fibula)⁴¹

The posterior sulcus of the fibula has a variable contour, thus it is considered a predisposition to contribute to dislocation in some athletes/patients.^{42,43} The two tendons are in a fibro-osseous tunnel bordered anteriorly by the posterior fibular fibro-cartilaginous sulcus (distal 4 cm), posteriorly and laterally by the superior peroneal retinaculum (primary constraint

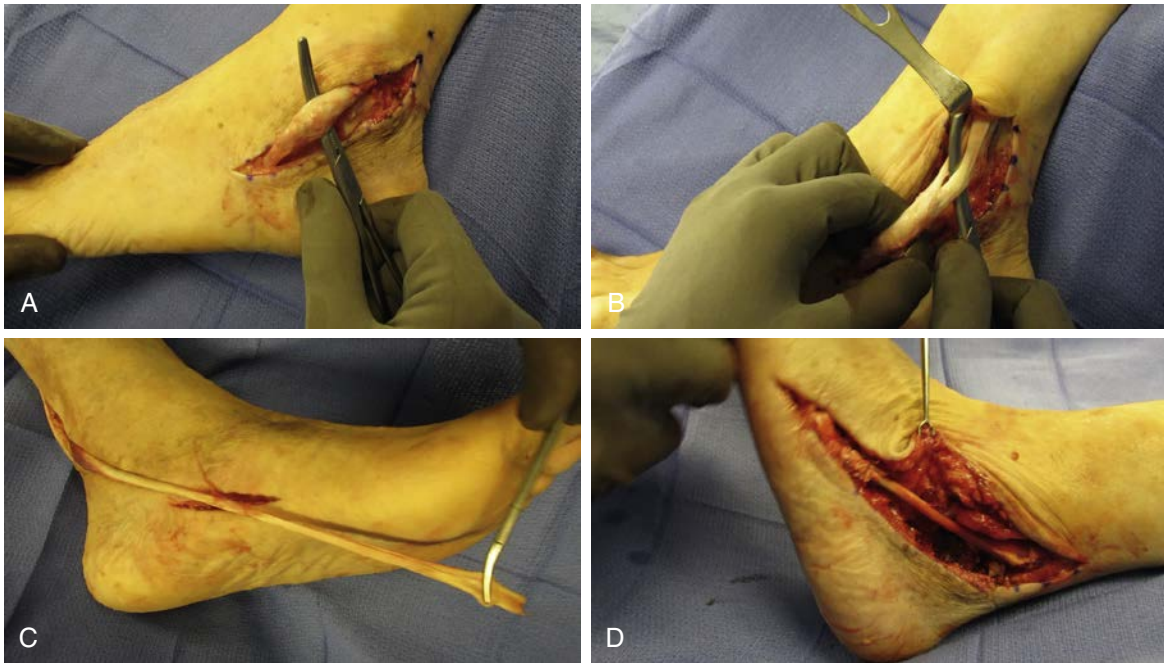


Fig. 8.14 This patient had undergone two prior surgeries with a tenodesis of the longus to the brevis as the last procedure. (A) Note the markedly thickened tendon. (B) The tendon was noted to be torn at the level of the prior attempted tenodesis. There was no good muscle noted on magnetic resonance imaging (MRI) of the leg, and a transfer of the flexor hallucis longus FHL was performed. (C) Note the length of the tendon that can be obtained by cutting the tendon under the interphalangeal (IP) joint of the hallux. (D) The FHL tendon is passed through a drill hole in the base of the fifth metatarsal and then sutured back on itself for excellent fixation, which will facilitate immediate bearing of weight.

to dislocation) and medially primarily by the calcaneal fibular ligament (CFL; a though the posterior talofibular ligament and posterior-inferior tibio-fibular ligament form part of the medial border). There is also a posterolateral fibrocartilaginous meniscus-like structure that adds depth and lateral constraint to the retromalleolar groove/sulcus.^{42,43} This is the structure that is often avulsed with acute tendon dislocations and degenerated in chronic dislocations. The posterior fibular anatomy has a varied surface contour. Eighty-two percent are concave, 11% are “flat,” and 7% are actually convex.^{41,44} The flat and convex anatomy in association with peroneal dislocation requires a formal fibular osteotomy for groove deepening in addition to reconstruction of the superior peroneal retinaculum, regardless of the acuity of the dislocation. It should be noted, however, that Adachi and coworkers⁴² did not find a difference in the inherent anatomy between subjects treated for peroneal dislocation and a similar population of subjects that did not have a peroneal tendon instability history. Schon’s lab has demonstrated that a peroneal groove-deepening procedure can reduce the pressure within the peroneal sheath.⁴⁵

The Nature of the Problem

A shallow, flat or convex posterior fibula is thought to lead to poor bony stability of the peroneal tendons. After a dislocation of the peroneal, lack of inherent bony stability puts a significant strain on the repaired or reconstructed superior peroneal retinaculum. It has been concluded that groove deepening of the posterior fibula is not only helpful but necessary in situations where there is no deep bony stability. Even mild concavity to the

posterior fibula is typically enough to prevent redislocation after appropriate superior peroneal repair in acute settings, but it could still need deepening in chronic cases that require retinacula reconstruction. The earliest approach to groove deepening was by Kelly⁴⁶ and involved a true rotational fibular osteotomy. We should note here that another approach taken by several authors involved rerouting the peroneal under the CFL by osteotomizing the CFL attachment to the distal fibula.⁴⁷⁻⁵⁰

History and Exam

The injury history can be very similar to a common lateral ankle sprain. It is commonly seen during sport and as mentioned, was first reported commonly in downhill skiers.³⁴ However, it can occur with any sport or active lifestyle activity. There is often a sensation of a “pop” and occasionally a feeling “like something came out of place.” The athlete notes pain in the lateral ankle and swelling in the posterior lateral ankle. Tenderness is more specific to the posterior lateral fibula than the anterior lateral ankle seen with lateral ankle sprains. In the acute setting, it is difficult to get the peroneals to dislocate on exam in the office. Two to three weeks after the injury, or in the chronic setting, the peroneals can be dislocated by plantarflexing the ankle, placing it in eversion and displacing the tendon over the posterolateral fibula to confirm the diagnosis (Video 8.1 and Video 1.11). A thorough ankle exam also needs to be performed to rule out concomitant lateral ankle ligament instability/injury. Routine ankle series x-rays are helpful and required. For the peroneal dislocation, assessment for a small posterolateral fibular avulsion or other associated bony/osteochondral lesion

(OCL) injuries is needed. In the acute setting, any type of bony fractures is evaluated well in with plain x-rays. Advanced imaging is reserved for chronic setting to assess for a concomitant peroneal tendon tear (most commonly a peroneus longus tear if present).⁵¹ Also, the superior peroneal retinaculum can be assessed in the acute setting and is seen best on the axial imaging. Thomas has described using ultrasound to evaluate the dislocating peroneals.⁴⁰

Treatment^{36,52}

Nonoperative treatment has not been shown to be effective for stabilizing the tendons. We have used a “J” or “U” pad around the lateral fibula to allow some athletes to complete a season. Operative stabilization is the mainstay of treatment.

The Author's Preferred Approach⁵³

We first determine if there is an acute dislocation. That is, are we seeing the athlete/patient within the first 1–2 weeks of injury, or is this a chronic recurring dislocation that has been re-occurring greater than 4 weeks from the time of initial injury?

For the acute dislocation, we still believe that operative treatment is the treatment of choice. In some situations, the posterior lateral cartilaginous rim is still intact and the posterior fibular anatomy has a reasonable concave contour. In this situation, with a good underlying groove, we will initially try acute repair of the superior peroneal retinaculum (SPR).^{54,55} We have had good success when there is a naturally good deep groove in the acute setting. However, we do not take this acute repair only approach in the chronic setting.⁵⁵

For the chronic recurring dislocation, there are numerous approaches described.^{38,46-49,53-74} We always perform a groove-deepening procedure. In this chronic setting, the posterior lateral cartilaginous rim is almost always absent, and we have not found the posterior fibula to have enough concavity to support the peroneal tendons and their position.

Surgeons have described several operative approaches to groove deepening of the fibula. We have categorized the osteotomies into direct groove deepening and indirect groove deepening. However, the general principles we believe are universal. We think the following principles are crucial to an optimal outcome:

1. A fibular osteotomy is necessary to give adequate groove deepening if not inherently present.
2. The fibular osteotomy must be deepened to a point that the posterior border of the peroneal tendons, when replaced within the deepened groove, is flush with, or anterior to, the posterior border of the resultant groove.
3. Tightening and imbrication of the superior peroneal retinaculum must be undertaken also to give appropriate soft tissue constraint to dislocation. Reattachment of the SPR must be undertaken in instances of true dislocation (intrasheath dislocations do not require reattachment of SPR, just imbrication).
4. Rehabilitation must include early range of motion (ROM) to prevent scarring and subsequent pain from tendon restriction. We believe early weight bearing is advantageous for accelerated recovery.

Direct Groove Deepening Approaches

RE Kelly in 1920, reporting from Liverpool England in the *British Journal of Surgery*,⁴⁶ first proposed groove deepening for a recurrent peroneal tendon dislocation. He described a sagittal “veneer”-type osteotomy of the distal 2 inches above the distal fibular tip and then rotated the lateral bony “veneer” of bone posterior to create a deepened groove.⁴⁶ Zoellner and Clancy⁷¹ proposed a hinged posterior flap of the retromalleolar groove that produced a more direct posterior deepening. Their technique involved the following: “the groove for the tendons is deepened by removal of some inner fibular substance, while the smooth tenosynovial channel is maintained as an intact periosteal flap on the fibula.”⁷¹ Slatis and co-workers⁶⁵ modified the Zoellner and Clancy⁷¹ report of direct deepening by removing the cartilaginous gliding layer of bone from the retromalleolar fibular groove, removing further cancellous bone using a curved chisel and replacing the cartilaginous gliding bone by impacting it into the deepened groove.⁶⁵ Porter, McCarroll, and co-workers⁵³ also modified the Zoellner and Clancy approach by removing the posterior cortical cancellous retromalleolar groove with the intact serosa surface, deepened the distal posterior groove by removing cancellous bone with a motorized 4.0 egg burr, and then replaced the gliding surface by reattaching it with sutures in the depth of the deepened groove.⁵³ This detailed groove deepening and standard retinacula reconstruction allowed the authors to offer a more accelerated protocol in their rehabilitation. The authors reported on 13 athletes (14 ankles) who were allowed early weight bearing (1–2 weeks PO); these only used intermittent immobilization with a walking boot (4 weeks total and then 2 weeks wean into a stirrup brace) and reported earlier return to sports (3 months). No dislocations and near-normal ROM were achieved (Fig. 8.15).

Zhenbo and colleagues⁷⁰ report in 2014 an approach reminiscent of the Kelly procedure. Their approach also involved a sagittal osteotomy in the fibula with a posterior slide of the osteotomy fixed with absorbable screws.⁷⁰

An oblique 20-degree (toward the sagittal plane) osteotomy was made anteromedially with a small oscillating saw extending from about 3 cm above the lateral malleolus to the fibular apex. When the saw nearly reached the posterior edge, the osteotomy exit(s) posterolaterally without damaging the cartilaginous ridge. The graft (3 × 2 × 0.5 cm) (is) slid 20 to 30 degrees (3–5 mm) posteriorly to ensure an adequate block to dislocation. The graft was secured to the distal fibula with 2 or 3 absorbable self-reinforced polylactide (SR-PLLA) screws (Conmed Biofix SmartScrew, Conmed Linvatec, Espoo, Finland). The SPR and sheath are attached to the graft, using the modified Das De technique.⁶⁸

Indirect Groove-Deepening Approaches

Shawen and Anderson first described the indirect approach.⁶⁴ The tip of the fibula is exposed and a “suitable sized” reamer from the Arthrex biotodeses screw set is used to “thin” the posterior cortex of the groove. A moderate, wide bone impactor is used to deepen the posterior surface in an indirect manner, recreating the concavity needed to insure stability. Walters and co-workers describe using a 3.5-mm drill to make multiple passes under the posterior; a “small” osteotome is utilized

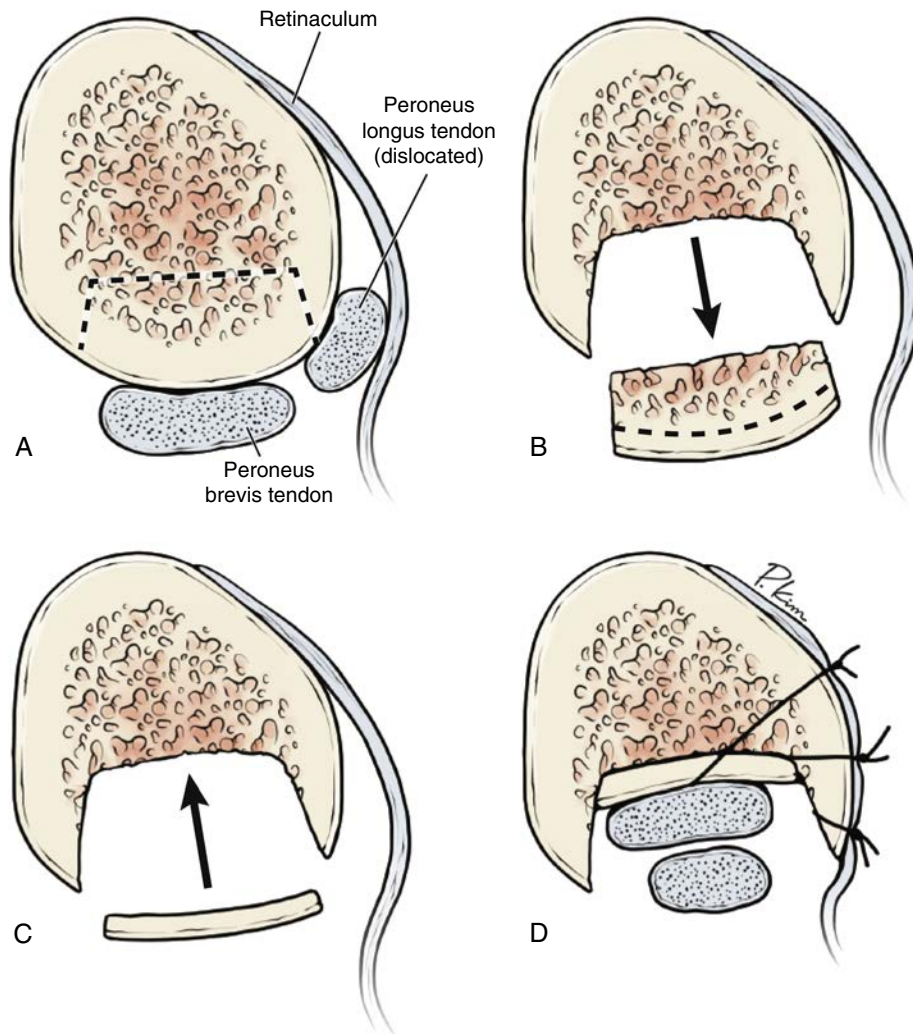


Fig. 8.15 A-D Porter and associates approach to direct fibular osteotomy for groove deepening. A modified approach to the Zoellner and Clancy⁴³ approach involves removing the posterior cortical cancellous sulcus (rather than hinging), deepening the cancellous groove, and replacing the posterior sulcus in the deepened cancellous bed with reattachment of the superior peroneal retinaculum. (From Porter D, McCarroll J, Knapp E, Torma J. Peroneal tendon subluxation in athletes: fibular groove deepening and retinacular reconstruction. *Foot Ankle Int.* 2005;26:436-441.)

to perforate the medial and lateral border of the retromalleolar groove; and the posterior cortical serosal surface can then be impacted/“compacted” to “deepen” the groove to a depth of at least 5 mm utilizing a wide blunt bone tamp/impactor. The posterior cartilage rim is retained if it is present.

Intrasheath Peroneal Dislocation Without SPR Avulsion

Intrasheath dislocations typically present as “snapping tendon” (see Video 1.11).³⁸⁻⁴⁰ There may or may not be a definable history of a lateral ankle injury. It is proposed that the SPR has been either torn or stretched but not avulsed, and thus there is added redundancy to the superior peroneal retinaculum. Thus, the peroneus longus subluxes laterally around the brevis but still stays within the peroneal sheath, creating this “snapping” sensation that can, at times, be audible.

Our approach is the same as the chronic dislocation. We surmise that to adequately return the tendons permanently to their

rightful position (longus posterior to the brevis) requires a deep groove to provide both bony and soft tissue constraints to subluxation. We have not attempted to treat these athletes/patients with a soft tissue correction only. Vega, Guelfi and co-workers^{75,76} from Barcelona have reported experience with tendoscopic treatment with six of the eight patients not requiring a fibular osteotomy but resection of a peroneus quartus and/or resection of low-lying peroneus brevis muscle with good results.

Our rehabilitation of these subluxations post-surgery is the same as the rehabilitation of a chronic condition, and is discussed below.

Rehabilitation Principles and Approaches

We advocate an early weight bearing and early ROM approach to alleviate some of the risk of scarring and stiffness than can come with prolonged immobilization. We utilize cold and compression therapy and a walking boot immobilization to allow the athlete to come out of the boot after week 2 to begin active ROM in

dorsiflexion, plantarflexion, and inversion. We do not allow eversion until 6 weeks PO to protect the SPR repair/reconstruction. We utilize biking with the walking boot at 2 weeks, we transition to a stirrup brace between weeks 6 and 8 and allow StairStepper-like exercises. Running is allowed after 10–12 weeks, functional progression is initiated after the athlete can run in a straight line with no pain, and full sports are allowed when the athlete can pass the functional progression test, typically 3–4 months postop.

Complications and Potential Pitfalls

Recurrent dislocation is uncommon, especially with groove-deepening procedures. We have only had one re-dislocation and it was in a female with Ehlers-Danlos on whom we tried using her native tissues. She did well with revision and use of allograft tissue. Infection should be rare, reported in less than 0.5% of cases. Wound healing problems are rare in the athlete but can be common if the patient is a smoker or a diabetic. Even with early ROM, we did not see a high propensity for wound healing issues. Peroneal tendon tear is common at the time of surgery in chronic, recurring dislocation cases, but is otherwise rare. We have found direct repair and/or tabularization has a high success rate. Non-union should be uncommon, and is not commonly reported. The osteotomy is performed in well-vascularized cancellous bone. If secured, we have found no non-unions even with early weight bearing and early ROM. There is a higher risk with the earlier rotational osteotomies in theory, but none had been reported. Stiffness can be a real potential for disability if the ankle is immobilized for a prolonged period of time. To counteract this potential complication/risk, we chose to secure the bone flap in the deepened groove, fully place the posterior bone flap in the groove, maintain the serosa surface on the posterior flap, and encourage early ROM. This necessitated boot/splint immobilization, rather than casting, and has now shown to be both reliable and near complication free.⁵³ Fibula fractures with large fragment fractures requiring open reduction and internal fixation are rare. Small fracturing of the posterior lateral lip of the lateral deepened groove can and has occurred in our series, but it responds well to boot immobilization for 3–4 weeks and then back into a stirrup brace with rare long-term difficulty and no requirements for further surgery. Sural nerve injury or neuritis is potentially debilitating complication because the nerve can be in the operative field for all peroneal tendon surgery approaches. If the surgeon stays close to the posterior border of the fibula, this takes the surgical field anterior to the sural nerve. Drifting the incision or dissection more posterior certainly puts the nerve at risk.

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Video Legend - <https://www.kollaborate.tv/link?id=5c925f43c5078>



Video 8.1 Title: Clinical Evaluation for Peroneal Tendon Dislocation

Legend: This video demonstrates evaluation of an athlete with chronic or subacute peroneal tendon dislocation. It demonstrates dislocation of the peroneus longus tendon across the fibula with manipulation. Typically, it is the peroneus longus that dislocates.

Achilles Tendon Disorders Including Tendinopathies and Ruptures

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INTRODUCTION

The Achilles tendon is formed by a coalescence of fibers from the gastrocnemius and soleus muscles. This complex spans both the knee and ankle joints, making it more susceptible to injury than muscles that span a single joint. The Achilles tendon is notably susceptible to injury with concomitant knee extension and ankle dorsiflexion. The medial and lateral heads of the gastrocnemius originate from the medial and lateral femoral condyles, respectively. The soleus muscle originates from the posterior proximal tibia and fibula. More distally, the medial and lateral gastrocnemius and soleus tendons coalesce to form the triceps surae complex. The Achilles tendon then rotates 90 degrees such that the medial gastrocnemius position is more posterior and superficial. This rotation may result in torque stresses that can increase the risk of tendinopathy.¹ After passing distal to the posterior superior calcaneal tuberosity, the Achilles tendon inserts into the posterior and plantar calcaneal tuberosity about halfway between the dorsal and plantar aspects of the calcaneus.

The retrocalcaneal bursa lies between the distal Achilles tendon and the posterior superior calcaneal tuberosity. It is horseshoe shaped and sits around the insertion of the Achilles, which has more fibers centrally and proximally. Anteriorly it is composed of fibrocartilage, whereas posteriorly it blends with the paratenon and commonly connects to the posterior Achilles

tendon. The pre-Achilles bursa lies superficial to the Achilles between the Achilles and the skin. These bursae, composed of synovium, provide lubrication to assist with tendon gliding and to minimize tendon irritation. A large, sometimes abnormal prominence of the posterior superior calcaneus, Haglund's deformity,² may create repetitive frictional irritation on the Achilles tendon that can lead to tendinopathy (Fig. 9.1A–B).

The Achilles tendon is the strongest and longest tendon in the body, measuring approximately 12 to 15 cm in length. Although it is the main plantarflexor of the ankle, it also functions to invert the heel during late stance phase and thereby locks the transverse tarsal joint for push-off along with the posterior tibial tendon. It is subject to forces up to 10 times body weight during running, experiencing up to 7000 N of force.^{1,3,4}

The blood supply to the Achilles tendon is segmental and is predominantly derived from anterior branches of the paratenon. Additional sources include intratendinous vessels, the posterior tibial artery, and distal osseous and periosteal branches. A relative zone of hypovascularity exists within 2 to 6 cm proximal to the calcaneal insertion, corresponding to the site of most Achilles tendon ruptures and noninsertional tendinopathy.^{5,6}

Similar to other tendons, the Achilles is composed of predominantly type I collagen. Collagen fibrils are bundled into fascicles, which contain elastin, lymphatics, and neurovascular structures and are held together by the endotenon. The epitenon

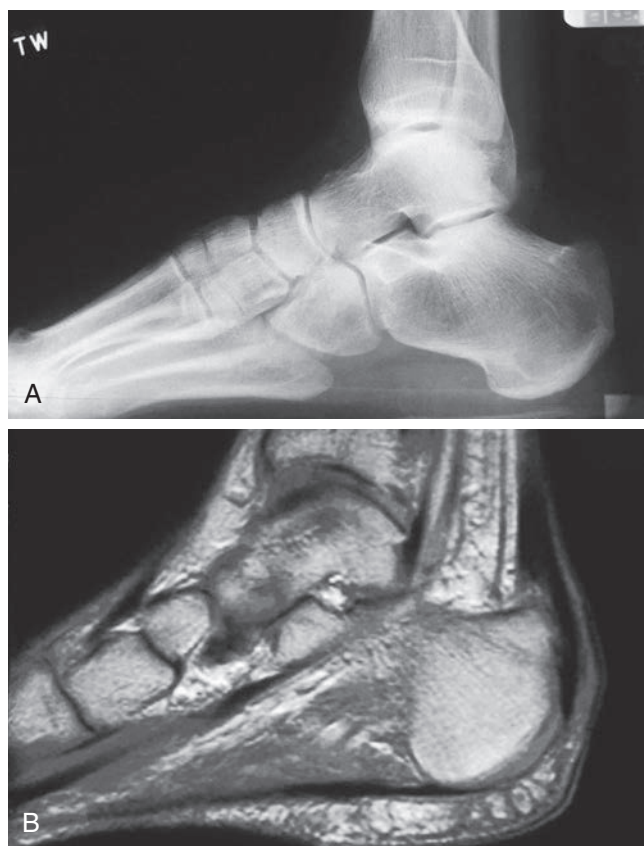


Fig. 9.1 (A) Lateral radiograph of calcaneus demonstrating Haglund's deformity. (B) Sagittal magnetic resonance imaging of the same patient showing changes at Achilles tendon from bony prominence and its effect on Achilles tendon, with thickening and fibrosis as it passes by the bone and more proximally.

surrounds the group of fascicles, forming the structural unit of the tendon. The paratenon further surrounds the epitenon and consists of an inner parietal layer, lying directly on the epitenon, and an outer, visceral layer. The paratenon, containing a small amount of fluid between its layers, facilitates glide and minimizes posterior adhesion formation.

The nomenclature used to describe tendon disorders has been confusing, as multiple terms are used to describe the same disease process. *Tendinitis* is the term that has traditionally been used to mean chronic pain and dysfunction of tendons with inflammation as the main underlying etiologic factor. However, histologic examination has demonstrated the absence of an acute inflammatory process or minimal inflammation as compared to what was originally anticipated.⁷⁻¹² On the contrary, several other studies have demonstrated that in the early stages of tendon damage, there is an acute phase of tendon injury that involves inflammatory cells.^{13,14} *Tendinosis* describes a chronic degenerative process within tendons that lacks an inflammatory process. The term *tendinopathy* encompasses a broad spectrum of acute and chronic disease processes affecting tendons. It is most likely that inflammatory and chronic degenerative processes both play integral roles during tendon pathology. For the purpose of this chapter, the term *tendinopathy* will be used to encompass acute and chronic degenerative stages of tendon pathology.

Achilles Tendinopathy

Achilles tendinopathy is common among athletes, affecting nearly 18% of runners.¹⁵⁻¹⁷ Repetitive impact-loading activities (overuse) such as jumping are responsible for the majority of cases.¹ Other predisposing extrinsic risk factors include overuse, training errors, environmental conditions, poor footwear, and improper training techniques (excessive running, sudden increase in intensity, uphill running). Other intrinsic predisposing factors include poor extremity biomechanics (foot pronation, cavus foot, genu varum), increased age, increased body mass index, gender, prior history of tendon disorders, medical comorbidities, and genetics.^{16,18} Another risk factor includes the previous use of fluoroquinolone antibiotics. Athletes commonly affected by tendinopathy are involved in running, jumping, dancing, tennis, racquetball, basketball, and soccer

ACHILLES TENDINOPATHY RISK FACTORS

Repetitive impact-loading activities
Abnormal lower-extremity biomechanics (foot pronation/supination, stiff joints, genu varum)
Improper training techniques (intensity, frequency, duration, speed, terrain)
Poor footwear selection
Fluoroquinolone antibiotics

Puddu et al.¹⁹ classified Achilles tendinopathy into three clinical categories. Peritendinitis is characterized by inflammation affecting the paratenon. Peritendinitis with tendinosis refers to both inflammation involving the paratenon and degeneration of the Achilles tendon. Tendinosis reflects Achilles degeneration. Clain and Baxter¹ later created an anatomic classification, separating tendinopathy into insertional disorders, affecting the area of the enthesis, and noninsertional disorders, commonly affecting the tendon 2 to 6 cm proximal to the calcaneus.

Whereas noninsertional tendinopathy occurs more often in younger, more active athletes, insertional Achilles tendinopathy develops more often in those athletes who are older, less active, and sometimes overweight. Although bilateral insertional tendinopathy will most likely be due to mechanical overuse, it may be associated with inflammatory disorders, including seronegative spondyloarthropathies.^{20,21}

Noninsertional Tendinopathy

Noninsertional tendinopathy can present with peritendinitis. The Achilles tendon itself is less involved. In chronic cases, adhesions may form between the paratenon and tendon, leading to more profound pain and tenderness. Pain is noted most often at the initiation of activity (start-up pain) and improves with continued exercise. Acute pain typically resolves with rest. In chronic cases, however, the pain may persist and significantly impair further athletic participation. On examination, a localized, increased diameter that more commonly affects the medial side is appreciated with palpation of the tendon. Tenderness, and at times crepitus, is noted throughout all ankle range of motion (Fig. 9.2). Radiographs generally are unremarkable.

Peritendinitis with tendinopathy represents further inflammation with associated intratendinous degeneration. Pain is



Fig. 9.2 Tenderness with squeezing the Achilles and crepitance with range of motion are hallmarks of peritendinitis. The discomfort related to peritendinitis will be constant in location as the ankle is brought through a range of motion. With Achilles tendinosis, the tenderness moves with the thickened tendon during range of motion.

more marked and constant. The tendon is thickened and infrequently has palpable intrasubstance calcifications (Fig. 9.3). The painful arc sign may help to distinguish between tenderness associated with peritendinitis and that associated with tendon degeneration. Tenderness related to peritendinitis will be constant in location as the ankle is brought through a range of motion, whereas tenderness associated with tendinopathy will change position with ankle motion.²²

Isolated tendinopathy, or noninflammatory atrophic degeneration, is associated with normal aging and typically is accelerated by overuse. Most affected are middle-aged, recreational athletes. With repetitive trauma, microtears develop within the tendon, mostly in the hypovascular zone, leading to further fibrosis and degeneration.²³ These athletes complain of weakness in push-off, with pain and swelling localized to the area approximately 2 to 5 cm proximal to calcaneus. Whereas ankle dorsiflexion commonly is limited, tendon elongation may develop with an associated increase in passive ankle dorsiflexion. Pathologic examination reveals fatty degeneration with disorganized collagen. Calcific deposits may be present

Insertional Achilles Tendinopathy

Insertional tendinopathy is an inflammatory reaction within the Achilles tendon affecting the enthesis, or tendon insertion onto the calcaneus. This disorder more commonly affects older, heavier, and less active athletes but can be seen in competitive athletes as well.²¹ An abnormally enlarged, bony prominence may aggravate this condition. There is a high association with Haglund's deformity and retrocalcaneal bursitis, but unlike these disorders, insertional tendinopathy involves the tendon itself. This most often results from chronic overuse and poor training habits. Improper techniques include inadequate stretching, rapid increase in training, running on harder surfaces, and heel running. Although pain initially follows exercise, particularly uphill running, symptoms may become more persistent despite activity modification.

Pain, swelling, and warmth are noted specifically at the enthesis. In athletes, there often is a localized area of pain with a small spur. Ankle range of motion is painful, with dorsiflexion typically limited because of a tight or painful Achilles tendon. External irritation from a shoe's heel counter plays less of a role in provoking symptoms in athletes with Achilles tendinopathy than in retrocalcaneal bursitis and Haglund's deformity. This is because the insertion lies within the center of the heel cup and tends not to get rubbed unless the tendinopathy extends more proximally near where the edge hits the shoe. Radiographs generally reveal calcifications or a bony spur at the most distal aspect of the Achilles insertion (Fig. 9.4A). Magnetic resonance imaging (MRI) will show thickening of the tendon with longitudinal striations of fibrosis or splits where the tendon attaches to the calcaneus. Often there will be localized bony edema in the calcaneus at this junction (Fig. 9.4B).

SURGICAL ALTERNATIVES

Paratendinitis: release paratenon
 Tendinopathy: degree of width involved <50%, ellipse and repair; >80%, ellipse and augment
 1–3 cm gap after debridement: V-Y
 3–5 cm gap after debridement: turndown
 >5 cm gap after debridement: turndown with consideration for FDL or FHL tendon transfer

FDL, flexor digitorum longus; FHL, flexor hallucis longus

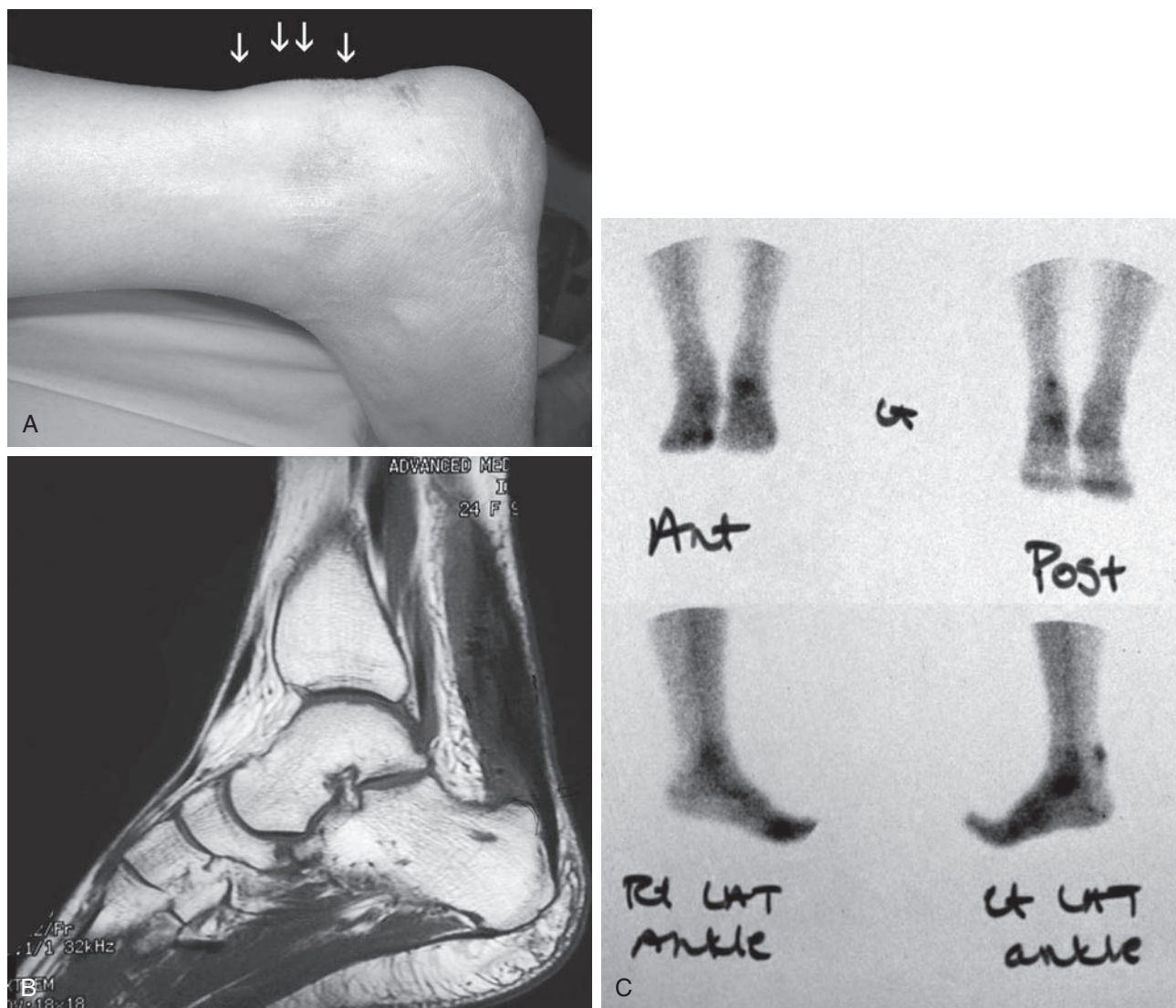


Fig. 9.3 (A) Noninsertional Achilles tendinopathy with characteristic swelling 2 to 5 cm above dorsal aspect of calcaneus. (B) Magnetic resonance imaging shows thickened Achilles tendon. A Haglund's deformity also is noted. (C) Technetium bone scan demonstrating increased uptake in the Achilles tendon; the third phase of the scan, indicative of advanced intrasubstance degeneration.

Haglund's Deformity

Haglund is credited with first describing the presence of a prominent posterolateral superior calcaneal tuberosity in 1927.² This enlarged superolateral tuberosity predisposes the precalcaneal bursa to be compressed between it and any tightly fitting shoe heel counter, possibly leading to skin irritation and inflammation. Because of the association of footwear, this disorder also has been referred to as a “pump bump” and “winter heel.” Although there is a frequent association with retrocalcaneal bursitis and insertional Achilles tendinopathy, Haglund's deformity initially generally does not involve the Achilles tendon.

Poorly fitting shoes in conjunction with this genetic bony prominence generally are responsible for the development of symptoms of Haglund disorder. Other predisposing risk factors include the presence of a cavus foot and hindfoot varus. In rare cases, childhood apophyseal trauma may be a cause. In the nonathletic population, repetitive injury or trauma may

result in bone overgrowth. Most affected are young women who wear fashionable high-heeled shoes. In the athletic population, we have observed this condition more commonly in males who participate in running sports. Long-distance runners are susceptible to this condition and the other Achilles tendon disorders (see Figs. 9.1 and 9.5).

On examination, the affected heel has a swollen, red, and tender posterior prominence, predominantly on the lateral side of the calcaneus. The Achilles tendon itself is not tender. Numerous radiographic measurements have been used to quantify the size of the posterosuperior prominence. These techniques generally are not used by orthopaedists as they do not always correlate with the clinical findings.

Retrocalcaneal Bursitis

Retrocalcaneal bursitis refers to inflammation affecting the bursa immediately anterior to the Achilles tendon. As with Haglund's deformity and Achilles tendinopathy, this condition

is common in running athletes. In the general population, as with insertional tendinopathy, those most commonly affected are older, less active recreational athletes. As the disorder becomes chronic, the bursa enlarges and may become adherent to the Achilles tendon. A prominent posterosuperior bony projection may be present.

Athletes typically complain of pain with activities that force the ankle into dorsiflexion, particularly uphill running, and thereby compress the inflamed bursa between the posterosuperior calcaneus and the Achilles tendon. Schepesis et al.²³ described the two-finger squeeze test, in which pain is noted when two fingers compress medially and laterally immediately superior and anterior to Achilles insertion. This area will be warm with a notable soft-tissue bulge. Pain is elicited with passive dorsiflexion. Radiographs may show a subtle soft tissue fullness and loss of the retrocalcaneal soft-tissue shadow, as well as the presence of a posterosuperior bony prominence. MRI demonstrates the bursal enlargement anterior to the Achilles tendon above its insertion in the retrocalcaneal region (Fig. 9.6).

TREATMENT OF ACHILLES TENDINOPATHY

Nonsurgical Treatment

The treatment for Achilles tendinopathy is nonoperative at first with surgical intervention for recalcitrant cases. Initial treatment should include anti-inflammatory medications, gentle Achilles stretching and activity modification. Relative rest with limitations on intensity, duration, or frequency of training and concomitant institution of nonstressful cross training (exercise bike, pool running, elliptical trainer) should be helpful. An open-back shoe may benefit those with no heel counter pressure (Fig. 9.7). A padded heel sleeve can be comfortable. If there is no response, a heel lift (one-fourth to three-eighths inch), night splint, or temporary immobilization in neutral or slight plantarflexion with a removable walking boot or cast may be required. If the athlete has notable foot pronation, a semirigid orthotic may improve overall foot biomechanics by supporting the medial arch.

Intratendinous corticosteroid injections should be avoided because local use of these injections has been associated with tendon attrition and potential rupture. Although there is no strong evidence of similar deleterious effects after paratendinous corticosteroid injections, there are similar worries with an injection in the bursa. It would be advisable to immobilize the ankle temporarily after a retrocalcaneal injection because the retrocalcaneal bursa has a direct communication to the Achilles and may make it structurally vulnerable.^{21,24–26} In general, we advise against corticosteroid injections.

For refractory peritendinosis we have found that brisement may provide symptomatic relief in a third to half of total cases.²⁷ Brisement consists of injecting 5 to 10 ml of sterile saline or local anesthetic agents into the Achilles tendon sheath; this may forcibly disrupt any adhesions between the paratenon and Achilles tendon. Repeating the injections two to three times over several weeks may be necessary to achieve success.^{23,27}

After initial symptoms resolve, it is imperative to correct predisposing factors, including improper technique, excessive training, inappropriate footwear, and poor flexibility. In

addition, it may be critical to temporarily or permanently eliminate provocative, more rigid, and less compliant surfaces and terrain.

Reported results of nonoperative treatment of insertional and noninsertional Achilles tendinopathy have been generally successful. Studies have found that 70% to 90% of patients have found symptomatic improvement after corrections in their footwear, training habits, and mechanics.^{16,27–31} There are, however, fewer predictable results with nonsurgical management in those with chronic tendinopathy and in the older athlete, as a result of greater degenerative tendon involvement.³⁰

In our experience, athletes with isolated Haglund's deformity can be managed with footwear modification about 50% of the time. The presence of a prominence does not mandate surgery. We have seen improvement in about 30% of patients with retrocalcaneal bursitis and in about 25% of those with insertional tendinopathy.

Extracorporeal Shock Wave Therapy

The use of extracorporeal shock wave therapy (ESWT) for the treatment of Achilles tendinopathy has not been conclusively studied. The most information on shock wave therapy comes from upper extremity tendinopathy (e.g., tennis elbow) and plantar fasciitis. Shock wave therapy works by creating a pressure change that propagates rapidly through a medium. When transmitted through a water medium, it can either directly create high tension at a given structure or indirectly create

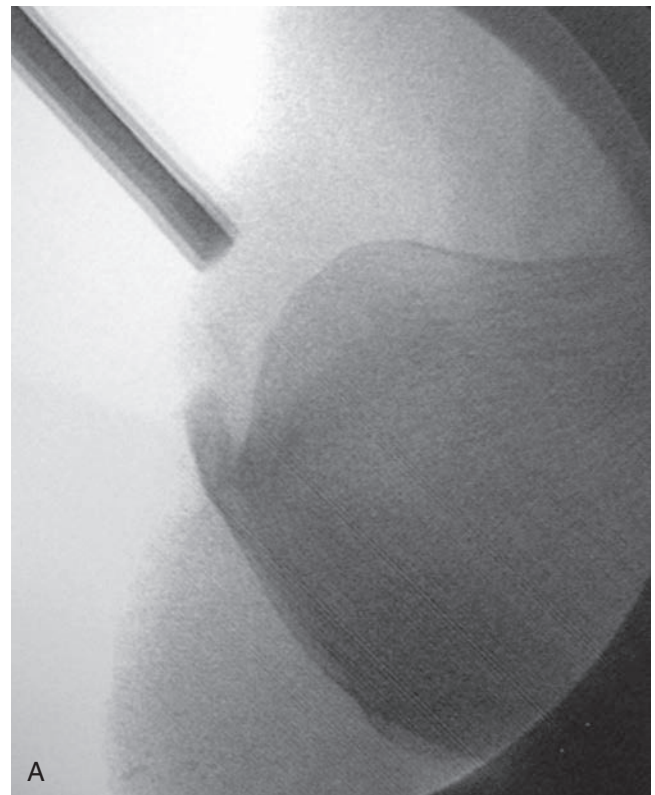


Fig. 9.4 (A) A lateral x-ray and (B) a series of sagittal magnetic resonance images of the same patient with insertional degeneration of the Achilles tendon with tendon thickening and fibrosis from about 2 cm proximal to its insertion on the calcaneus.

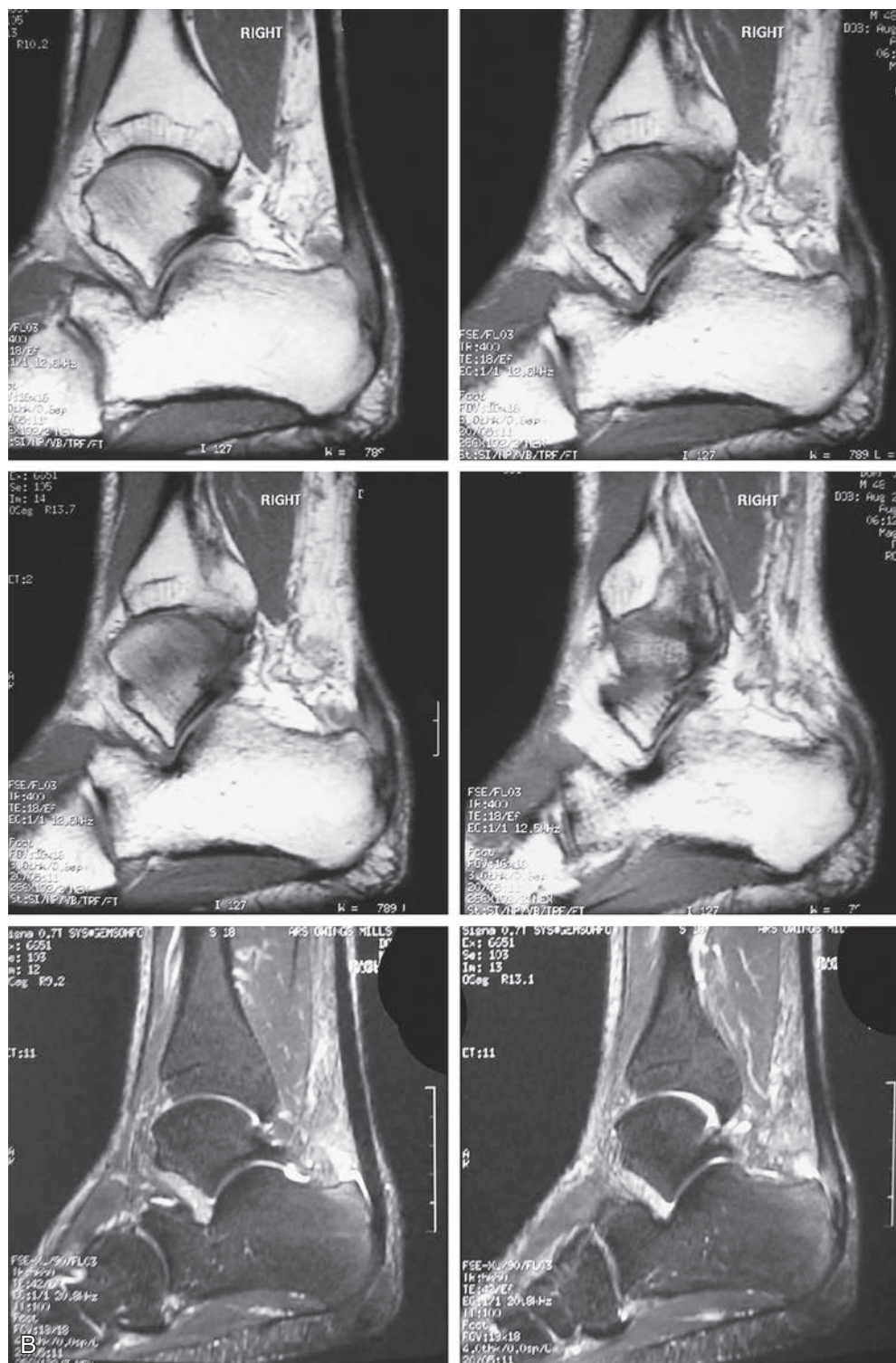


Fig. 9.4, cont'd.

microcavitations. Theories behind its analgesic effect in orthopedic applications include an alteration of the permeability of neuron cell membranes and induction of an inflammatory-mediated healing response by increasing local blood flow.³² Studies have also shown that ESWT decreases tenocytes expression of matrix metalloproteinases and interleukins.³³ Studies on ESWT on Achilles tendinosis have shown a success rate of approximately

30%–40%.^{34,35} A systematic review by Al-Abbad et al.³⁶ demonstrated evidence for the effectiveness of ESWT in the treatment of chronic insertional and noninsertional Achilles tendinopathies with a minimum of 3 months follow-up before undergoing surgery. Furthermore, they demonstrated that ESWT in conjunction with eccentric loading shows superior results. In our experience, we have found a success rate of approximately



Fig. 9.5 Clinical photograph of Haglund's deformity.



Fig. 9.6 Magnetic resonance imaging demonstrates Haglund's deformity with enlarged posterior superior aspect of the calcaneus.

50% in athletes. Even with this lower success rate, we try ESWT and then wait 3 months before surgery given the minimal side effects. Depending on the immediate results, we may allow sports play with only 1 or 2 weeks off. If the athlete is in midseason, then this modality is his or her best chance to resume play. If the athlete is at the end of the season, then we may try shock wave therapy and a boot brace for 2 to 6 weeks and then allow the athlete to resume impact activities. After the season, when there is more time for recovery, decisions regarding further treatment can be made. Contraindications to ESWT quoted in the literature include pregnancy, coagulopathies, bone tumors, bone infection, and skeletal immaturity.³²

Saggini et al.³⁷ noted successful outcomes after two treatments with no complications using shock wave therapy on

Achilles tendinopathy. Several later studies reported promising results after ESWT with those affected with chronic Achilles tendinopathy.^{38–40} The cost of shock wave treatment can be an important consideration because the therapy may not be covered by insurance. With lower-energy shock wave machines, three treatments are used, at a total cost of \$500 to \$1500. The higher-energy machines usually require one treatment (\$1000 to \$3000) but this typically involves a regional or general anesthesia given the magnitude of the pain. Authors (LCS and JPT) do not generally recommend ESWT for those patients with bony prominences (e.g., Haglund's or large insertional spurs).

Autologous Platelet-Rich Plasma, Bone Marrow Concentrate, Allogenic Factor Injection, and Other Biologics

Injection of autologous platelet-rich plasma (PRP), bone marrow concentrate or allogenic factors—derived from decellularized placental/chorion/amniotic tissue—can also be considered for resistant cases (See Chapter 29). For the PRP, patients need to have their blood drawn and equipment is needed to process the blood and harvest the platelets via centrifugation or filtration. Costs can be between \$500 and \$2000. The techniques of the platelet isolation and the subsequent quantity of platelets and white blood cells (WBCs) vary. Clinical studies have not been able to demonstrate a clinically significant improvement of tendon healing with PRP injection.^{41,42} However, platelet-rich plasma has been shown to stimulate cell proliferation, enhance the activity of growth factors like vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), and increase total collagen production in a controlled laboratory study.⁴³

For the bone marrow concentration, the patients require some pre-medication and may benefit from sedation in the operating room or procedure room. The bone marrow is harvested in 5ml aliquots via multiple trajectories from the iliac crest. Typically a specialized chamber is used to perform centrifugation to separate the red blood cells (RBCs) from the plasma and the platelet rich cellular buffy coat. Unlike in PRP, the concentrate contains endothelial progenitor cells, mesenchymal signaling cells, hematopoietic stem cells, and other progenitor cells in addition to the platelets. For the allogenic factor injections, the materials and methods also are diverse. For both, the method of injection and post injection rehabilitation vary as do the results in the literature. When there is a bony element that causes a mechanical prominence (e.g., large Haglund's or spurring in the insertional Achilles tendinopathy) the results can be less than 30% successful. If there is only the bursitis or tendinopathy, the results can be up to 75% for PRP or growth factors or 90% for bone marrow concentrate in the authors' (LCS and JPT) experience.

Surgical Treatment

Surgical intervention is considered after approximately 3 months of nonoperative treatment measures. The surgical technique is chosen on the basis of location of pathology. For a symptomatic athlete with a normal tendon, determined by

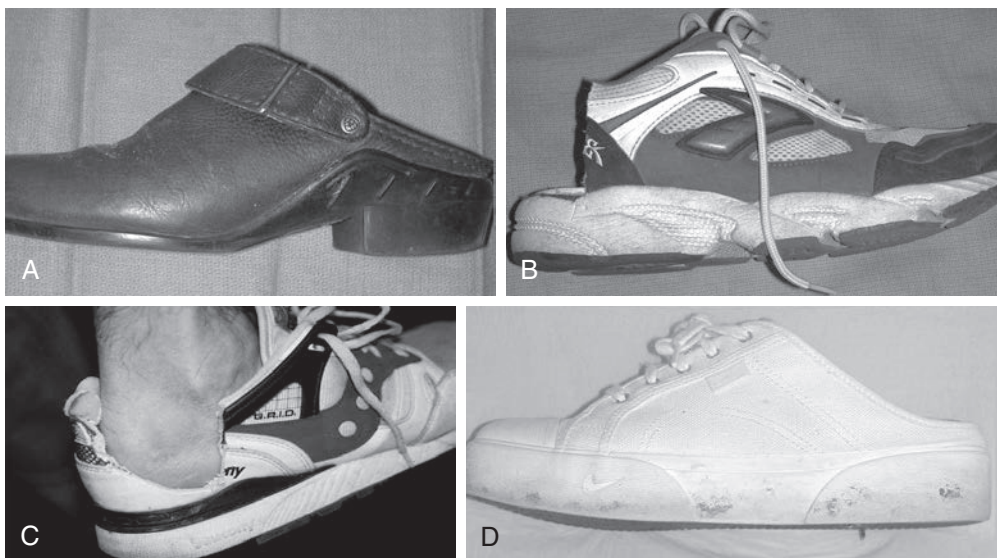


Fig. 9.7 Shoe Alternatives and Modifications for Patients with Achilles Tendinopathy. Clockwise from the top left: (A) A higher-heeled, backless shoe, “a mule.” (B) A completely removed heel counter. (C) A partial heel counter cut to relieve external pressure. (D) A backless sneaker.

physical examination and possibly by MRI, we generally try to avoid a procedure that may irritate or traumatize the tendon. A medial or lateral approach 5 to 10 mm anterior to the Achilles tendon and paralleling its course is best in these cases because it runs through thicker skin and subcutaneous tissues. If the tendon is involved, this approach also provides excellent access to the tendon with the creation of full thickness flaps down to the level of the paratenon. Debridement of the tendon depends on the location of the clinical and radiographic findings – above, at, or below the insertion of the tendon. In general, the points of tenderness dictate where the exposure must occur. Thus, if there is more medial and central Achilles tendinopathy, the tendon in the medial and central aspect must be elevated off the calcaneus, the tendon must be debrided there, and the underlying bone must be resected and recontoured.

Noninsertional Tendinopathy

For noninsertional tendinopathy, the choice of procedure is based on whether the disease involves the paratenon, tendon, or both. In peritendinitis, all adhesions are excised, and the surgeon also performs a limited resection of any thickened paratenon. The extremity is immobilized for 3 to 5 days, followed by a range-of-motion program to limit the recurrence of scar formation (Fig. 9.8).

When there is tendinopathy and peritendinitis, elliptical excision of the tendon and longitudinal paratenon release is performed. Maffulli et al.⁴⁴ have reported a success rate of approximately 70% after percutaneous longitudinal tenotomy of the middle third of the Achilles tendon. In this technique a no. 11 or no. 15 blade is introduced posteriorly through the skin and tendon. With the blade held stationary, the ankle is dorsiflexed, and the tendon moves, allowing it to be cut longitudinally. Next the blade direction is reversed 180 degrees and the ankle is plantarflexed. The process is repeated through four additional incisions in the zone of the degenerative tendon (Fig. 9.9). This creates a controlled tendon trauma and triggers a healing response.



Fig. 9.8 Release of the Paratenon. Note that the incision is made 1 cm anterior to the margin of the Achilles tendon. The incision can be made much smaller than shown in this case.



Fig. 9.9 The Maffulli Technique. An incision is made with a no. 11 or no. 15 blade. The blade is held stationary and the ankle is dorsiflexed creating a longitudinal cut in the tendon. The blade then is turned 180 degrees, and the ankle is plantarflexed. The process is repeated until there are five longitudinal cuts.

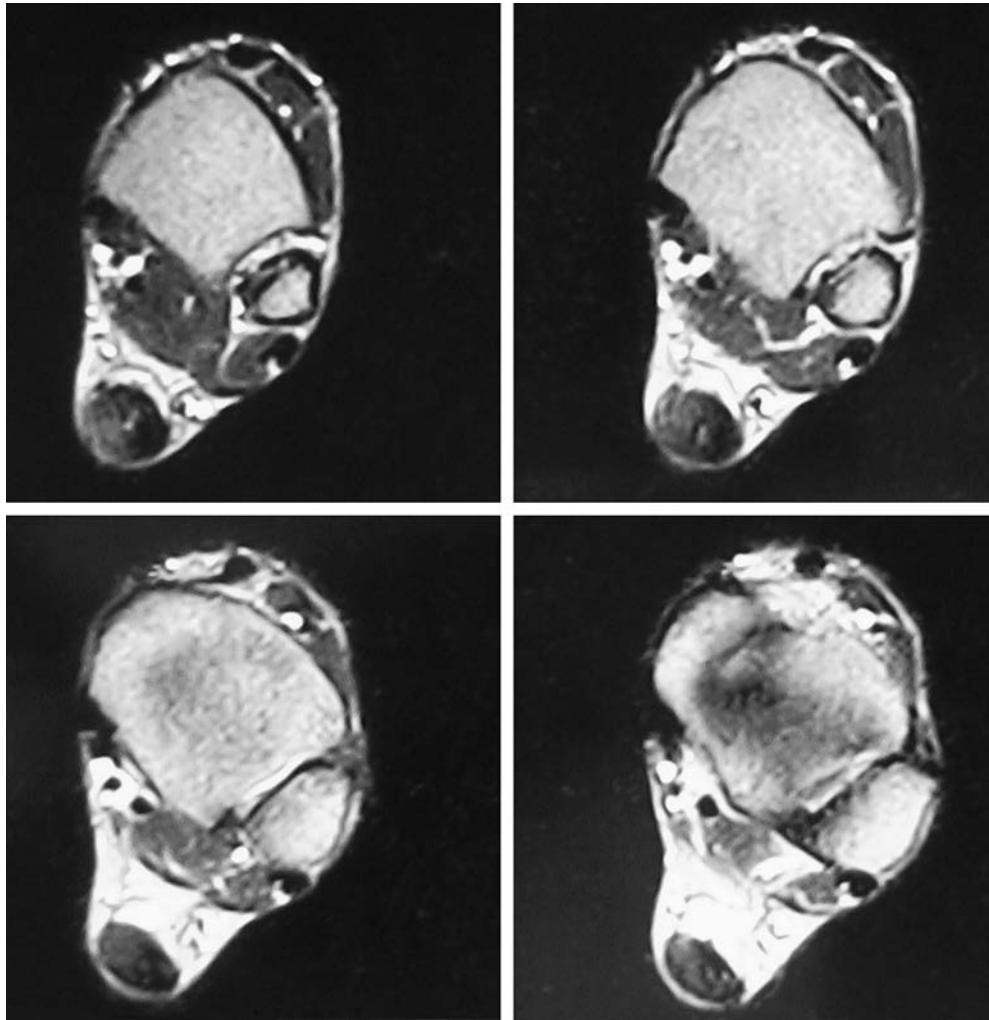


Fig. 9.10 Magnetic resonance imaging cross section of the Achilles tendon demonstrating more than 80% tendon involvement. This would indicate the need for tendon augmentation or transfer following debridement.

The type of procedure chosen for treatment of tendinopathy depends on many factors, the largest, in our experience, being the extent of tendon involvement, determined by clinical findings, ultrasound, or MRI. When less than 50% of the tendon is involved, we longitudinally ellipse the diseased tendon; when more than 80% of the tendon is involved, a debridement and tendon augmentation (e.g., turndown) or transfer is recommended (Fig. 9.10). When there is between 50% and 80% involvement, the decision is determined by the patient, the sport, and the surgeon's preference.

Debridement of tendon. For tendinopathy, typically the degenerative portion of the Achilles tendon is debrided and the paratenon is released. If less than 50% of the tendon width is debrided, then the remaining section of intact longitudinal tendon should be strong enough to withstand stresses.

Typically, a medial incision is made just anterior and parallel to the border of the tendon that is thickened, and the paratenon is entered. On the basis of maximal tenderness, MRI, or ultrasound localization of the degenerative zone of the tendon, an elliptical longitudinal excision of the diseased tendon is performed, leaving intact the anterior and posterior surfaces of the tendon. Essentially the zone of ellipsed tissue should include

the degenerative fibers and the thickened tendon (Fig. 9.11). The tendon then is repaired with internally placed, nonabsorbable sutures with buried knots. The subcutaneous tissues are apposed, followed by closure of the skin. The leg is immobilized for 3 to 5 days in a splint, followed by range-of-motion exercises, strengthening, and nonimpact activities. A boot brace is worn for 6 to 12 weeks during ambulation to unload the healing tendon. Jogging and running may be introduced at 3 months, depending on the extent of involvement and the nature of the patient's athletics.

Tendon transfer. If more than 50% of the tendon width is involved, then one must consider the risks and benefits of either longitudinal tenotomy, debridement, or tendon transfer. The decision to consider tendon transfer is determined by the structural weakening of the tendon that may result from a large debridement.

Because most athletes use all their tendons for ultimate, lower-extremity performance, it is difficult to justify harvesting a working structure to improve the function of the Achilles. Thus, depending on the demands of the athlete and nature of his or her skills, we must balance the pros and cons of using the tendon transfer. If 50% to 80% of the width of the tendon is resected, we

consider these factors. However, if 80% or more of the tendon is involved, our experience has been that the transfer becomes more critical to restore function. Alternative procedures in this latter scenario include a turndown procedure, tendon allograft, and V-Y advancement.

The flexor hallucis longus (FHL), flexor digitorum longus (FDL), or, less commonly, the peroneal tendons can be transferred. Authors (LCS and JPT) prefer to use the FDL tendon in a nonsprinting athlete, nondancer, or rock climber.⁴⁵

Transferring the FHL or any other tendon in a sprinting athlete or ballet dancer could lead to loss of agility, power, or balance. In these athletes, it is better to consider performing a turndown procedure, a V-Y advancement, or an allograft if a wide area of tendon is involved.

The patient can be positioned prone or supine and both legs are prepped for any tendon transfer, turndown procedure, or V-Y advancement because it usually is necessary to compare resting tensions with those of the contralateral side.



Fig. 9.11 Intraoperative photo of debridement of the Achilles tendon. Once the degenerative tissue is ellipsed from within the substance of the tendon, buried sutures are used to close the defect.



Fig. 9.12 After debriding the distal Achilles tendon, an incision is made distal to the medial malleolus over the neck of the talus. The flexor digitorum tendon is identified just inferior to the posterior tibial tendon.

Achilles debridement and FDL transfer. Patients are placed in a semi-lateral position, with the operative side down, to allow access to both the posterior and medial aspects of the foot and ankle. Patients who have had prior surgery on the Achilles tendon through a posterior incision were placed in the prone position. Surgeries are performed under regional block with general sedation. The surgeons (LCS and JPT) caution not to use the tourniquet.

Exposure of the diseased Achilles tendon is done via a posteromedial approach, except in patients who had prior midline posterior approach. Dissection is carried out sharply down to the Achilles tendon with minimal undermining distally to preserve a full-thickness flap for closure. The Achilles is debrided. If indicated, the insertion of the Achilles and/or the posterior superior prominence of the calcaneus is resected.

A 1–3-cm separate incision is made longitudinally over the medial aspect of the hindfoot just over the proximal medial neck of the talus. After the posterior tibialis tendon (PTT) sheath is identified, the dissection is carried down inferior to the PTT to identify the FDL tendon. The right-angle clamp is used to tension the FDL tendon to confirm that the appropriate tendon is found. (Fig. 9.12.)

Another small separate incision is made 6–10 cm above the tip of the medial malleolus at the posterior edge of the tibia. After the posterior compartment sheath is open, the FDL tendon and muscle are identified and freed. By pulling on the FDL distally, the FDL proximally can be found. (Fig. 9.13.) Then the FDL tendon is transected under direct visualization with the toes in maximum plantarflexion at the medial hindfoot incision. The FDL stump is carefully retracted through proximal tibial incision leaving the FDL muscle belly on the tendon. (Fig. 9.14) Tunneling is created from the tibia incision through the subcutaneous tissue above the superior retinaculum down to the Achilles tendon insertion. (Fig. 9.15) Finally, the end of the FDL tendon is fixed to the calcaneus using a 5.0 mm anchor and the Achilles tendon is repaired with a nonabsorbable suture and a 5.0 mm anchor under appropriate tension with 20 degrees of ankle plantarflexion.



Fig. 9.13 Just posterior to the medial tibia, the flexor digitorum longus is found 6–10 cm proximal to the medial malleolus.

The wounds are closed in a layered fashion, and the leg is placed in a bulky plaster-reinforced splint with the foot in resting plantarflexion for 2 weeks. Postoperatively, patients are kept nonweight-bearing in a boot for 2 to 6 weeks and then advanced to weight bearing in the boot as tolerated until 8 to 12 weeks.

Achilles debridement and FHL transfer. In the FHL tendon transfer, our preferred technique is prone with a medial approach to the Achilles tendon, typically staying 1 cm anterior to the medial edge of the tendon. The incision is extended more inferiorly and centrally over the posterior calcaneus. The paratenon is opened, the degenerative tendon is excised, and the deep fascia between the superficial and deep compartment is released. It is felt that, by opening the fascia and exposing the deeper FHL muscle belly, there is an improved vascular bed for the Achilles. Ranging the big toe should facilitate identification of the moving FHL muscle belly and tendon. The FHL tendon may have a more distal origin and may not be viewed readily in the wound. Care should be taken while dissecting along the course of the muscle because the tibial nerve runs immediately medial to the tendon (Fig. 9.16A–E).

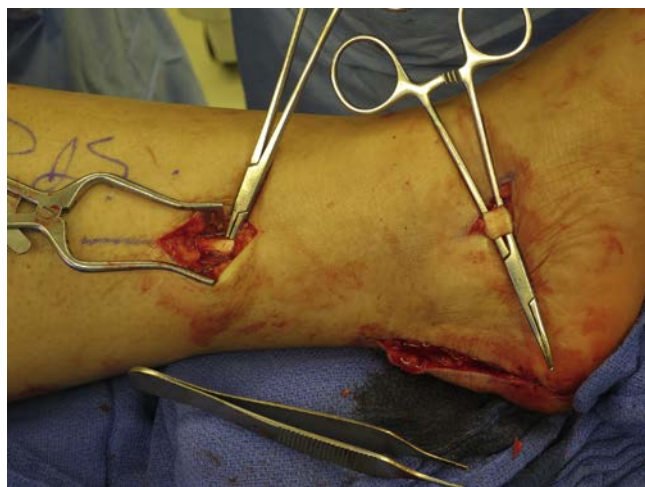


Fig. 9.14 The flexor digitorum is found proximally with the assistance of pulling on the tendon distally.

Follow and release the FHL tendon from the sheath (fibro-osseous tunnel) as it travels between the medial and lateral tubercles of the posterior talus. Continue to release the tendon for as much length as possible from the posterior approach, dissecting toward the underside of the sustentaculum tali. Cut the tendon as distally as possible, again avoiding the tibial nerve. Alternatively, the FHL can be harvested with the patient prone or supine from the medial midfoot with a small longitudinal incision just inferior to the naviculocuneiform joint in order to obtain more length for transfer. Once the FHL tendon is harvested, it can be sewn to the Achilles repair or inserted into the calcaneus or its periosteum, depending on tendon length. A useful technique involves drilling a hole the width of the tendon (typically 5 mm) through the calcaneus from dorsal to plantar (Fig. 9.17). A small incision made over a K-wire passed through this tunnel can facilitate placement of a small-bore suction tip over the wire from plantar to dorsal and out the planned entry point for the tendon. The whip suture in the FHL tendon then can be passed through the suction tip and pulled plantarly to permit tensioning. An interference screw can be inserted through the tunnel. Alternatively, an anchor can be placed obliquely in the tunnel wall just distal to the opening but not obscuring the passageway. After the proper tension has been determined, the tendon is secured. Occasionally there is a need to resect the posterior superior calcaneus; this procedure is determined by the presence of Haglund deformity and bursitis. We do not close the deep fascia between the compartments because the FHL muscle belly may provide for improved healing following the Achilles repair.

V-Y advancement. A V-Y advancement may be required if more than 80% of the tendon width and 2 to 3 cm in length is involved. With this large amount of tendon involvement, the remaining normal tendon may not be thick or wide enough to safely flap. The V-Y advancement is accomplished by extending the initial posterior incision more proximally toward the musculotendinous junction (Fig. 9.18A–C). A V-shaped fascial incision is made with the apex proximal. With traction on the tendon distally, an advancement of 2 to 3 cm then can be achieved; this should close the distal gap sufficiently. The distal repair can

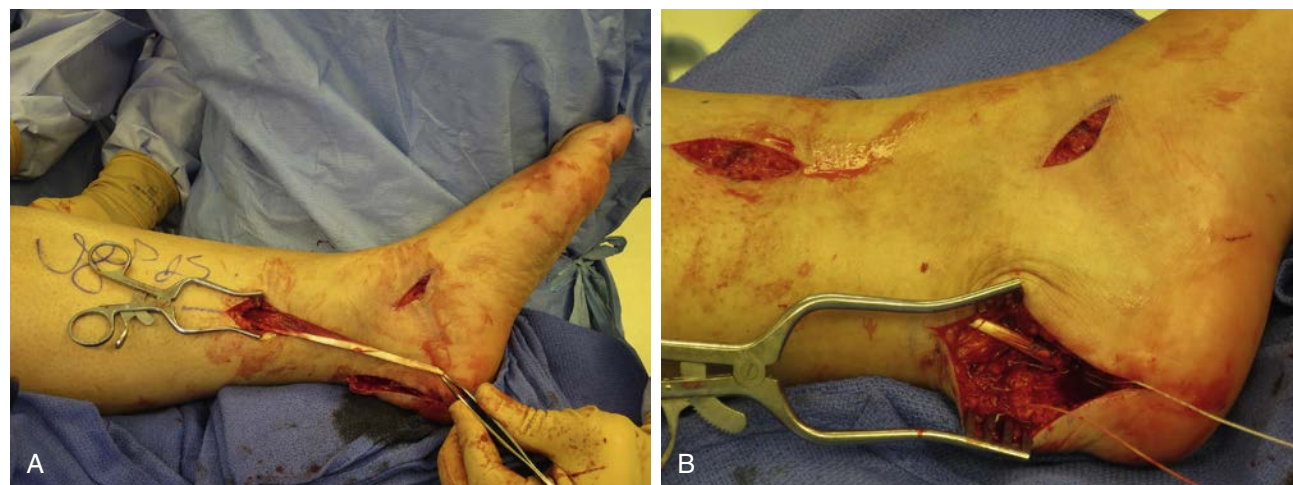


Fig. 9.15 A. The flexor digitorum longus is delivered proximally. B. The flexor digitorum longus is passed deep to the subcutaneous fat but superficial to the tarsal tunnel and secured to the dorsal medial calcaneus.

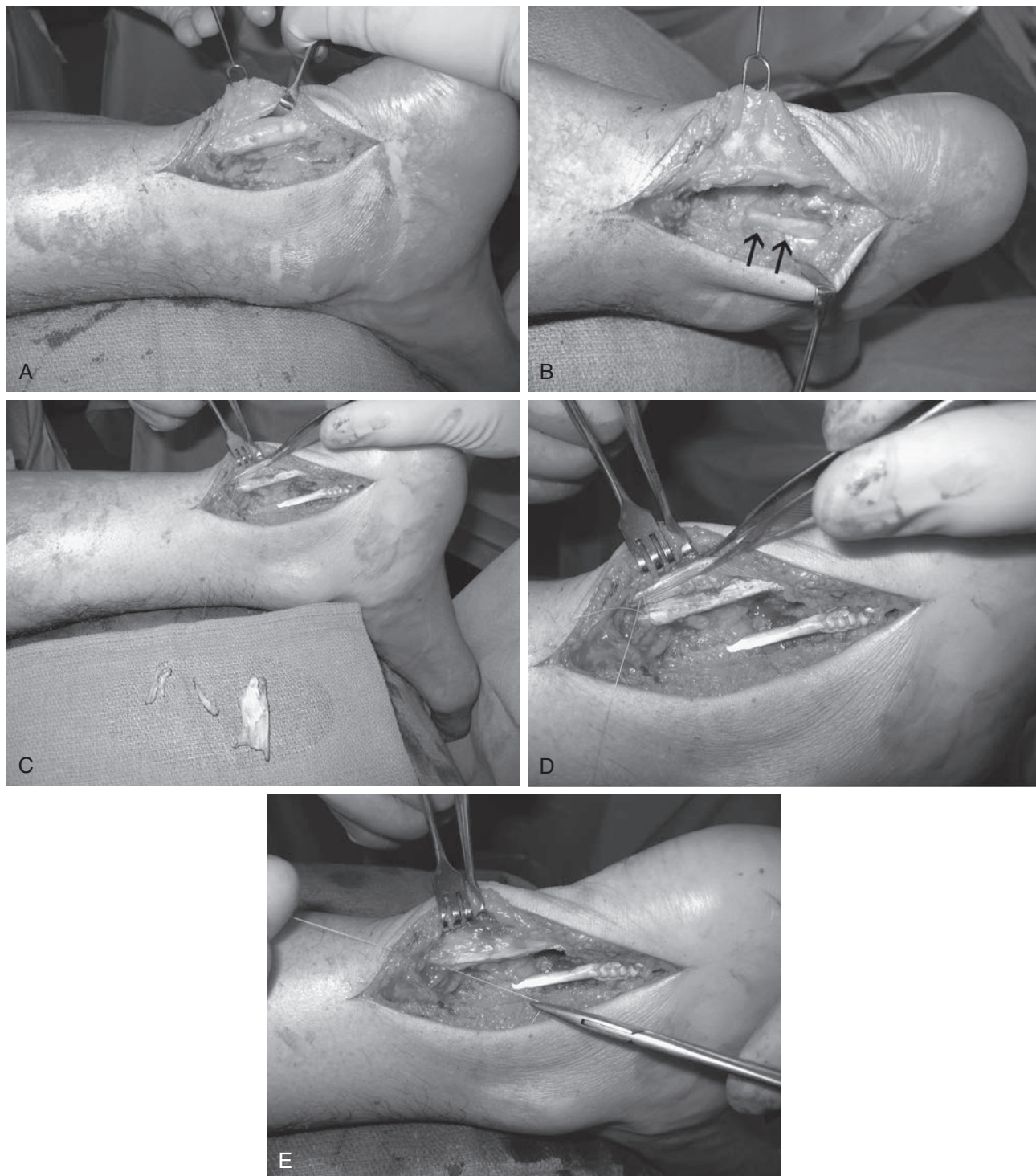


Fig. 9.16 (A) A medial approach 1 cm anterior to the medial edge of the Achilles tendon. (B) The deep fascia between the superficial and deep compartment is released. Ranging the big toe should allow palpation and identification of the moving flexor hallucis longus (FHL) (*two black arrows*). The tibial nerve runs immediately medial to the tendon; therefore dissection of the tendon must be carefully performed. (C) The tendon is released distally and secured with a whipstitch. The degenerative Achilles tendon is excised. (D) 4-0 or 2-0 nonabsorbable suture is buried within the tendon. (E) The defect is closed and the FHL tendon is sewn to an anchor into the calcaneus. This area of the calcaneus is prepared by locally elevating the periosteum. In this case the Achilles tendon length was normal, so the FHL was tensioned to permit full dorsiflexion.

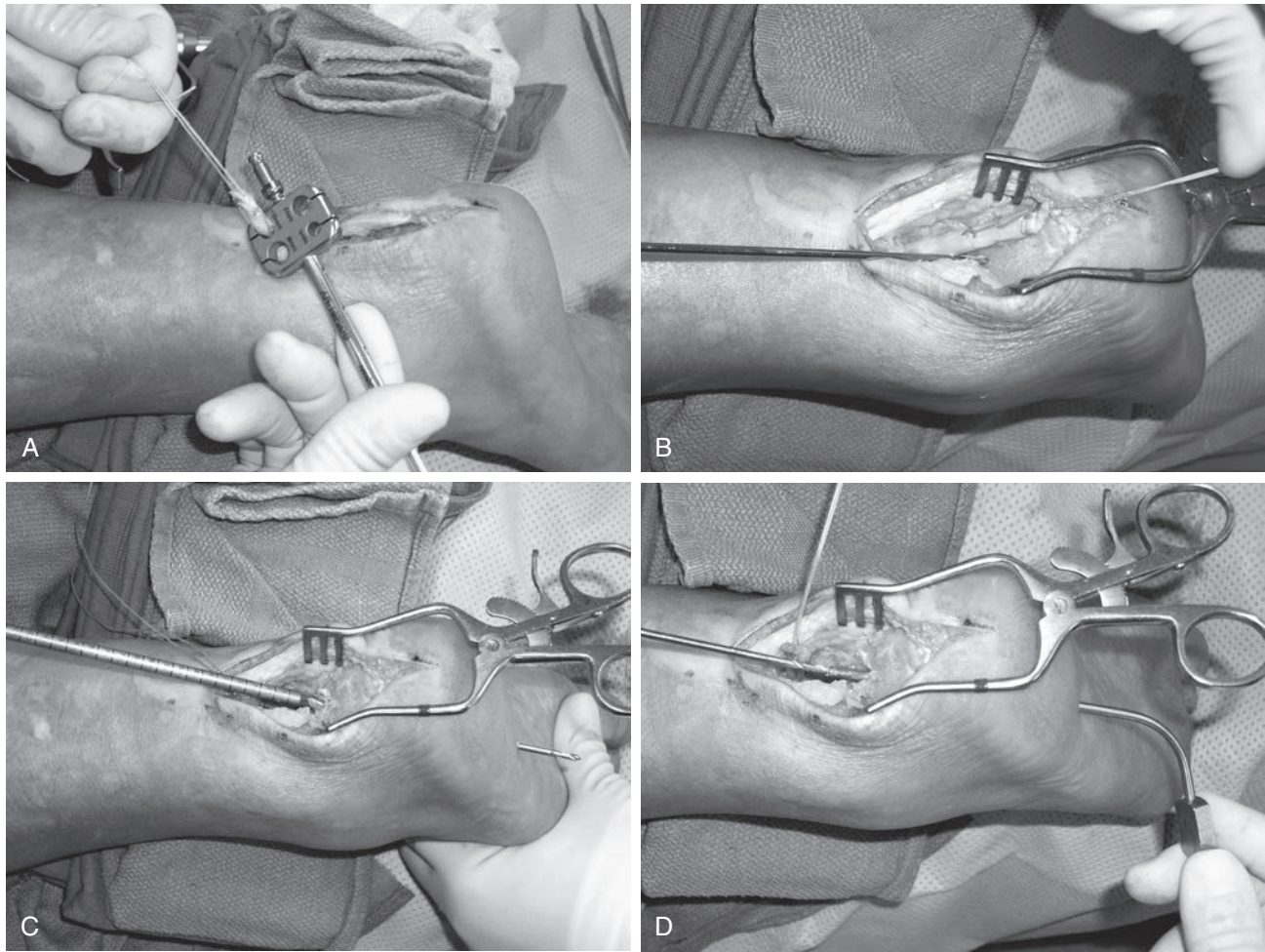


Fig. 9.17 (A) Following debridement of the Achilles and harvest of the flexor hallucis longus (FHL), the thickness of the FHL is determined to properly select the drill size. (B) A guidewire is passed through the calcaneus and then is advanced to pierce the plantar soft tissues. The exit point plantarly is just anterior to the fat pad of the heel. (C) A drill matching the width of the FHL is used to create a channel in the calcaneus but should not penetrate the soft tissues. (D) A small incision is made and a small-bore suction tip is placed over the wire from plantar to dorsal and out the planned entry point for the tendon.

be performed with a modified Krackow or whipstitch, and then balancing of the tendon tension is performed by checking for the resting posture of the foot and testing the “springiness” of the foot as it sits in the normal, slightly plantarflexed position.

The V-Y advancement can be performed in the supine or prone position. Because it usually is necessary to compare resting tensions with those of the contralateral side, both legs are prepped as for any tendon transfer or turn-down procedure. However, the tendon may begin to tear and pull off the muscle base beyond an advancement of 3 to 5 cm. If it appears that there is not enough fascia/tendon substrate or if too large a defect exists for advancement, then a turn-down or an allograft tendon transfer such as with a semitendinosus tendon may be used as a connecting bridge.

Turn-down procedure. A turn-down procedure provides substrate for healing and may limit the possibility of rerupture when there is between 50% and 80% of tendon width involvement. The patient is positioned prone with both legs prepped to compare the two and recreate normal resting tension. A medial incision is used, with care taken on deeper exposure to avoid branches of the sural nerve. After the rupture or degeneration site is exposed,

the end of the proximal tendon is mobilized, then grasped with Alice clamps and gently distracted by pulling distally on the Alice clamps for 5 to 10 minutes (Fig. 9.19A).

The size of the gap is measured while the foot is maintained in a neutral position (Fig. 9.19B). An additional 4 cm then is added to the tendon defect (a 2-cm distal hinge that is overlapped by the turned-down flap, or 2 cm plus 2 cm). Another 1 cm is added to account for the intended 1-cm overlap of the tendon ends distally (Fig. 9.19C). Thus, the flap begins proximally at a point 5 cm more than the size of the gap. For example, if the gap is 6 cm, then a flap is initiated 11 cm proximal to the gap (Fig. 9.19D). A strip of tendon approximately 1 cm wide and 1 cm thick is harvested centrally. The tendon graft now can be turned distally to span the void (Fig. 9.19E–F). At approximately 2 cm proximal to the defect, two no. 1 Ethibond sutures are used to anchor the corner of the turned-down graft, reinforcing the high stress junction so there is no propagation of the split between the strip and the main body of the tendon (Fig. 9.19G). To decrease the bulk created by this method, the tendon then is passed anteriorly deep to the tendon instead of posteriorly.

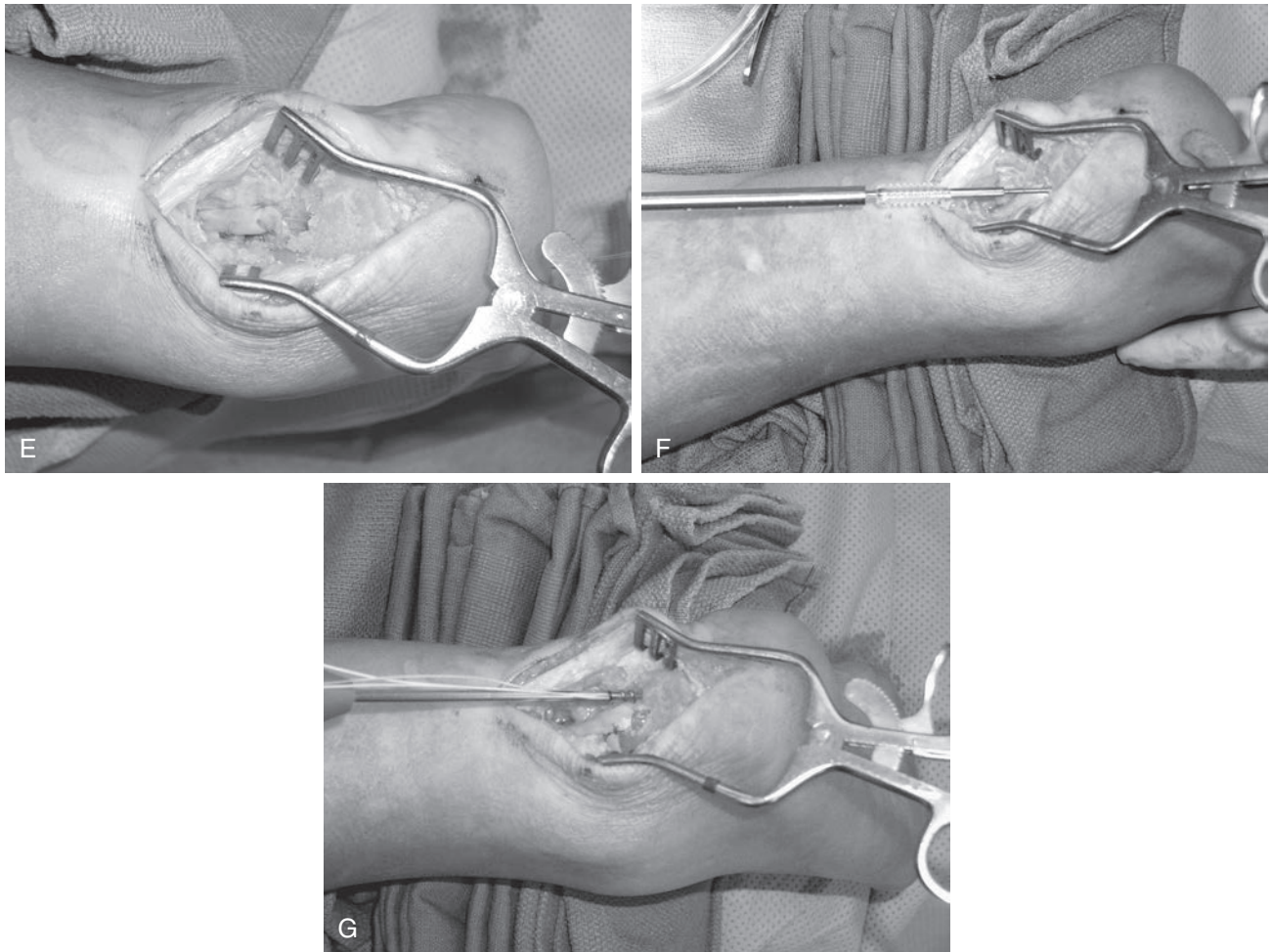


Fig. 9.17, cont'd. (E) The whip suture in the FHL tendon then can be passed through the suction tip and pulled plantarly to permit tensioning. (F) An interference screw is inserted through the tunnel. As an alternative, an anchor can be placed obliquely in the tunnel wall just distal to the opening but not obscuring the passageway. (G) A corkscrew anchor is inserted to repair the Achilles tendon onto bone.

The distal tendon end then is secured to the remaining viable Achilles or to the bone. Tensioning of the graft requires checking the range of motion and the springiness of the operative side versus the normal side. Usually, the foot should have a resting position of 15 degrees of plantarflexion. The graft and the turned-down flap are held in place by hand or by suture. Once the appropriate tension and position are established, whipstitches are used for final anastomosis. The resting tension and springiness are checked once again at the end of the procedure.

NONINSERTIONAL TENDINITIS SURGICAL ALTERNATIVES

- Paratendinitis: release paratenon
- Tendinosis: degree of width involved
 - <50% ellipse and repair
 - >80% ellipse and augment
- Tendinosis: degree of length involved
 - 1–3 cm V-Y
 - 3–5 cm turndown
 - >5 cm turndown with consideration for FHL or FDL tendon transfer

Insertional Tendinopathy

Insertional tendinopathy is surgically treated by excising the retrocalcaneal bursa and any prominent posterosuperior bone. Additionally, the Achilles tendon is debrided of any intratendinous calcifications and degenerated tissue, including detachment of part of the tendon's insertion. Achilles tendon reattachment through calcaneal bone tunnels or with suture anchor is advised. A central splitting, paralateral, and paramedian Achilles approaches have been advocated.²¹ Our preference is a paramedian approach with full thickness flaps down to the paratenon which facilitates excellent visualization while avoiding a wound directly over the Achilles tendon and the relatively thin skin in this location (Fig. 9.20A–H). The direct posterior central approach affords optimal visualization, but risks putting the incision under tension and through thinner skin found proximal to the calcaneus that is potentially more problematic with delayed healing of a wound.

Through the paramedian incision, we debride both the bone and the tendon at the junction and resect the posterior superior process. Tendon augmentation may be required, depending on the extent of debridement. When more than 80% of the tendon is involved or when the Achilles is degenerative at the insertion

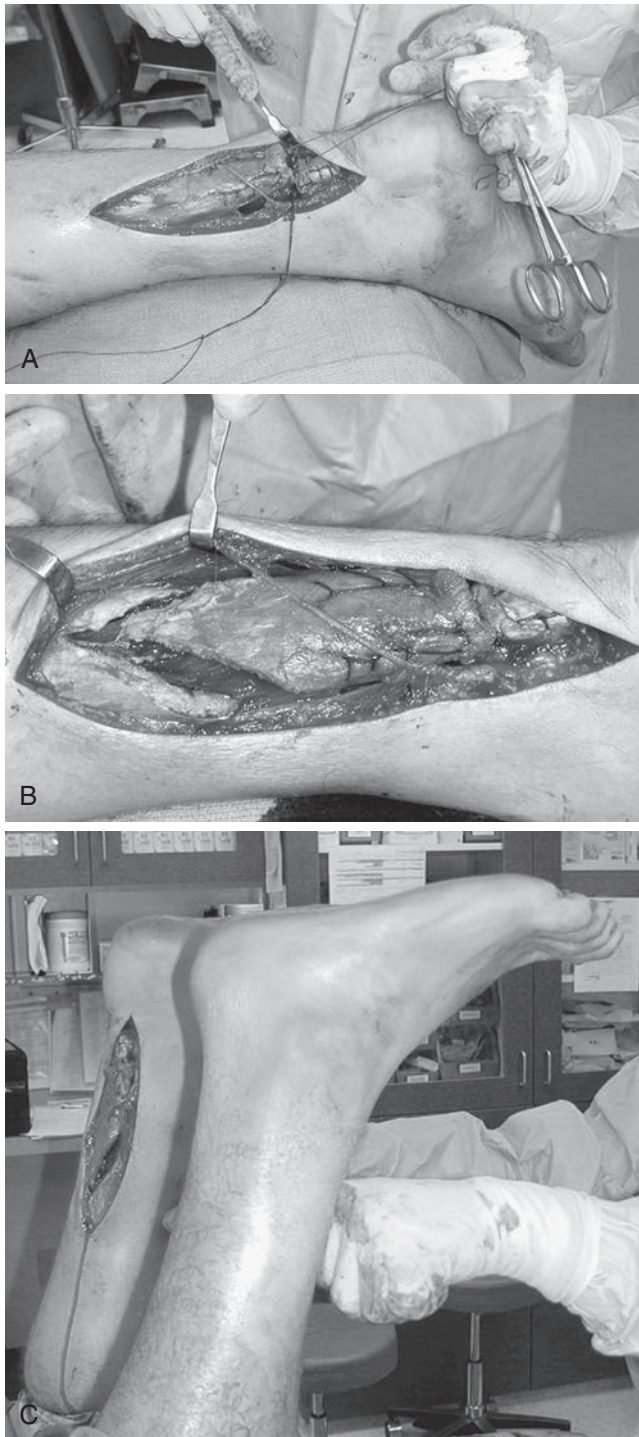


Fig. 9.18 The V-Y advancement. (A) The section of the diseased Achilles is debrided, and the new edges are sutured with a whipstitch for anastomosis. (B) A V-shaped fascial incision is made with the apex proximal. With traction on the tendon distally, an advancement of 2 to 3 cm can then be achieved. (C) The final tendon tensioning is performed by checking the resting posture of the ankle and testing the “springiness” of the foot as it sits in the normal slightly plantarflexed position. A comparison with the other side is helpful. The V-Y is then sutured. If it appears that there is too large a gap to close, a turndown or a tendon transfer may span the defect.

and proximally, an FDL or FHL graft should be considered (Fig. 9.21A–C). The FHL or FDL can be harvested in the arch of the foot or, as we prefer, behind the ankle. After the FHL is attached

to the bone through a tunnel or into a trough, the Achilles tendon is reattached with one or two suture anchors.

A biomechanical study by Kolodziej et al.⁴⁶ demonstrated that as much as 50% of the Achilles tendon may be safely resected. Despite this study, we still recommend placing suture anchors to optimize tendon bone contact and healing. When reattaching the tendon, it is important to restore normal resting tension, using the remaining intact portions of medial and lateral slips of tendon as a guide. Inadvertent overtensioning of the repair when using anchors could cause an equinus contracture or difficulty squatting or lunging.

Haglund’s Deformity

Surgical treatment for Haglund deformity and retrocalcaneal bursitis focuses on resecting the enlarged posterior bony prominence, including the attached precalcaneal bursal projection. Any inflamed retrocalcaneal bursa also is excised. Because the lateral side is more commonly affected, it is easier to approach through a lateral incision (Fig. 9.22A). A medial approach is warranted when the bony prominence is found medially (Fig. 9.22B). When the tendon is not involved, the insertion can be avoided through the lateral or medial approaches. Either way, it is critical to resect a sufficient amount of bone to prevent impingement on the tendon and avoid creating a sharp edge after resection that may irritate the tendon (Fig. 9.22C–D). Too much resection can weaken the tendon insertion, and the subtalar joint may be penetrated if the surgeon is not careful. Jones and James⁴⁷ advocated a combined medial and lateral approach to ensure a thorough bony resection (Fig. 9.22E). This exposure helps to avoid inadvertent creation of a sharp, bony edge.

We recommend a lateral approach or the combined medial and lateral exposure. The central posterior approach for Haglund deformity should not be used because it is better to avoid disrupting the insertion of the tendon unless there is a clinically relevant component of insertional tendinopathy (Fig. 9.23A–D). Bone resection should be performed just proximal to the insertion of the tendon (Fig. 9.23E). A power reciprocating rasp should be used to help contour the cut edges beside the tendon (Fig. 9.23F). A mini-C arm should be used to help identify any remaining prominences (Fig. 9.23G).

Endoscopic Haglund resection as presented by Niek van Dijk (see Chapter 16) has gained popularity. In this technique, the patient is placed in a prone position, and a lateral incision is made just dorsal to the calcaneus and anterior to the tendon (Fig. 9.24). A 4.5-mm, 30-degree arthroscope is introduced. A spinal needle then is introduced medially just dorsal to the calcaneus, and the 5.0-mm full-radius resector is inserted. With the shaver on the superior surface of the bone, the periosteum is removed. In plantarflexion the Haglund’s prominence can be resected with the shaver. A burr may be needed to remove bone at and near the insertion point of the tendon. The site of the burr placement should be confirmed with fluoroscopy. Adequate decompression is achieved at the posterior medial and lateral edges by alternating portals. The portals are sutured after the bursa and Haglund prominence have been removed with fluoroscopic assistance. This technique avoids the creation of tender scars in this region, has low morbidity, and may shorten recovery relative to the open procedures.

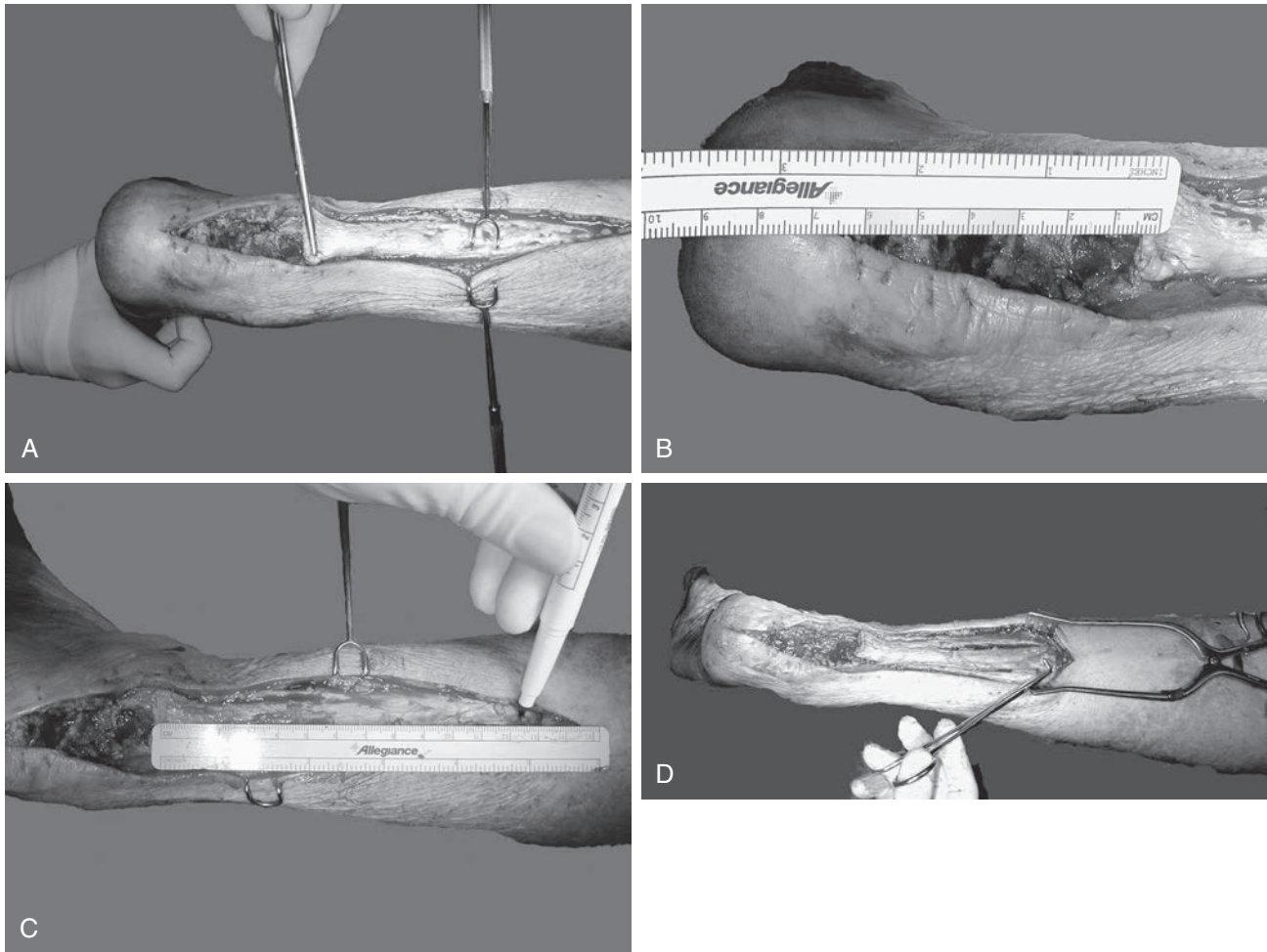


Fig. 9.19 The Turndown Procedure. (A) After the rupture or degeneration site is exposed, the end of the proximal tendon is mobilized and then grapsed with Alice clamps, and tension is pulled. (B) The size of the gap is measured while the foot is maintained in a neutral position. (C) An additional 4 cm then are added to the tendon defect (a 2-cm distal hinge that is overlapped by the turned-down flap, or 2 cm plus 2 cm). Another 1 cm is added to account for the intended 1-cm overlap of the tendon ends distally. Thus, the flap begins proximally at a point 5 cm more than the size of the gap. (D) A strip of tendon 1 cm wide is harvested centrally.

Alternatively, some authors have advocated a dorsal closing wedge osteotomy of the posterior tuberosity of the calcaneus. It is rarely used in athletes because of inherent complications with this procedure, including nonunion or malunion, potentially longer recovery times, difficult fixation, painful prominent hardware, broken hardware, and altered mechanics. This procedure may have a role if an athlete has a noticeably deformed posterior and superior calcaneal prominence, which we call the “pregnant heel” (Fig. 9.25). It should be reserved for atypical cases.

SYMPTOMS AND SURGERY

Haglund’s/retrocalcaneal bursitis

- Tender over posterior superior bony prominence and/or anteromedial and anterolateral Achilles
- Surgery: resect bone through medial and/or lateral incision over prominence

Insertional Achilles tendinitis

- Tender at the insertion of the Achilles halfway between the dorsal and plantar aspect of the calcaneus
- Surgery: central posterior Achilles splitting incision, debride tendon and bone

Postoperative Management

Our postoperative management for the athlete is determined on the basis of the extent of tendon involvement. If there is no tendon repair or reconstruction, a nonweight-bearing posterior and U-splint is applied in mild plantarflexion for the first 10 days. The sutures then are removed, and progressive, full weight bearing is permitted with a boot brace in neutral position. Between 6 and 12 weeks, the athlete is weaned out of the boot as tolerated. Early range-of-motion and strengthening exercises are encouraged. Impact activities are avoided for about 8 to 12 weeks. Swimming and exercise bicycling are encouraged by 3 weeks, followed by the elliptical trainer by 4 weeks. Beyond 12 weeks, the athlete may progress to sports-specific activities. Our average time for athletes to return to sports has been 3 to 6 months.

If the tendon was repaired or reconstructed or if a tendon was transferred, a postoperative splint is applied in a relaxed resting position (25 to 45 degrees of equinus). At 10 days, the sutures are removed, and a boot brace is applied in 20 degrees of plantarflexion. We permit active dorsiflexion progressively up

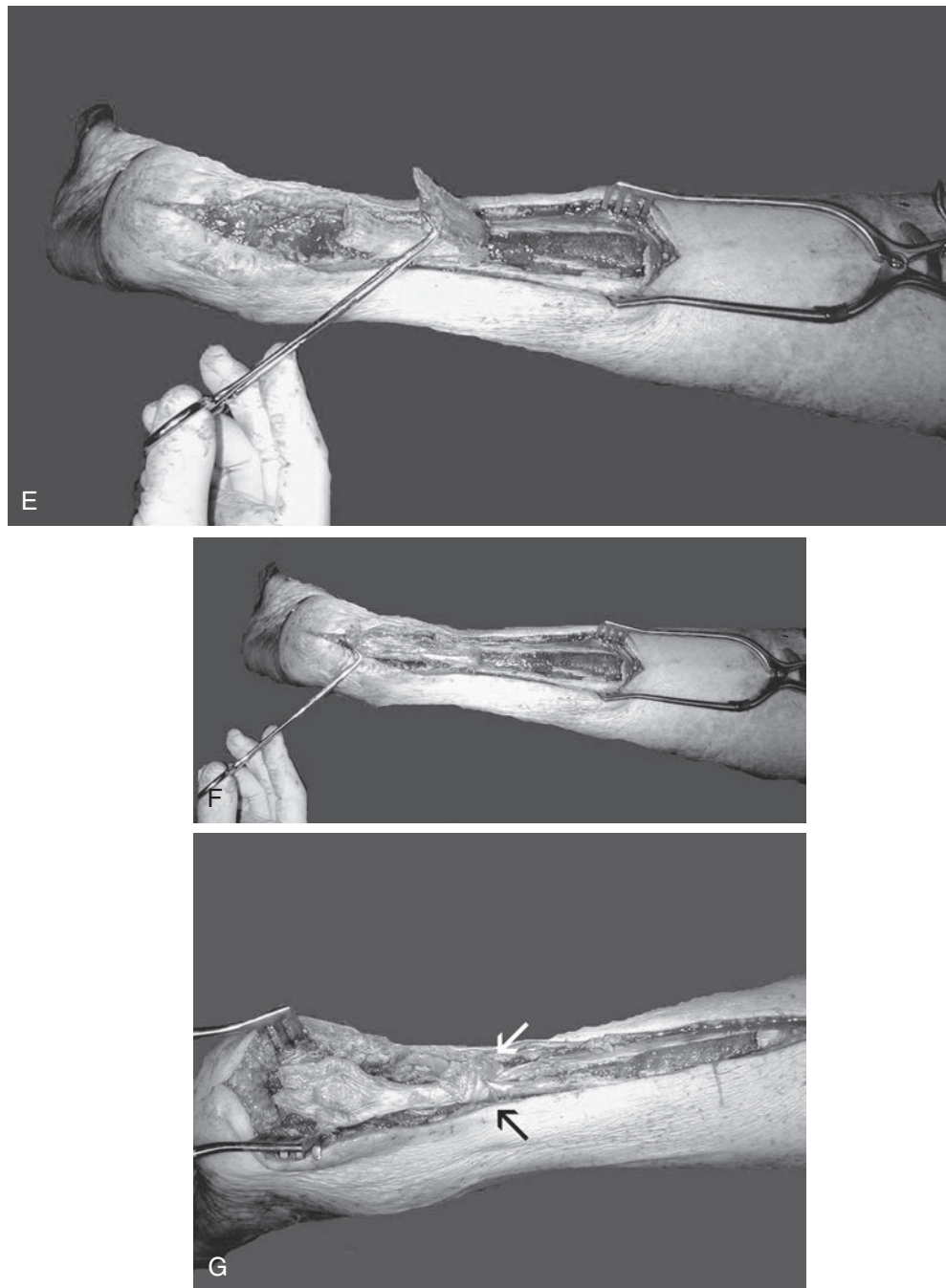


Fig. 9.19, cont'd. (E and F) The tendon graft now can be turned distally to span the void. (G) Two no. 1 Ethibond sutures are used to anchor the corner of the turned-down graft (arrows) reinforcing the high stress junction. The central slip typically is passed anteriorly deep to the tendon to decrease the bulk. The distal tendon end then is secured to the remaining viable Achilles or to the bone. Comparison with the nonoperative side facilitates tensioning of the graft.

toward the neutral point but recommend reaching the neutral point at 6 weeks. Exercises are encouraged with the knee flexed to eliminate extra pulling of the gastrocnemius muscle during dorsiflexion. Ankle inversion and eversion strengthening may be performed. Flexing the toes against resistance is avoided if a tendon transfer was performed but encouraged if no transfer was done.

Partial weight bearing in a boot is allowed while maintaining the “triple flex walk” (flexion of hip, knee, ankle) (Fig. 9.26).

With the ankle in plantarflexion and the ipsilateral leg remaining anterior to the body at all times, the patient leads with that leg in gait and keeps the sole of the foot in contact with the ground by flexing the knee and hip, similar to a fencer’s advance. Although the appearance is awkward, this method permits ambulation without crutches.

Ankle neutral position is achieved by 6 weeks, permitting full weight bearing with the boot adjusted at a right angle to the leg. Then progressive dorsiflexion exercises beyond neutral

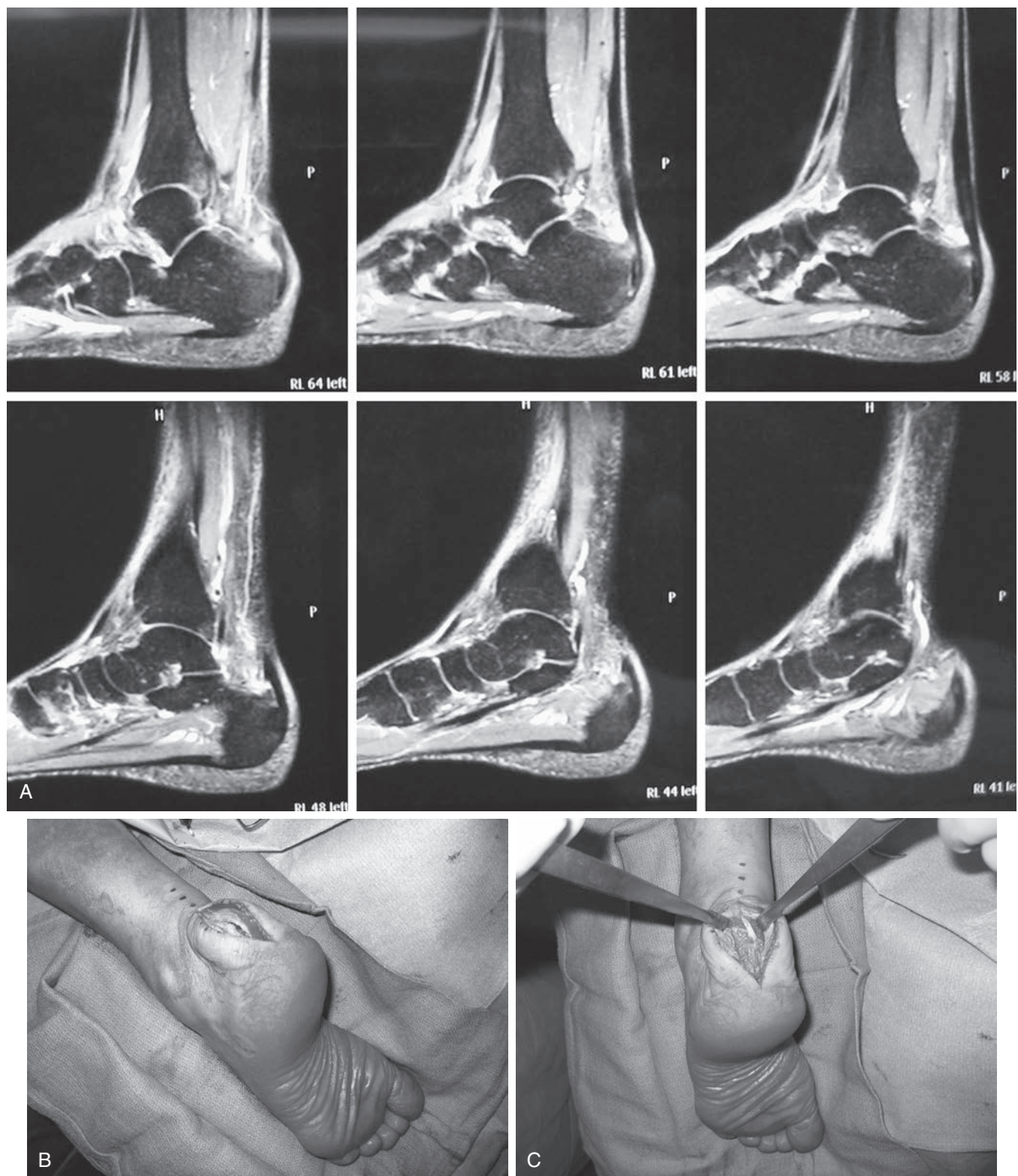


Fig. 9.20 Insertional Achilles Tendinopathy. (A) Sagittal magnetic resonance imaging of a patient with insertional Achilles tendinopathy and retrocalcaneal bursitis. Note the bony prominence, the fluid in the bursa anterior to the tendon, and the abnormal signal at the insertion consistent with degeneration at the interface. (B) A central incision is made through the site of maximal tenderness through the tendon down to the bone. (C) The tendon is released from its insertion posteriorly and the posterior superior calcaneus is exposed.

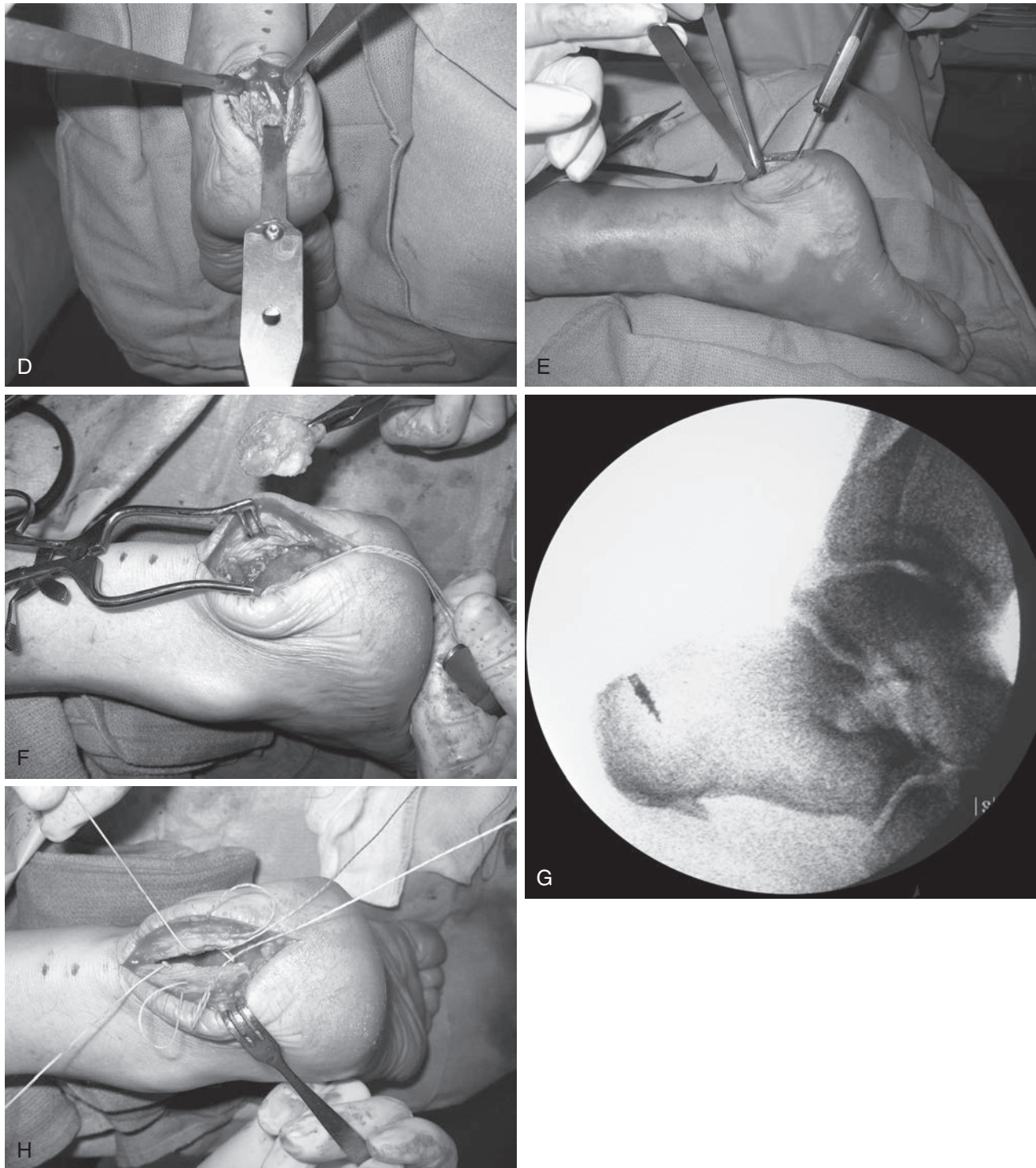


Fig. 9.20, cont'd. (D) Two human retractors are placed, and the chisel is used to resect the insertion site and the posterior superior bony prominence. (E) A side view of the chisel angle to resect the bony prominence. Care is taken not to inadvertently penetrate too anteriorly and end up in the subtalar joint. The medial lateral and dorsal edges are checked for remaining bone. (F) The bone has been resected and the suture anchor is placed centrally into the calcaneus about 5 to 8 mm proximal to the previous insertion site. (G) An intraoperative image demonstrating the anchor placement. (H) The sutures are placed close to the midline, penetrating the tendon directly over the anchor with very minimal divergence to maximize tendon apposition to the bone. The knots should be buried so that postoperative irritation is avoided.

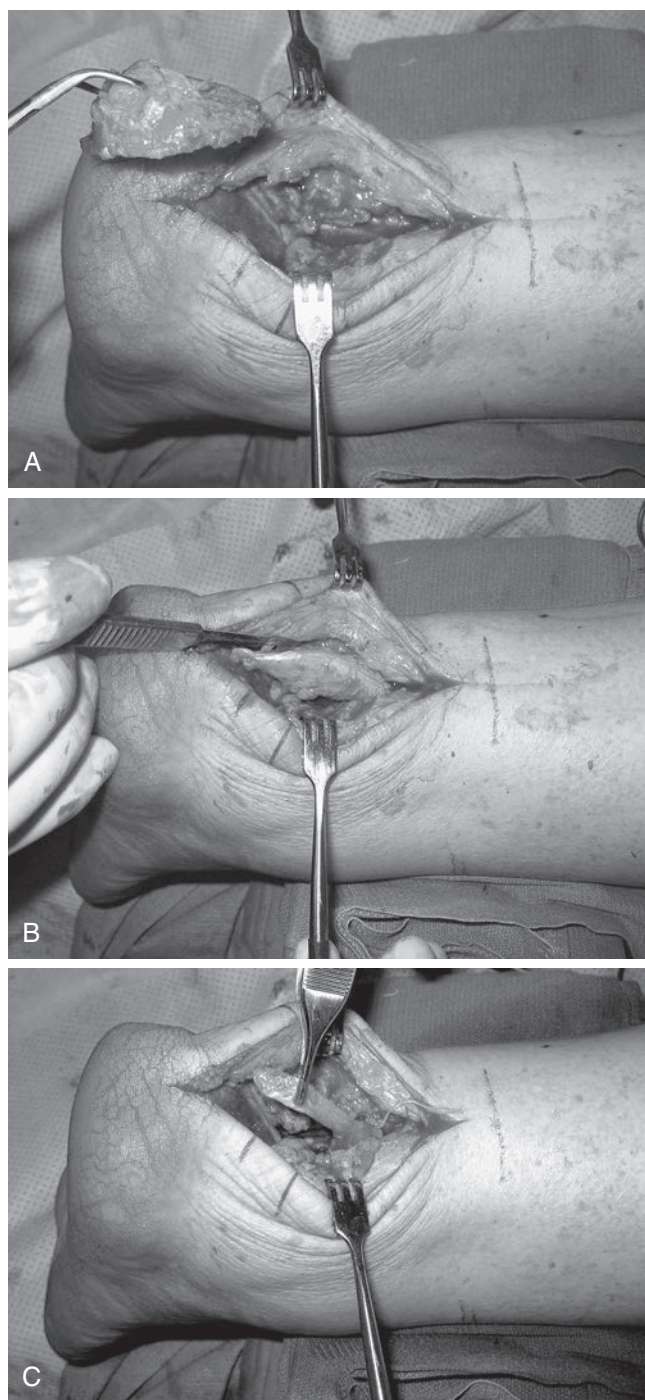


Fig. 9.21 When the Achilles is degenerative at the insertion and proximally or when more than 80% of the tendon is involved, a flexor hallucis longus (FHL) graft should be considered. (A) The central approach is used to detach the Achilles posteriorly, and the prominent bone is resected. (B) The degenerative tendon is debrided. (C) The FHL tendon is harvested from behind the ankle and will be reattached through a tunnel or into a trough before repairing the Achilles tendon.

are performed, with caution not to overstretch. Dorsiflexion is progressed slowly, depending on the integrity of the repair. Swimming, bicycling, and other nonimpact activities are commenced at 6 weeks. At 12 weeks, the boot is discontinued, and lower-impact activities such as the elliptical trainer are instituted and increased. The patient then is progressed to jogging and then running. It may take 4 to 6 months to return to play,

or perhaps more than 9 months, depending on the extent of the tendon disease and the integrity of the repair.

Surgical results typically correspond to the athlete's age, with patients younger than 50 years generally having less tendon involvement, more rapid return to work and sports, and fewer postoperative problems. Patients older than 50 years typically have more tendon involvement, require greater debridement, and have longer postoperative recovery.^{27,30}

Expected Success Rates and Return to Sport

Success rates have ranged from 50%–90%, depending on the extent and location of tendon involvement. Surgical debridement for Haglund syndrome and chronic tendinosis generally has less favorable results.^{48,49} Watson et al.⁵⁰ reported that those with insertional tendinosis are older athletes, have greater tendon involvement, take a longer time to recover, and often do not achieve results as satisfying as those with isolated retrocalcaneal bursitis. Leach et al.⁵¹ reported in a small series of athletes that the long-term success rate was 85% following surgical treatment. However, the authors noted that symptoms recurred in two patients and required reoperation. A study by Schepsis et al.⁴⁸ also demonstrated initially high satisfactory results, mostly in athletes with paratendinitis, although long-term results deteriorated with time. In a recent study by Saxena,⁵² return to activity was fastest in elite athletes requiring only soft tissue procedures, particularly peritenolysis. Return to competition was approximately 6 months. Although many studies in the literature have quoted high success rates, this optimism for surgical treatment must be tempered by the fact that these were retrospective analyses that often did not differentiate among the various Achilles tendon disorders.^{23,27,30,47–49,51,53,54}

ACUTE ACHILLES TENDON RUPTURE

Introduction

Although once believed to have a lower incidence than other lower extremity tendon ruptures (i.e., patellar tendon, quadriceps tendon), the incidence of Achilles tendon ruptures has been increasing over the past few decades to become the most common tendon rupture in the lower extremity.^{55–61} Most Achilles tendon ruptures occur in males in the fourth or fifth decade of life with nearly 70% of injuries occurring from sporting activity. Thus, the increased incidence of Achilles tendon ruptures seems related to the higher number of middle-aged males who participate in high-demand sports only on occasion; the so-called “weekend warriors.”⁵⁶

In the United States, in decreasing order of incidence, basketball, tennis, and football, have been the most commonly involved sports, accounting for 73% of all sports-related Achilles tendon ruptures.⁵⁶ However, with the increasing popularity of professional and amateur soccer within the United States, the incidence may increase further since soccer is the most common sport involved in acute Achilles tendon ruptures in European and Canadian studies.^{57–60}

Although direct injuries to the Achilles tendon can occur from a laceration or a direct blow, most athletes experience an indirect mechanism with the foot in a dorsiflexed position with an extended knee. The tensioned tendon experiences a rapid eccentric load seen in explosive accelerations or jumps



Fig. 9.22 The Incisions for Treatment of Haglund's Deformity. (A) A lateral incision is more common because the prominence is usually more pronounced on this side. (B) A medial approach is warranted for a medial bony prominence. (C and D) Radiographs preoperatively and postoperatively show sufficient bone resection without impingement at the tendon insertion.

that place athletes at an increased risk for an Achilles tendon rupture.⁶² A 2 to 6 cm hypovascular region of the tendon located superior to the calcaneal tuberosity has been found to be susceptible to degenerative changes and can predispose an athlete to an indirect injury.⁶³ Risk factors for tendon disruption include chronic tendon degeneration,⁶⁴ use of fluoroquinolone antibiotics,⁶⁵ oral or local injection of corticosteroids, gout, systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis,⁶⁶ renal failure, hyperthyroidism, infection, and tumor.^{67,68} A recent study reported that there was no difference in injury rate related to game play on grass versus artificial turf.⁶⁹

A focused history and physical examination is usually sufficient to diagnose acute Achilles tendon ruptures. An athlete may report a feeling of a “gunshot to the heel” with or

without an audible pop or snap during a jumping or push-off maneuver.⁷⁰ The player will not return to play and walks with a limp from weak plantarflexion and pain. On a side-line examination, increased passive dorsiflexion along with a palpable gap in the tendon are found in most circumstances other than the most distal tears and avulsions. In late-presenting Achilles tendon ruptures, significant swelling can obscure the gap between the tendon ends making the diagnosis less obvious. The Thompson test, or calf squeeze test, is the most commonly used and most sensitive physical exam test to assess an Achilles tendon injury. The athlete kneels on a chair with the foot extending beyond the chair edge. The calf is squeezed and an intact tendon will briskly plantarflex the foot. On the injured side, plantarflexion of the foot is absent (a positive test).⁷¹ The knee flexion test can also be performed

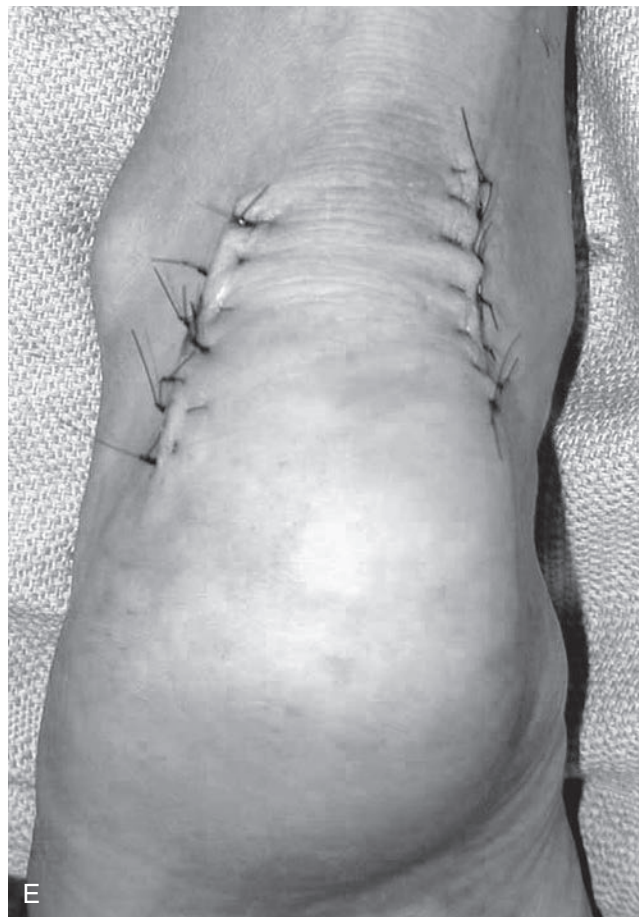


Fig. 9.22, cont'd. (E) A combined medial and lateral approach ensures a thorough bony resection and edge contouring.

with the patient prone with the knees flexed to 90 degrees (Fig. 9.27). The uninjured ankle will rest in plantarflexion, while the injured ankle falls into neutral or dorsiflexion (see Fig. 9.28).⁷²

In equivocal or late presentations of Achilles tendon ruptures, diagnostic studies are useful. A lateral radiograph may reveal an avulsion of the tendon from the calcaneus, intratendinous calcifications, or a loss of the Kager triangle (a lucency created by the anterior border of the Achilles tendon, posterior border of the deep flexors, and posterosuperior border of the calcaneus). MRI is useful to provide information on chronic tendon degeneration and to distinguish between a partial or complete tear in equivocal situations (see Fig. 9.29).⁷³ Ultrasound is an effective imaging method and is becoming more widely used due to its low cost, convenience, and ability to dynamically determine where the two ends of the Achilles tendon are located. The use of ultrasound has been expanded to help predict the risk of rerupture and poorer outcomes in patients with Achilles tendon ruptures.⁷⁴ Sports-related injuries are most often the result of a rapid eccentric load that is applied to a tensioned tendon with ankle dorsiflexion and simultaneous knee extension. This occurs during the loading phase of a rapid push-off or sudden jump. Most ruptures occur

in an area of relative hypovascularity of the Achilles tendon, 2 to 4 cm proximal to the superior aspect of the calcaneus.⁶⁸ Additional risk factors include intratendinous degeneration, vascular impairment, corticosteroid or fluoroquinolone use, mechanical malalignment, and systemic disorders such as gout, hyperthyroidism, and renal insufficiency.^{67,68}

An athlete typically reports an audible snap and a sensation of being struck or shot from behind following a misstep or sudden jump. The player will note significant loss of push-off strength and normally will be unable to continue sports participation. Diffuse swelling, ecchymosis, and residual strength from remaining ankle plantarflexors can make diagnosing an initial injury difficult. However, findings consistent with an acute Achilles tendon rupture include a palpable tendon gap (Fig. 9.30), positive Thompson test (absence of passive ankle plantarflexion with calf squeeze in prone position; see Fig. 9.27 and Video 9.1), loss of the normal plantarflexion resting tone while prone in comparison with the unaffected side, inability to perform a single toe heel rise, and weak active plantarflexion. Although imaging generally is unnecessary in acute cases, lateral radiographs may show an avulsion fracture (Fig. 9.31). MRI and ultrasound are useful in equivocal or late cases (Fig. 9.29).

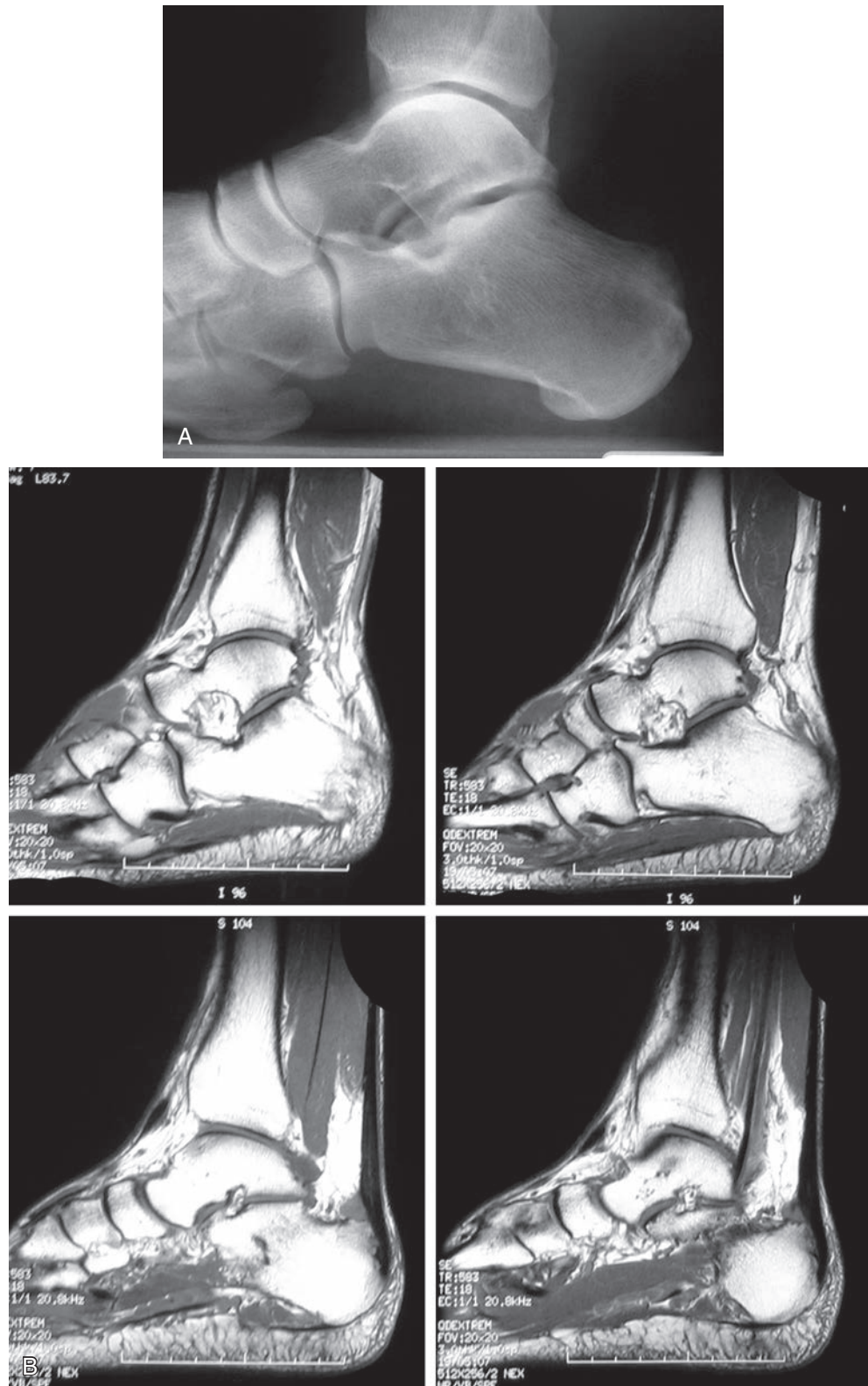


Fig. 9.23 (A) through (C) This athlete's x-rays and sagittal magnetic resonance images demonstrate a Haglund deformity, retrocalcaneal bursitis, posterior calcaneal bony edema, and some insertional Achilles tendon changes. (D and E) Because all the tenderness and prominence were lateral and there was no tenderness as the Achilles insertion, a lateral approach was chosen, with the intraoperative option of an additional medial incision to contour the sides. (F) Bone resection should be performed just proximal to the insertion of the tendon. A power reciprocating rasp should be used to help contour all edges by the tendon. (G) A mini-C arm should be used to help identify any remaining prominences.

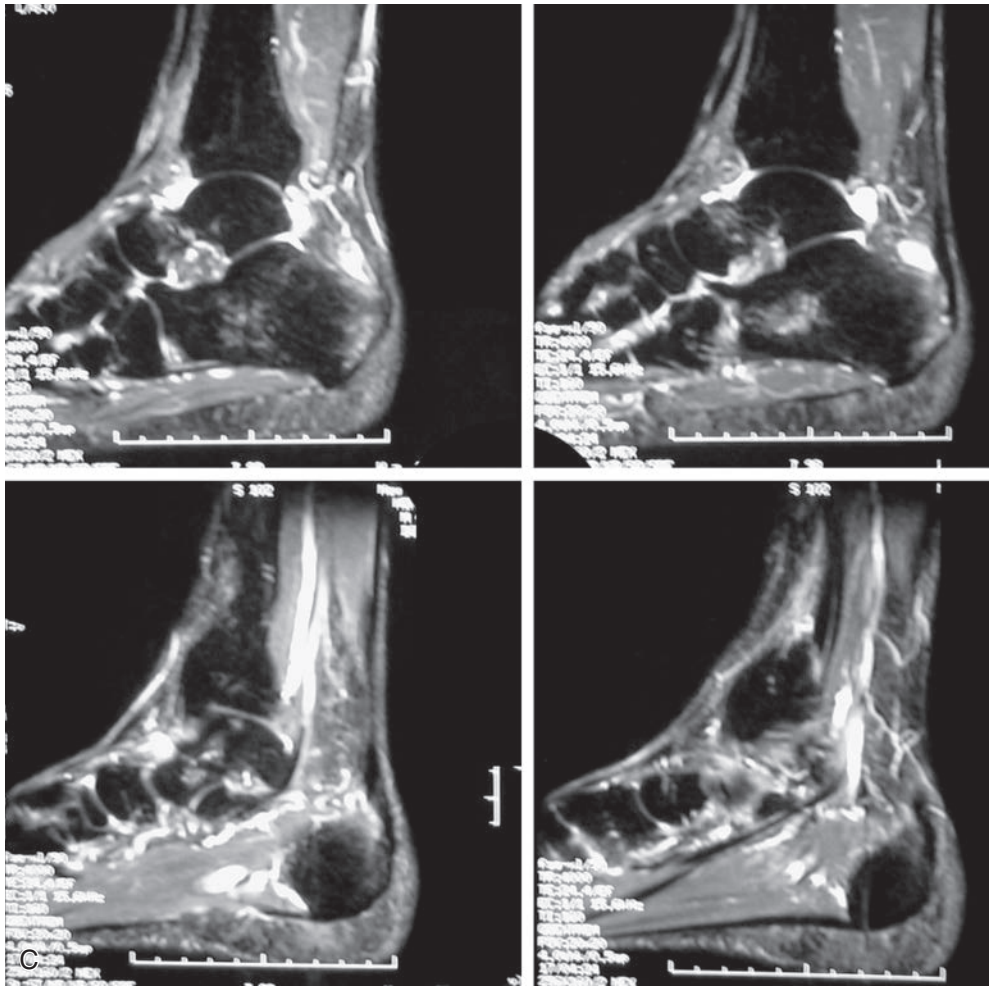


Fig. 9.23, cont'd.

Nonsurgical Treatment

Although treatment of choice for most athletes with an acute Achilles tendon rupture is surgical repair followed by early, protected range of motion and weight bearing, nonoperative treatment of Achilles ruptures using cast or boot immobilization followed by functional rehabilitation has some advocates. The ill effects of “cast disease”^{75,76} include calf atrophy and resultant muscle weakness, and the consequences of immobilizing joints, ligaments, and uninvolved muscles and tendons. Additionally, tendon elongation, loss of muscle strength, extended rehabilitation, additional time-loss from sport, and decreased function are persistent concerns in Achilles tendon ruptures treated nonoperatively in the high-level athlete.^{77,78} Although operative management is associated with inherently potential surgical risks, including poor wound healing, infection, and nerve injury, the risks are balanced by a lower incidence of tendon rerupture rates, less than 2%, compared with 13% to 35% after nonoperative care.^{75,77,79,80} Newer less invasive, mini-open techniques and knotless repairs have lessened the risk of these complications.^{81,82} Further, studies have demonstrated improved strength and ankle motion with a greater potential of sports resumption following surgical repair.^{75,83}

Nonoperative management generally is better than operative management in those with systemic disorders, such as diabetes, peripheral vascular disease, lower-extremity edema, or overlying skin conditions. However, these comorbidities are not often found in the athletic population. Following nonoperative management, a short-leg, nonweight-bearing cast or a controlled ankle motion (CAM) walking boot in slight equinus is used for 6 weeks. This is followed by a weight-bearing cast or walking boot with progressively increased dorsiflexion.

Results similar to those of operative management have been reported for nonoperative treatment with a functional boot brace guided by ultrasound.⁸⁴ Ultrasound is used to ensure that the ends of the torn tendon remain apposed as the ankle is progressively dorsiflexed during the first 6 weeks. Additionally, a successful nonoperative protocol has been reported using a functional brace to minimize the ill effects of immobilization and to shorten the time needed for rehabilitation and return to activities.⁸⁵ A more recent study found similar success with operative and nonoperative treatment in the athlete.⁸⁶

More recent postoperative trends have focused on a functional rehabilitation program with early controlled range-of-motion and strengthening exercises.⁸⁷⁻⁸⁹ Early mobilization limits the dystrophic effects of prolonged cast use and has been

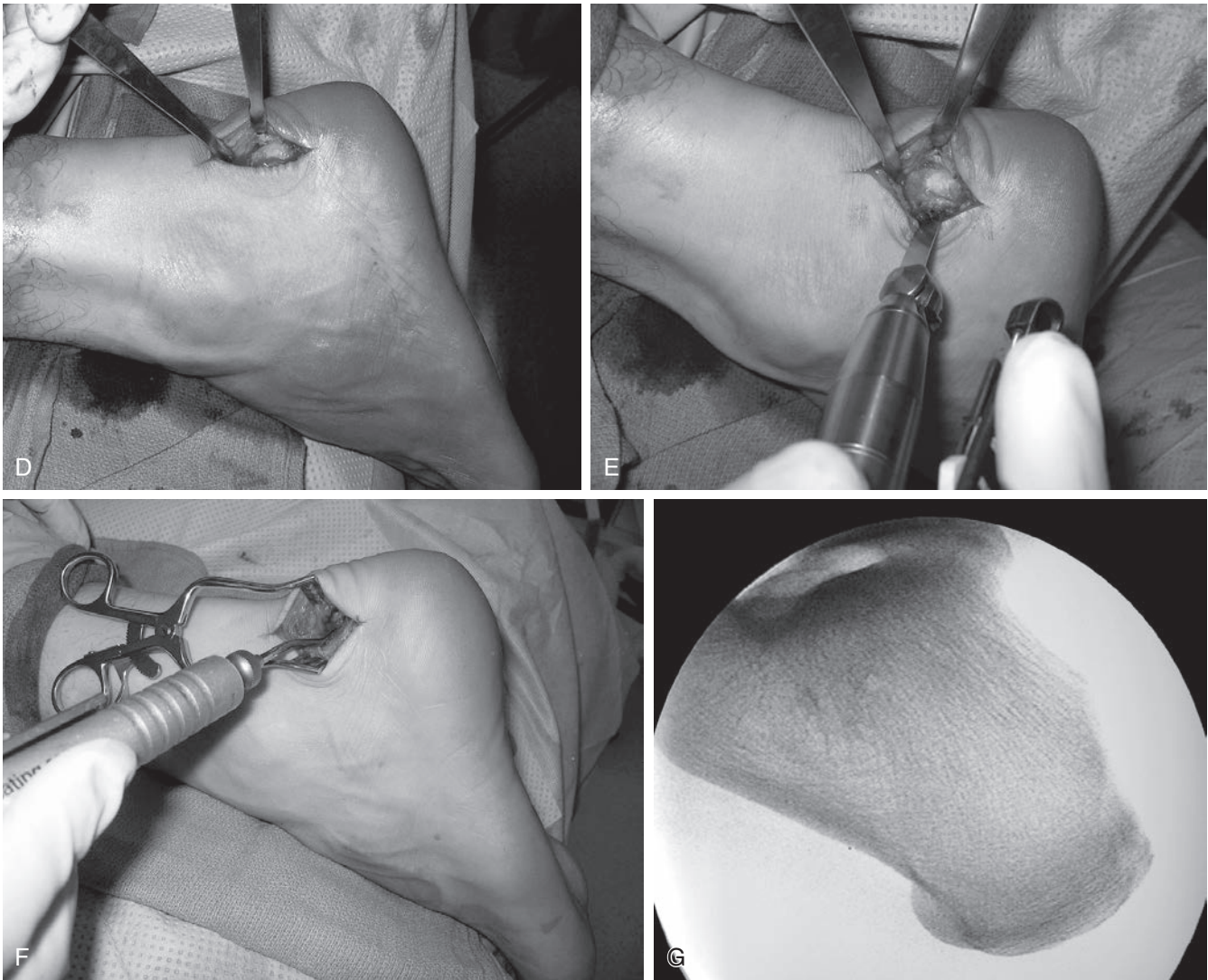


Fig. 9.23, cont'd.

shown to reduce tendon adhesion, improve healing, and maximize tendon strength without increasing the risk of rerupture or infection. We favor operative repair unless contraindicated.

Surgical Treatment

Surgical treatment is the mainstay of treatment for the athlete with an acute rupture of the Achilles tendon. Level 1 literature suggests that operative repair increases strength and improves clinical outcomes compared with nonoperative treatment at 6 and 18 months.⁹⁰ Athletes with well-controlled systemic disorders, such as diabetes, should be considered for operative treatment. This higher-risk situation requires close attention to wound closure and postoperative management, including meticulous and frequent follow-up.

Standard Technique

Acute Achilles tendon ruptures in an athlete should be treated operatively with the goal of recreating normal tendon length and tension. The patient can be positioned in the supine or prone position. Additionally, it is helpful to prep and drape the contralateral extremity to help match resting ankle tension. A

medial approach 1 cm anterior to the Achilles tendon border avoids injury to the sural nerve and is located in relatively thick tissues, which are biomechanically better suited to provide a healthy closure farther away from the tendon (Fig. 9.32). Care is taken to minimize soft-tissue handling. Meticulous soft tissue handling is used to protect the incision and reduce the risk of wound breakdown. A Krackow-type stitch technique with nonabsorbable, no. 2 sutures is used to reapproximate the “mop end” rupture (Fig. 9.33). The hematoma is evacuated, and the tendon ends are found. The suturing begins in the proximal end so that tension can be progressively applied to the tendon, allowing the contracted muscle proximally to relax. Next, the distal end is sutured, and the tendon ends can be anatomically reduced. The sutures are then tied.

Whenever possible, the paratenon is reapproximated to minimize scar formation and improve tendon glide. Additionally, reapproximation of the fat pad anterior to the tendon can be performed. Initially, we immobilize the leg for 10 days until the wounds have healed. The same postoperative protocol described above for tendon reconstructions is used. Return to sport for the athlete after repair is 4 to 6 months.

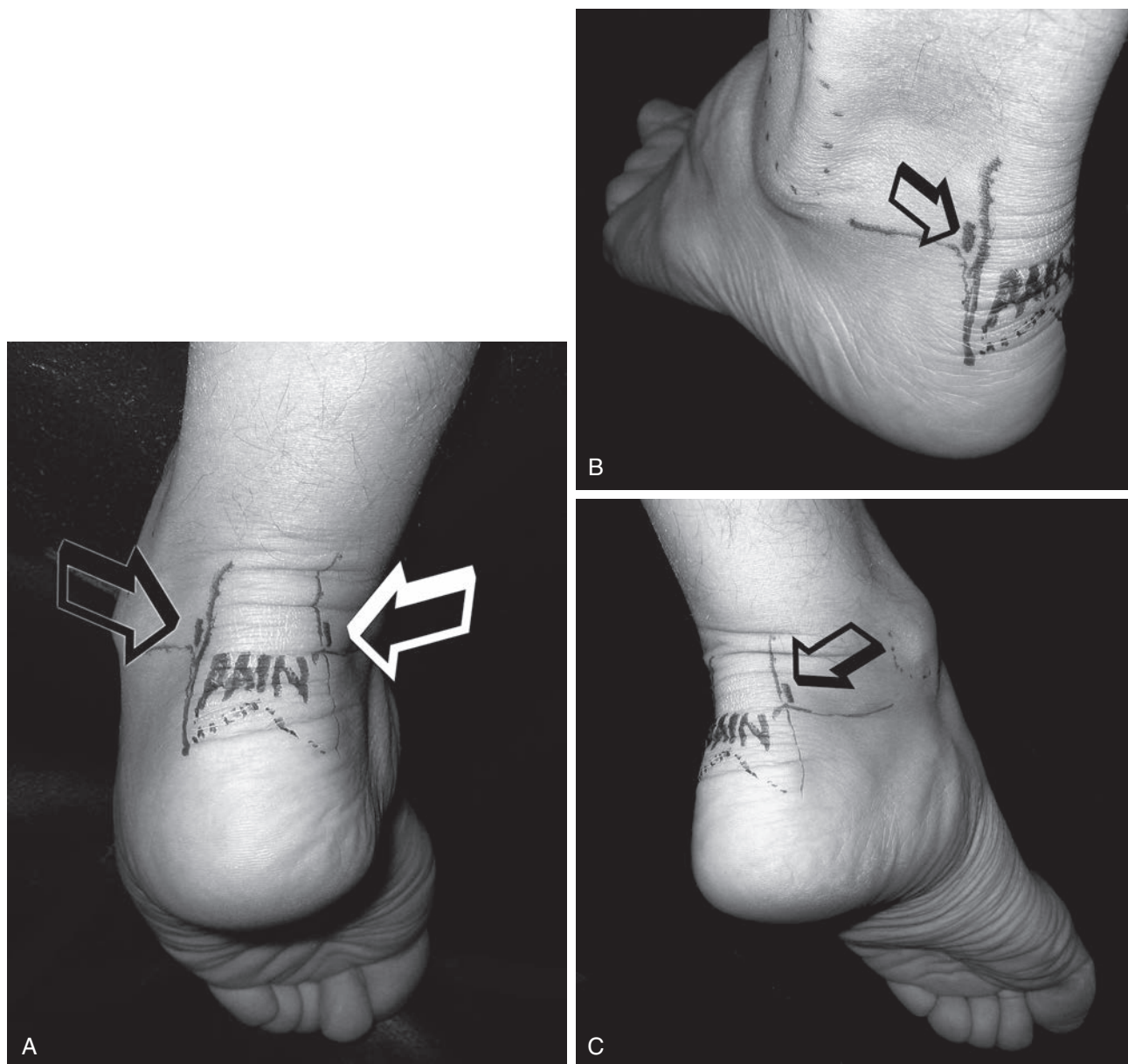


Fig. 9.24 Endoscopic Portal Landmarks With the patient in a prone position, the lateral incision is made just dorsal to the calcaneus and anterior to the tendon. (A) The posterior view demonstrating the lateral portal (*black arrow*) and the medial portal (*white arrow*). The area of pain is marked on the patient's skin. A dotted line is marked at the superior site of the Achilles insertion below the area of pain. (B) The lateral perspective of the portal site. (C) The medial perspective of the portal. A 4.5-mm, 30-degree arthroscope is introduced laterally. A spinal needle is introduced medially just dorsal to the calcaneus. The 5.0-mm full-radius resector then is inserted.

The method of suturing and the suture material have been a source of research. Since both early weightbearing and early motion are essential to improving clinical outcomes,^{91,92} the technique used must be strong enough to withstand the forces applied during rehabilitation. Traditionally, a Krackow-type stitch technique was used with nonabsorbable No.2 sutures to repair the tendon.⁹³ However, studies have focused on the specifics of suture constructs, suture caliber, and number of core strands crossing the repair in an effort to strengthen the construct.

In terms of suture repair constructs, no difference in strength was seen between a double Bunnell, double Kessler, and double

Krackow stitch.⁹⁴ Several modifications such as the “Gift Box” or the “triple bundle” technique have shown greater strength as compared with standard Krackow sutures⁹⁵⁻⁹⁷, though strangulation of the tissue should always be a concern in these types of suture constructs. Typically, the strength of a tendon repair is directly related to the number of core strands of suture and the strength of the suture itself. For example, a biomechanical study using a modified Kessler technique found that a construct consisting of four strands of No. 2 sutures and two strands of 2-mm tapes and a construct with 12 No. 2 suture strands were able to survive the later stages of a simulated rehabilitation protocol. Substituting suture tape for two core strands or doubling the



Fig. 9.25 *Top left corner* shows the preoperative appearance of this large posterior prominence. It is uncharacteristically inferior, although the patient did not have insertional tendinopathy. The senior author LCS calls this the “pregnant heel.” This is a rare exception to consider, a dorsal closing wedge osteotomy of the posterior tuberosity of the calcaneus. *Top left* shows the lateral approach anterior to the Achilles tendon. *Bottom left* shows the preoperative radiograph. The *bottom right* demonstrates the calcaneus following the resection of the dorsally based closing wedge and fixation with a screw. The technique is used rarely because of inherent complications with this procedure, including nonunion or malunion, potentially longer recovery times, difficult fixation, painful prominent hardware, broken hardware, and altered mechanics.

core strands with a smaller-caliber suture both created a biomechanically stronger repair in a cadaver model.⁹⁸

TOC and JHM's preference for open repair has been a 6 core strand technique with 2-0 FiberWire (Arthrex Inc., Naples, Florida) placing one set of sutures central posterior, one set anteromedial and a final set anterolateral. This is followed by an epitenon repair with 3-0 Monocryl (Ethicon Inc., Somerville, New Jersey) and standard wound closure. Whenever possible, the epitenon is repaired to minimize scar formation and improve tendon glide. It also improves the strength to failure by 53--119%.⁹⁹⁻¹⁰¹ The leg is immobilized in a splint in resting equinus for 7 to 14 days until the incision has healed. We use a specific postoperative protocol to guide patients through their surgery and postoperative rehabilitation, including progressive dorsiflexion and weightbearing (Table 9.1A-B). The athlete is allowed to return to sports activity in 4 to 6 months at the earliest, but this is guided by objective testing. It can occasionally require a full year for the athlete to return to full sport participation.

Percutaneous Technique

The true percutaneous technique was introduced as a minimally invasive procedure to reduce the morbidity and complications associated with open surgery. The original percutaneous technique for repair of Achilles tendon ruptures was described by Ma and Griffith and used six stab incisions, a proximal Bunnell suture and a box suture in the distal tendon stump.¹⁰² Reduced surgical complications were noted in meta-analyses of this procedure,¹⁰³⁻¹⁰⁵ but sural nerve entrapment was as high as 16.7%.^{103,106} In order to reduce this risk of injury to the sural nerve,¹⁰⁷ techniques and devices used through a mini-open approach have been developed.^{108,109}

Mini-Open Technique

Recently, the mini-open approach has been used more widely in order to minimize surgical complications. The Achillon device (Integra Life Sciences Corp., Plainsboro, New Jersey) was designed to be used through a mini-open approach.¹⁰⁸ Early results of this technique compared with an open repair



Fig. 9.26 Partial weight bearing in a boot in plantarflexion is allowed while maintaining the “triple flex walk” (flexion of hip, knee, ankle). (A) With the ankle and the ipsilateral leg remaining anterior to the body at all times, the patient leads with that leg in gait and keeps the sole of the foot in contact with the ground by flexing the knee and hip, similar to a fencer’s advance. (B) The back leg is now brought forward but does not advance beyond the front healing leg so that the Achilles can be kept unloaded. Next the braced leg is advanced again as in A. This method permits ambulation without crutches.

showed lower complication rates and improved cosmetic appearance.¹¹⁰ A similar device by Giannini (Citieffe, Calderara di Reno, Bologna, Italy) is also effective. Another device, the Percutaneous Achilles Repair System (PARS) (Arthrex, Inc., Naples, Florida), has been available since 2010 and uses non-locking and locking sutures to gain greater purchase within the tendon stumps. A biomechanical study showed greater construct strength under cyclic and ultimate loads with the PARS repair as compared with the Achillon system.¹¹¹

To minimize the possibility of injury to the sural nerve inherent in a purely percutaneous suture technique, a mini-open approach can be used. A small stab incision is made, and then the subcutaneous soft tissue is spread bluntly before passing the suture/wire. These mini-open techniques permit the advantage of direct visual repair while minimizing potential complications of wound and nerve problems. A small skin incision is made, and the Achillon, Giannini’s device, or PARS is introduced under the paratenon. A needle with suture is passed from the external guide through the skin into the tendon and out the opposite side. Three sutures are passed through the proximal tendon end, and three are used in the distal tendon end. The

device and the suture ends are pulled out from under the paratenon and incision such that the ends of the sutures grasping the tendon now rest entirely within the paratenon. The tendon ends are reapproximated, and the sutures are tied.

Assal et al.¹⁰⁸ reported their experience using the Achillon device in 82 patients, noting that all patients who were elite athletes were able to return to their same level of competition. Maffulli et al.¹¹² found that the average time to full return to sport participation after percutaneous fixation of Achilles tendon ruptures in elite athletes was 4.8 ± 0.9 months. In a single-center, retrospective cohort study, Hsu et al. compared 101 PARS Achilles repairs with 169 open repairs. For PARS cases, a reusable metal PARS jig was used to pass sutures through the Achilles proximal and distal stumps in a way that pulls the sutures within the paratenon and reduces the risk of sural nerve injury. The PARS jig is used to place sutures in the proximal and distal Achilles tendon stumps and has the ability to lock one or two of the three to five sutures used through the device. With the foot in a plantarflexed position, the sutures in each end are tied under maximum tension nearest to farthest from the rupture site to avoid tendon bunching in the



Fig. 9.27 A positive Thompson test in the near leg with the Achilles tendon rupture. The calf muscles are squeezed and there is an absence of passive ankle plantarflexion in the prone position.



Fig. 9.28 Loss of plantarflexion angle after Achilles rupture.

center. The overall complication rate was 8.5% with no reruptures in either group. Of the 101 patients treated with PARS, 3 had superficial wound dehiscence treated with local wound care without antibiotics. Two other patients in the PARS group underwent reoperation for a foreign-body reaction to FiberWire suture that developed 4 months after the initial surgery. The study showed a significant difference in return

to physical activity at 5 months with 98% of PARS patients returning, compared with 82% of open repairs.⁸⁸

A knotless modification of the above mini-open technique was reported by McWilliam and MacKay and has become our preferred method. It uses the PARS device for placement of the proximal sutures and then anchors these sutures to each side of the calcaneus with bone anchors.⁸⁹ Based on biomechanical research,¹¹³ TOC and JHM have modified this technique slightly to include a FiberTape suture (Arthrex, Inc., Naples, Florida).¹¹⁴ In McWilliam and MacKay's preliminary report on 34 patients with follow-up of 2 to 36 months, there were no wound complications, reruptures, or nerve injuries, and all patients reported satisfaction with their outcomes (Fig. 9.34).⁸⁹

Expected Results of Acute Surgical Repair

Though most high-level athletes have near limitless rehabilitation, an Achilles tendon rupture can be a career-ending injury. Significant delay in return to play, and reduced playing time and performance have been shown in National Basketball Association (NBA) and National Football League (NFL) players who sustain an Achilles tendon rupture requiring surgical repair. Overall, a return to play rate of 66% to 78% was noted in these studies.^{63,115-120} In a study of 62 athletes, which included NBA, NFL, and Major League Baseball players, functional deficits were seen only at 1 year after surgery compared with matched controls. The players showed performance at a level comparable to uninjured controls at 2 years postoperatively.¹²⁰ In another study examining outcomes in recreational athletes at an average of 32 months following acute Achilles tendon repair, Porter et al. showed that while there were objective plantarflexion strength deficits in the operative extremity, subjective results indicated near normal pain level and function.¹²¹ Due to the various methods of obtaining and analyzing available information on professional athletes and their injuries, critical evaluation of scientific method and careful interpretation should always be applied to these outcomes studies of professional sports athletes.¹²²

One may expect return to sports generally at 4 to 6 months after acute repair and a program of early protected weight bearing.^{23,77,123,124} Cetti et al.⁷⁵ previously showed less calf atrophy and improved ability to resume preinjury level of athletic play after surgical repair as compared with nonoperative treatment.^{75,77,81-83,123} However, Heikkinen et al.¹²⁵ demonstrated that surgically repaired Achilles tendons are longer, more atrophied, and have less strength when compared with the uninjured leg. In a meta-analysis of acute Achilles tendon treatment, Bhandari et al.¹⁰⁵ confirmed a statistically significant reduction in rerupture rates after surgical repair (3.1%) as compared with nonoperative treatment (13%). However, infections occurred only in the surgically treated group (infection rates ranging from 4% to 20%). The proportion of patients who regained normal function was similar in the operative and nonoperative treatment groups.

In another meta-analysis, Khan et al.¹⁰⁴ identified 12 suitable papers for inclusion. They found that the relative risk of rerupture was 0.27 with operative versus nonoperative treatment. Complications including infection, adhesions, and altered skin



Fig. 9.29 Sagittal magnetic resonance image showing a long, complex Achilles tear (white arrows).

sensitivity had a relative risk of 10.6 (operative versus nonoperative). Functional bracing postoperatively had lower complications than casting postoperatively (relative risk 1.88). They concluded that operative treatment significantly reduced the risk of rerupture but significantly increased the risk of complications overall.

In a multicenter randomized trial, Willits et al.,¹²⁶ examined the outcomes of 144 patients with acute Achilles tendon ruptures treated operatively vs nonoperatively for which both groups were treated with an accelerated functional rehabilitation protocol including early weight bearing and early range of motion. The authors did not find a significant difference in rerupture rates between operative and nonoperative treatments. However, similar to findings presented in previous studies, they found an increased risk of complications in the operatively treated group. Despite these findings, a recent analysis of 12,570 patients treated for an acute Achilles tendon rupture demonstrated that the rate of surgically treated acute Achilles tendon ruptures is increasing in the United States.¹²⁷

CHRONIC ACHILLES TENDON RUPTURE

More than 20% of patients with an Achilles tendon rupture are missed on initial examination,²³ and it therefore is not

uncommon to diagnose a late injury. Chronic Achilles tendon ruptures generally present for delayed diagnosis or after a failed acute repair. Chronic ruptures typically are defined as those diagnosed more than 4 to 6 weeks after initial injury.^{124,128} After this period, the gap between the separated tendon ends fills with fibrinous material. This scar tissue contains disorganized fibroblasts and does not possess the same biomechanical strength as normal tendon. Over time, the tissue will elongate and lead to further functional weakness.²³ Those patients treated with nonoperative modalities or those with a failed acute repair can also fall into this group and can develop pain, weakness, and dysfunction due to the elongation of the tendon.¹²⁹

Typically, a patient will complain of loss in push-off strength and be unable to perform toe walking and repetitive heel rise. When the patient lies prone, the injured extremity will demonstrate less resting plantarflexion tone as compared with the contralateral ankle. The involved ankle will display a relative increase in passive dorsiflexion and significantly less plantarflexion with calf squeeze. A palpable tendon gap is not typically evident, but the contour of the tissues will be altered, with thickening and loss of defined margins. The appearance of the affected calf muscle can be different from the contralateral side. Often the muscle belly is more proximally situated (“balled up”)

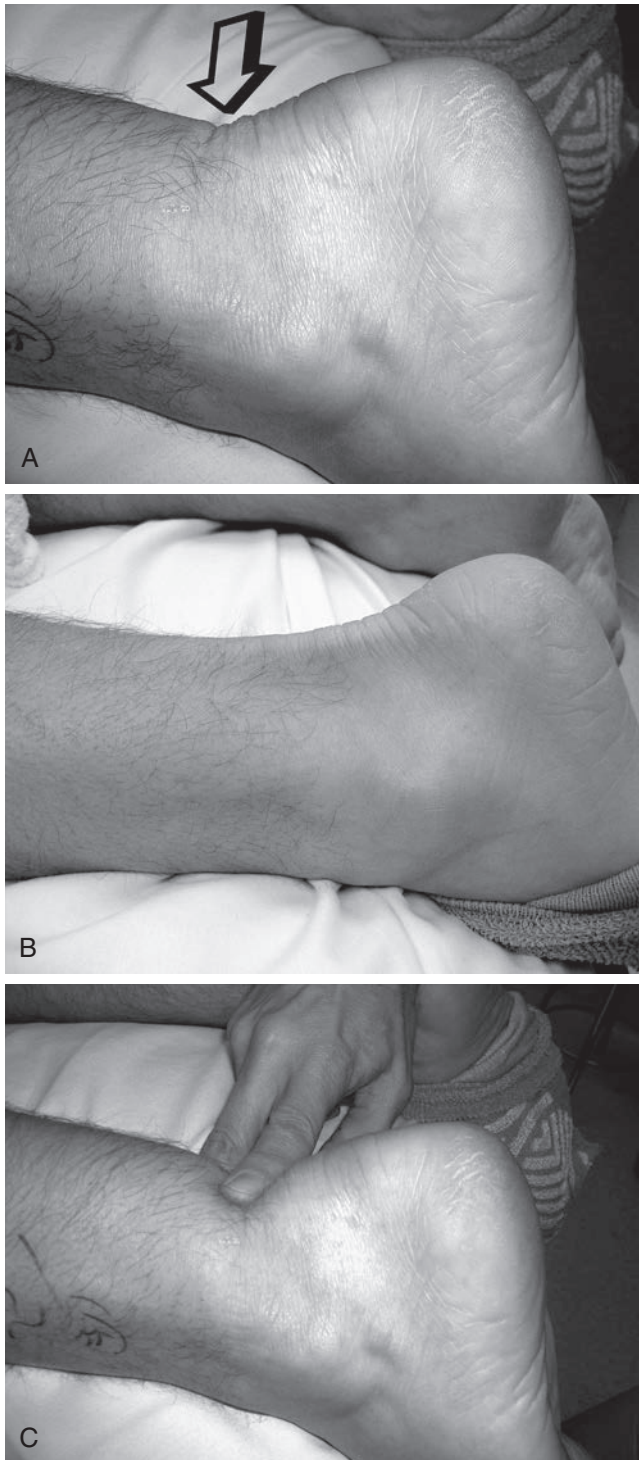


Fig. 9.30 (A) The side with the Achilles rupture has a visible indentation (arrow). (B) The normal side. (C) The ruptured side has a palpable defect.

as a result of its detachment distally. MRI and possibly ultrasound evaluation are useful in evaluating the size of the tendon gap and assist in surgical planning.

Nonsurgical Treatment

Nonoperative management may be considered in those patients without functional deficits or high-risk patients, but surgical management is certainly the treatment of choice for the athlete.



Fig. 9.31 A lateral radiograph shows an avulsion fracture in a patient with a history of retrocalcaneal bursitis. There was no history of steroid injection.

Since the improved results seen with nonoperative treatment using functional rehabilitation and early weight-bearing are based on diagnosing and treating the Achilles tendon rupture acutely, nonoperative treatment has little place in the management of athletic individuals with this injury.

Surgical Treatment

The goals of operative reconstruction are to restore anatomic musculotendinous length and restore strength and endurance. Regardless of the specific procedure used, pharmaceutical anticoagulation should be used for repair or reconstruction of chronic ruptures, as a significantly higher rate of venous thromboembolism has been shown to occur following surgical intervention for chronic versus acute Achilles tendon ruptures.¹³⁰

Due to the scarring, muscle retraction, and atrophy, direct repair is not always possible for the chronic Achilles tendon tear. The type of tendon reconstruction will depend on the size of the residual gap after debridement of scar tissue. Myerson described a scheme for surgical treatment of chronic Achilles tendon ruptures on the basis of defect size (Table 9.2).¹²⁴ Outcomes for surgical treatment options for chronic ruptures are limited mostly to small case series and case reports.

Primary Repair

Primary repair is an option in cases of chronic ruptures with less than 2 cm of retraction or in cases where the tendon has healed in an elongated position. Porter et al. published a report of 11 patients with neglected Achilles ruptures (between

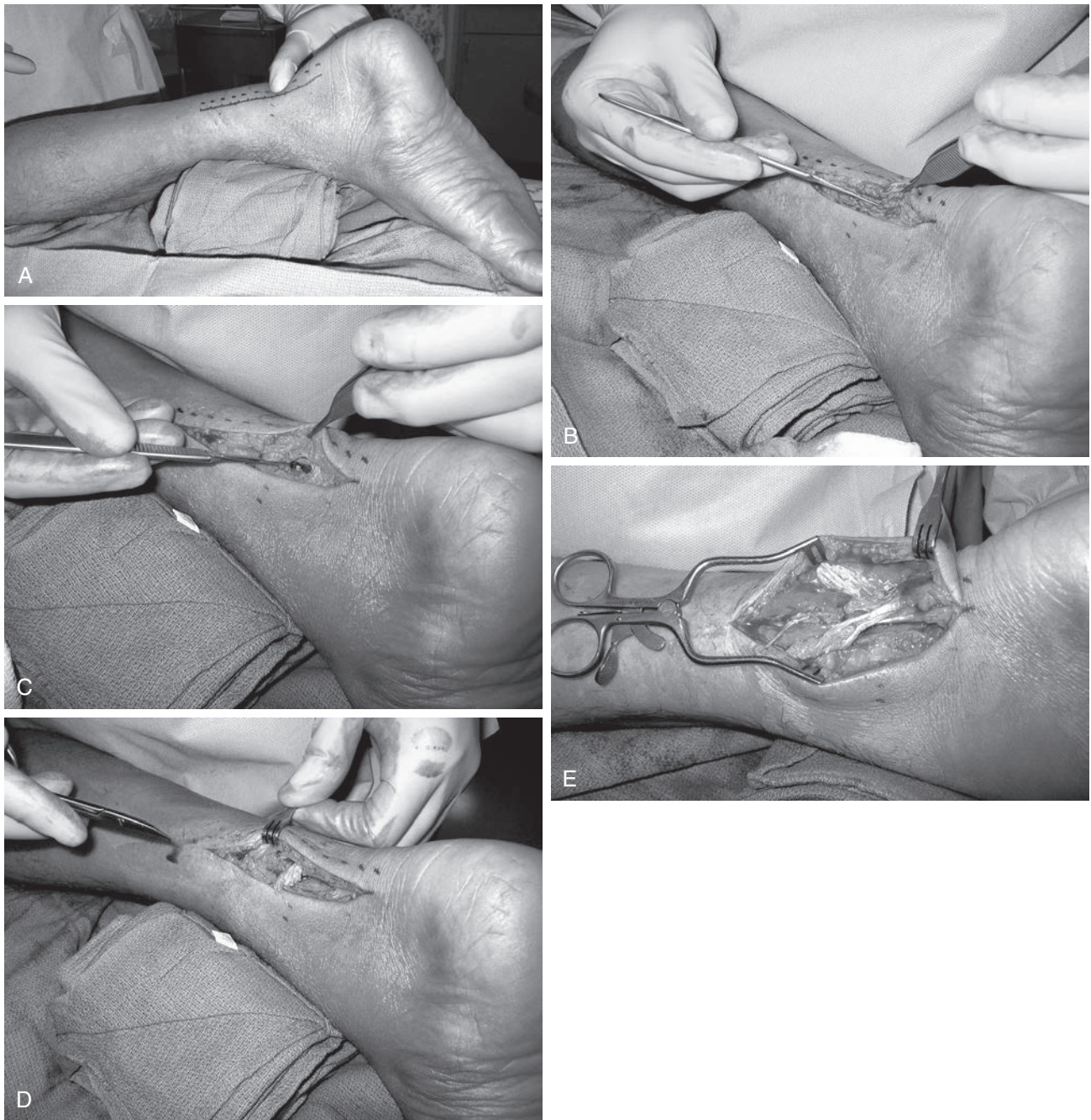


Fig. 9.32 (A) An acute Achilles tendon rupture repair is performed with the patient in a prone position. (B) A medial approach made 1 cm anterior to the Achilles tendon avoids injury to the sural nerve. (C) The approach is with the scalpel, avoiding blunt dissection. (D) The exposure is through relatively thick tissues, which are biomechanically better suited to provide a closure that provides a barrier to the tendon, which is 1 to 1.5 cm away from the incision. (E) The exposed mop ends of the tendon.

TABLE 9.1A General Guidelines for Postoperative Achilles Tendon Rehabilitation Following Surgery

POSTOPERATIVE REHABILITATION PROTOCOL	
Time	Therapy
Weeks 1–2	Nonweight bearing in a postoperative splint
Weeks 3–6	Walking boot with 4 heel wedges, start 4-week weight bearing progression, removal of 1 wedge per week, allowed to start active plantarflexion and dorsiflexion up to 5–10 degrees short of neutral. Formal PT can start at this time for range of motion (active and active-assisted, not passive)
Week 7	Wean from boot to shoe with 2 wedges, remove 1 wedge per week
Week 8	Start functional PT with sports progression
Weeks 12–16	Limit activities in athletes to practice. Risk of rerupture persists up to 4 months
Week 16	Start controlled practice with pain as guide
Months 6–12	Athletes able to return to full preinjury level of activity as symptoms allow

PT, physiotherapy

TABLE 9.1B Achilles Tendon Rupture Postoperative Protocol*

General Instructions

- The goal of this surgery is to return you to full strength, power, function, and comfort in the shortest amount of time. This time will vary from person to person. Following the guidelines outlined here give you the best chance for a full recovery.
- You will need to use crutches and a walking boot for 2–6 weeks. You will receive crutches (if you don't already have them) after surgery. You will receive a walking boot brace at your first postoperative visit. This boot brace will be worn when walking for up to 8 weeks. The boot may be one that allows for the ankle to move in a downward direction (plantarflexion) but will limit its ability to go in an upward direction (dorsiflexion) or you will be in a fixed-position boot with heel lifts that will be removed weekly and you will be able to remove the boot and work on moving the ankle within specific ranges using your muscles as described below.
- Avoid squatting, lunging, rising up on toes, bouncing, or jumping motions. Do not roll through the foot when walking for approximately 8–12 weeks. Reruptures of the repaired Achilles tendon are rare, but can occur between 6 weeks and 4 months after surgery. Wear your boot, brace, or heel lifts as prescribed.
- Weight bearing progression in the boot will begin following the first postoperative visit and you will receive a written protocol for guidance along with instructions by your physical therapist or athletic trainer.
- Do not force upward motion of the ankle. The tendon is repaired with tension applied which will limit your ankle's upward motion initially. This will gradually improve and allow more upward motion with time and therapy. If you overstretch the tendon before adequate healing has occurred, you increase your risk of losing strength, power, and function. It is better for it to be too tight rather than too loose.
- Compression socks that are at or near medical grade are beneficial for 3–6 months following surgery, especially when active during the day, at work, or exercising.
- Achilles tendons take considerable time to heal whether you are an elite athlete or nonathletic. The general rate of healing is 75% healed by 3 months and 90% healed by 6 months. Attempts to push for early return to high stress activities risk re-injury or stretching of the surgical repair. Some individuals take as long as 1–2 years to reach maximum improvement.
- Postoperative rehabilitation is best done under the direction of a physical therapist or athletic trainer knowledgeable in Achilles rehab. The advancement of exercises introduced over time is partly related to the standard time necessary for certain tissues to heal and gain strength, and in later stages of rehabilitation, this becomes more dependent on reaching specific goals for motion, strength, power, agility, and balance. Different individuals reach these goals in varying time. Rehabilitation involves retraining and conditioning the entire body, not just the Achilles tendon or foot and leg.

Postoperative Course

Day 1–4

- The foot will be wrapped in a plaster splint with lots of padding and a removable bandage. If for any reason your splint is uncomfortable or too tight, remove the ace wrap, loosen padding, and re-apply to your comfort level.
- It is important to ice and elevate the foot, take pain medication, and rest as needed.
- Expect numbness in the ankle for 4–12 hours and then anticipate the onset of pain. Your strongest pain will be the first 3–4 days after surgery.
- No weight bearing on injured foot.
- Do not get the foot or splint wet.
- Moving toes and moving the ankle to the degree allowed within the splint and trying to tighten your muscles (isometric contractions) in the leg, foot, and ankle help reduce swelling.
- As you feel better, you may perform exercises such as leg lifts and core exercise.

Day 4–10

- Pain should improve after the third day. If your pain has worsened since day 3 or you have a fever and/or chills, please call the doctor's office.
- Continue exercises as guided by the doctor's office, but with no weight on the surgical leg.

Continued

TABLE 9.1B Achilles Tendon Rupture Postoperative Protocol^a—cont'd

- Showers are possible if you can sit in the shower with the splinted leg in a water-impenetrable bag. Commercially available bags can come from the hospital, outpatient surgery center, or doctor's office. An alternative is the use of 2 plastic medium-size trash bags with each one sealed at the top with Duct Tape (one bag will usually leak).

Day 10 –to 3 weeks

- First postoperative visit in the office.
- Your sutures will be removed and the dressing will be changed.
- You will be placed in a walking boot brace with your ankle fixed in the position of slight tension on the tendon repair. This may be anywhere from 10–20 degrees of downward positioning (plantarflexion), but it should not be painful.
- You can remove the boot and begin movement exercises with your muscles. Begin drawing the alphabet with the foot and ankle and move the ankle up and down. The ankle should not move above a right angle to the leg (neutral position) during this time.
- Using a towel, do inversion, eversion, and toe crunch exercises.
- Your physical therapist/athletic trainer will work with you on these exercises and others.
- If the incision is healing well, you may get the foot wet in the shower 2 days after your first postoperative visit.

3–6 weeks

- Once the incision is fully healed, you may submerge the ankle in water in a bath or pool.
- Gentle exercise for conditioning on a stationary bike is permitted in the boot. Start with no resistance.
- Exercising in the pool is permitted (primarily deep-water exercise) with buoyancy conditions (e.g., pull buoys); however, swimming, pushing off, and kicking are not permitted.
- Walking in boot without crutches is permitted if stable and nonpainful.

6–8 weeks

- Boot brace comes up into neutral position or fixed boot should now have no heel lifts.
- Biking without boot is permitted in shoe with heel lift or pedal positioned toward heel.
- Swimming is permitted. No wall push-offs.
- Pool program can include gait retraining and heel raises in shoulder depth water for buoyancy. Start with double leg heel raises and progress to single leg as tolerated.

8–12 weeks

- May begin to wean out of boot brace gradually.
- Avoid lunging, squatting, jumping, or single heel raises.
- Walk progressively more as tolerated.
- Biking with resistance as tolerated.
- Treadmill on level surface.
- Balance retraining program.
- Pool therapy: walking, heel raises, gentle hopping, and jumping starting in shoulder depth and progressing to waist deep water.

12–16 weeks

- Treadmill walking on increased incline, gradual progression to jogging if symptom free.
- Road biking permitted.
- Pool therapy: pushing off as tolerated. Avoid diving and especially diving board.
- Progressive submaximal dorsiflexion and plantarflexion for endurance.
- Closed chain strength program. Start double leg heel raises.
- Continue balance on dynamic surfaces.

16–20 weeks

- Individualized strength and flexibility program.
- Progress to running but not sprinting.
- Progress resistive exercises to body weight exercises such as repeated heel raises.
- Progress proprioceptive and balance training.

20–24 weeks

- Sport-specific training with careful monitoring in accordance to pain and swelling.
- Running, jumping, and squatting are introduced and advanced.

^aThe detailed postoperative course and rehabilitation protocol used for patient education by the authors.

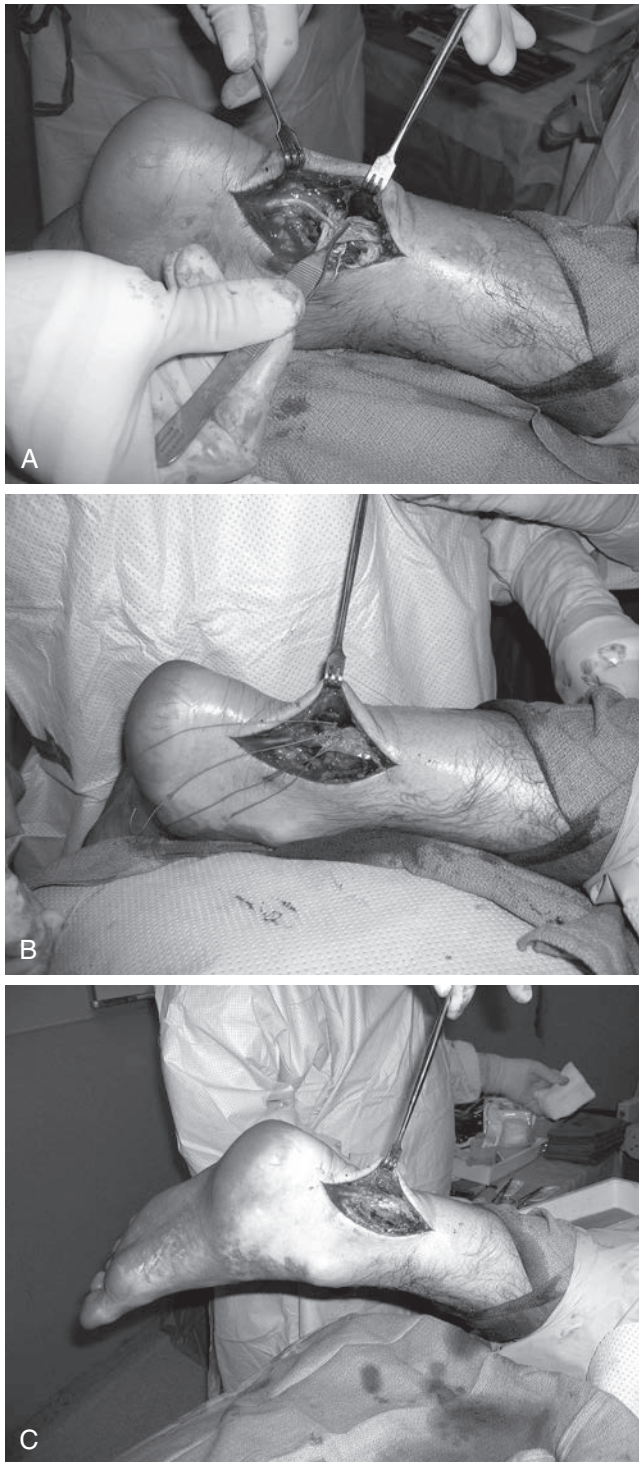


Fig. 9.33 (A) Another acute Achilles repair with exposure of the frayed tendon ends. (B) Care is taken to minimize soft-tissue handling. A Krackow-type stitch technique with nonabsorbable, no. 2 sutures is used to reapproximate the “mop end” rupture. (C) The edges of the anastomosis should be made neat with a 2-0 or 4-0 absorbable suture. The resting tension should be restored.

4- and 12-weeks postinjury) treated with proximal release of the gastrocnemius–soleus complex, imbrication of the early fibrous scar without excision of local tissue, and direct primary repair of the tendon. All patients were reported to have returned to their preinjury level of activity at a mean of 5.8

TABLE 9.2 Guideline for Treatment of Chronic Achilles Tendon Tears Based on Defect Size

Defect Size	Surgical Procedure
1–2 cm	End-to-end anastomosis and posterior compartment fasciotomy
2–5 cm	V-Y lengthening, augmented with tendon transfer, if needed
>5 cm	Tendon transfer alone or in combination with V-Y advancement or turnaround

Myerson, M. Achilles tendon ruptures. *Instr Course Lect* 1999;48:219–30. Reproduced with permission and adapted from Myerson M: Achilles tendon ruptures. *AAOS Instr Course Lect*. 1999;48:219–30.

months (range, 2.8 to 9 months).¹³¹ In a more recent study, Yasuda et al. performed a similar procedure in 30 patients with chronic ruptures ≥ 4 weeks old by utilizing resection of scar tissue (15 to 50 mm in length) between the healed stumps of the tendon prior to a direct repair of the tendon with No. 2 nonabsorbable polyfilament Krackow stitches. Mean AOFAS scores increased from 82.8 preoperatively to 98.1 postoperatively. At latest follow-up, no patients experienced reruptures or difficulty walking or climbing stairs. All 14 athletes (including three competitive) returned to their preinjury levels of participation.¹³²

V-Y Advancement

V-Y advancement is typically performed in cases of 2 to 5 cm defects.¹³³ An inverted V is incised on the proximal stump of the Achilles tendon with the apex of the V at the tendinous portion of the myotendinous junction. The arms of the V are made with a length that is 1.5 times that of the defect size. With larger defects (>5 cm), the arms of the V should be twice the defect size. (see Fig. 9.18). The proximal Achilles tendon is pulled carefully and slowly so that the myotendinous junction is stretched and not torn to allow suture repair of the two segments.¹³⁴

While studies of V-Y advancement alone are lacking, several studies combine the procedure with further reinforcement.^{134,135} Guclu et al. performed V-Y advancement with fascia turnaround in 17 patients with chronic Achilles tendon ruptures at an average of 7 months postinjury (range 4 to 12 months). There is no mention of the number of athletes. Mean defect size was 6 cm following debridement but ranged from 4.5 to 8 cm. AOFAS scores improved from a mean of 64 preoperatively to 95 postoperatively with no reruptures at an average follow-up of 16 years (range 13 to 19 years).¹³⁵ In another case series, Elias et al. described improved outcomes in 15 patients undergoing V-Y advancement and flexor hallucis longus (FHL) tendon transfer for gaps larger than 5 cm with an average of 2-year follow-up (range 1 to 4.5 years). While no patients in the follow-up portion of the study were reported to be athletes, one surgical patient did not attend follow-up and was dropped from the study because he was playing professional basketball in Europe.¹³⁴

For more chronic cases seen more than 12 weeks after a missed rupture or after a previous completely failed repair, a

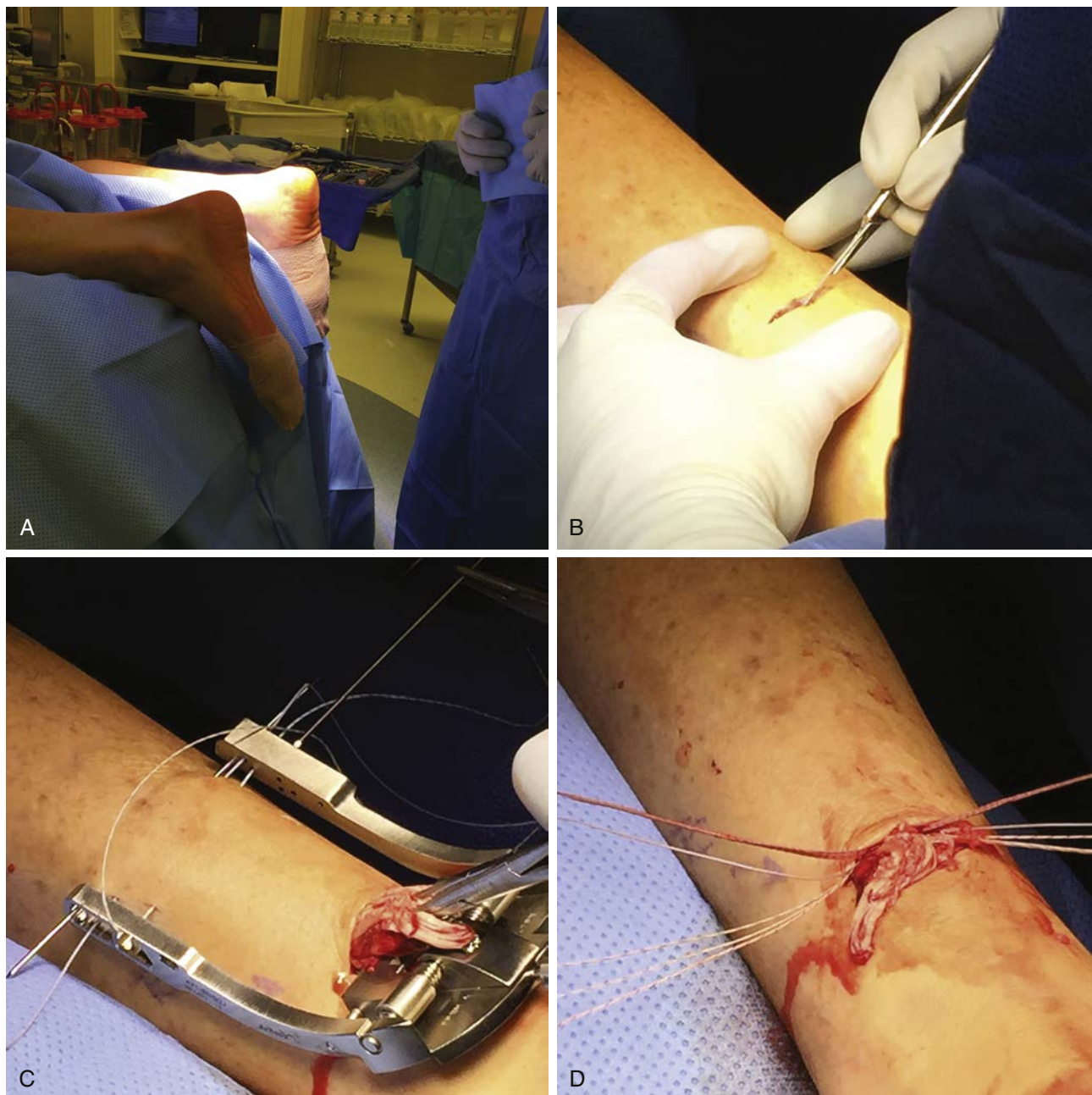


Fig. 9.34 (A) Acute rupture showing preoperative loss of carrying angle. (B) Transverse incision at rupture site for mini-open technique. (C) Percutaneous Achilles Repair System (PARS) jig in place for suture passage through proximal stump. (D) Sutures passed through Achilles' proximal stump within paratenon and brought out of transverse incision.

V-Y advancement or turndown likely will be required, depending on the size of the defect after repair. For defects between 2 and 3 cm, a V-Y advancement is possible, as mentioned previously. For defects longer than 3 to 5 cm, a turndown procedure with possible tendon augmentation is required, as discussed earlier (see Fig. 9.19).

Tendon Transfer

Chronic Achilles tendon ruptures are typically more difficult to repair than acute ruptures due to retraction of the tendon ends.¹³⁶ For those cases with preoperative atrophy of the

gastrocnemius/soleus muscle, an FHL or FDL tendon transfer may be considered (see Figs. 9.16 and 9.17). An FHL tendon transfer is most commonly used,¹³⁷ though a peroneus brevis tendon transfer can also be effective.¹³⁶ In addition to mechanical support, the FHL transfer provides additional blood supply to the ruptured tendon.¹³⁷ However, there are arguments that FHL harvesting eventually reduces push-off during the stance phase.¹³⁸ Wapner et al. were the first to report on a technique for harvesting the FHL tendon through an additional medial midfoot incision at the knot of Henry and transferring the tendon through the calcaneus.¹³⁹ This approach can put the



Fig. 9.34 cont'd. (E) Drill hole in lateral calcaneus for anchoring sutures in the knotless technique. (F) Restored carrying angle postrepair with anchors in medial and lateral calcaneus. (G) Position of slight tension on repair for postoperative splint.

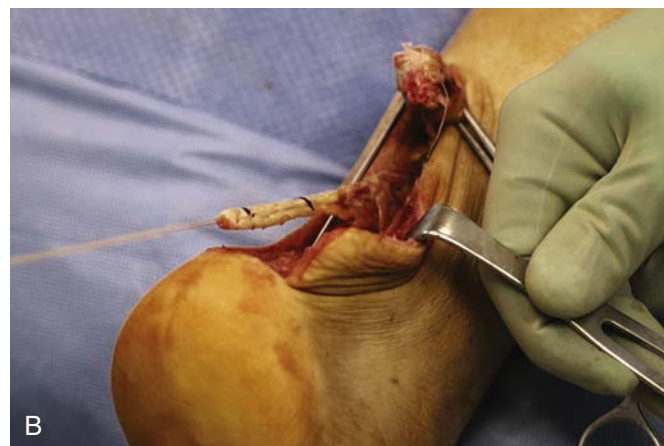


Fig. 9.35 Single incision FHL Transfer technique from a medially base "J" approach. (A) Exposure of flexor hallucis longus (FHL) tendon. (B) Harvested FHL tendon from deep within medial tunnel in single incision technique. (C) FHL tendon fixed in calcaneal tunnel with interference fit screw.

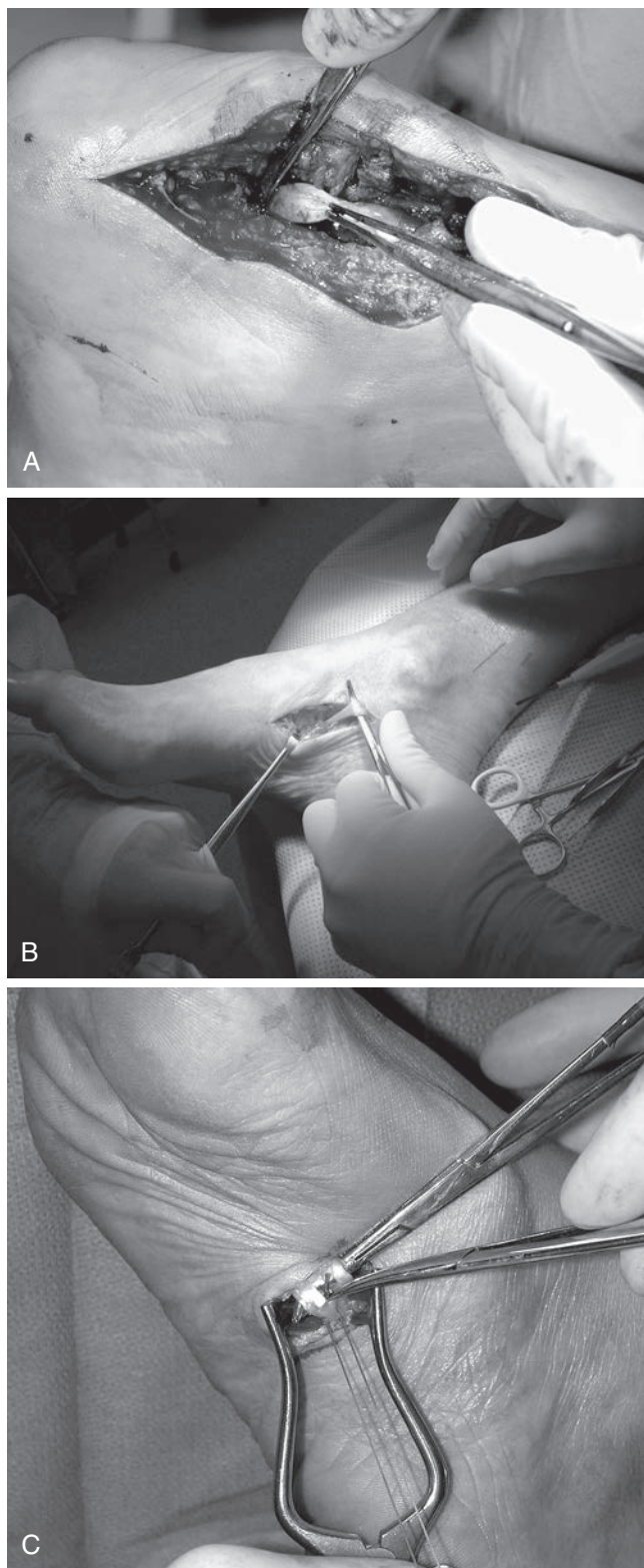


Fig. 9.36 (A) The flexor hallucis longus (FHL) can be harvested from the posterior ankle in the depths of the posterior approach to the Achilles. (B) The graft harvest through a medial approach just plantar to the posterior tibial tendon and the talonavicular joint. (C) The graft harvest through the plantar aspect of the foot. After incising the plantar fascia and reflecting the medial plantar nerve, the FHL and flexor digitorum longus (FDL) tendons can be found next to the bones. The FHL is sutured to the FDL distal to where the tendon will be transected. Next, the FHL is cut proximal to the tenodesis and withdrawn out the proximal ankle incision.

medial plantar nerve at risk with dissection. Newer technologies such as interference fit screw fixation, bone suture anchors, and endobuttons have allowed surgeons to achieve strong bony fixation with less tendon harvest length through a one-incision technique (Fig. 9.35).^{140,141}

The FHL can be harvested from the posterior ankle in the depths of the posterior approach (Fig. 9.36A) to the Achilles, or, if a longer graft is felt to be advantageous, from the arch of the foot. The graft harvest can be performed through a medial approach just plantar to the posterior tibial tendon and the talonavicular joint (Fig. 9.36B) or through the plantar aspect through the plantar fascia (Fig. 9.36C). For the latter approach, once the incision is made through the plantar fascia, care is taken to avoid the medial plantar nerve. The FHL and FDL tendons can be found in the depth of the wound next to the first metatarsal and medial cuneiform. Once the tendons have been identified, the FHL is sutured to the FDL distal to the point at which the tendon will be transected (at or near the knot of Henry). A suture placed in the FHL proximal to the transection is helpful in case the tendon does not pull through into the proximal wound.

Den Hartog¹⁴² described a technique of harvesting the FHL tendon near the tip of the medial malleolus for chronic Achilles tendinosis. Good to excellent results were reported in 23 of 26 treated patients without any deficit to first-toe function. A recent biomechanical study showed little pressure change under the first or second metatarsophalangeal (MTP) joint and no clinical functional deficit after FHL harvesting.¹³⁸ Prior studies also showed promising clinical results.^{139,143}

Autograft/Allograft Reconstruction

Chronic Achilles tears can also be managed by free autologous gracilis tendon grafts. In one study, this technique was reported as a safe but technically demanding procedure, and it was noted that plantarflexion strength could be significantly reduced compared with the contralateral side.¹⁴⁴ Donor site complications should be kept in mind when considering reconstruction with allograft tendon grafts (Fig. 9.37)

Synthetic augmentation has been reported to result in a strong reactive immune response after undergoing revision Achilles tendon surgery. Adverse immune reactions may lead to catastrophic complications that may eventually require removal of the graft.¹⁴⁵

CONCLUSION

Achilles tendon disorders are common in the athlete. If diagnosed early, the process usually is a tendinopathy and is amenable to nonoperative treatment such as intermittent immobilization, stretching, modalities such as ultrasound and iontophoresis, and use of anti-inflammatory medication. More chronic cases take longer to treat and have a higher risk of requiring more invasive intervention such as the shockwave, PRP, bone marrow concentrate, growth factors or operative intervention. Operative treatment typically is 70% to 90% successful but requires 3 to 6 months for return to athletic participation.

Achilles rupture typically will require operative treatment in the athlete, and 6 to 9 months can be a typical recovery period.



Fig. 9.37 (A) Achilles tendon reconstruction of chronic injury with large gap utilizing an Achilles tendon allograft with bone block. (B) Allograft tendon fixated to calcaneus with cancellous screws and attached to proximal stump with Pulvertaft weave. (C) Completed reconstruction. (D) Incision closure. (E) Lateral radiograph showing bone block fixation.

A wide variety of options are available for treatment of chronic Achilles tendon ruptures and gap size is a general guide for appropriate treatment.^{124,129,146} Beyond that, the use of a tendon transfer with the FHL, FDL, or peroneus brevis tendon versus an autograft reconstruction have the most support.¹²⁹ Careful consideration of individual patient circumstances together with the experience of the surgeon are critical factors in surgical management of chronic Achilles tendon ruptures since these patients tend to have more complications and worse outcomes in comparison with acute tears.^{129,146}

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 **Video9.1** <https://www.kollaborate.tv/link?id=5c9d1b2b536f4>
The Thompson Test for Evaluation of Acute Achilles Rupture The Thompson Test assessing for plantarflexion to evaluate the Achilles tendon. Good plantarflexion in the right lower leg indicates an intact Achilles, while lack of plantarflexion on the left indicates a positive Achilles rupture

Posterior Tibialis Tendon Injury in the Athlete

Kenneth J. Hunt

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INTRODUCTION

The tibialis posterior muscle and its tendon (PTT) play a vital role in most athletic activities. The tibialis posterior is the strongest inverter of the foot, it locks the triple joint during gait progression facilitating a rigid lever of push-off, and provides both power for acceleration and control for deceleration. Due to these important and repetitive roles, acute and chronic injury to the PTT are commonplace in athletes. A knowledge of disease progression, risk factors, and treatment measures is vital for treating physicians.

Acute posterior tibialis tendon injury in the athlete is rare^{1,2,3} but must always be considered in the differential diagnosis when an athlete presents primarily with tenderness, swelling, and pain over the medial ankle or plantar medial midfoot. Antecedent to the acute presentation, there often is a history of less severe prodromal symptoms more consistent with a chronic tendinopathy or tendinosis. In most cases, there are identifiable mechanical or physiologic risk factors that contribute to this development. The chronic picture is seen more often in the middle-aged to elderly athlete.⁴⁻⁶ While most of the literature on the topic focuses on chronic PTT dysfunction,^{1,2,7-10} it is important to identify acute injuries and understand them in the context of the individual athlete.

ANATOMY AND BIOMECHANICS

The tibialis posterior muscle occupies the deep posterior compartment of the leg, originating along the proximal one third of the tibia and intraosseous membrane. Distally, its tendon travels posterior, then inferior, through the medial malleolar groove, changing direction abruptly almost 90 degrees. The stout retinaculum of the

long flexors prevents the tendon from subluxing over the medial malleolus.¹¹ Because the posterior tibialis tendon lacks a mesotenon, there is an area of relative hypovascularity from this acute turn at the medial malleolus to the medial navicular insertion. These factors of hypovascularity and the mechanical stress of a relatively acute turn of the tendon as part of a strong, weight-bearing leg muscle (second only to the gastrocnemius), make the tendon predisposed to acute and overuse injury in this area.

Because the posterior tibial tendon travels posterior to the axis of the ankle and medial to the axis of the subtalar joint, it functions as a strong ankle plantarflexor and foot inverter via the transverse tarsal joint (talonavicular and calcaneocuboid joints).¹² The tendon also has multiple slip attachments to the capsule of the naviculocuneiform joint, all three of the cuneiforms, the cuboid, and their respective metatarsal bases in the plantar arch.^{5,13} The posterior tibialis tendon therefore is primarily a midfoot inverter and dynamically supports and elevates the medial longitudinal arch. It also indirectly supports the hindfoot because of its medial malleolar pulley action and intimate relationship to the deep deltoid ligament, plantar medial talonavicular joint capsule, and spring ligament (calcaneonavicular ligament).¹⁴ The dynamic role of the PTT places it at risk of elongation and degenerative tearing or even rupture.

The resulting loss of dynamic function can lead to progressive loss of static support structures, such as the ligaments and plantar capsules of the midfoot joints, and ultimately a pes planovalgus deformity (Fig. 10.1). As the deformity progresses over time, other structures are affected, including the talonavicular joint capsule, deltoid ligament, and spring ligament. The stretching out or even frank rupture of these structures eventually leads

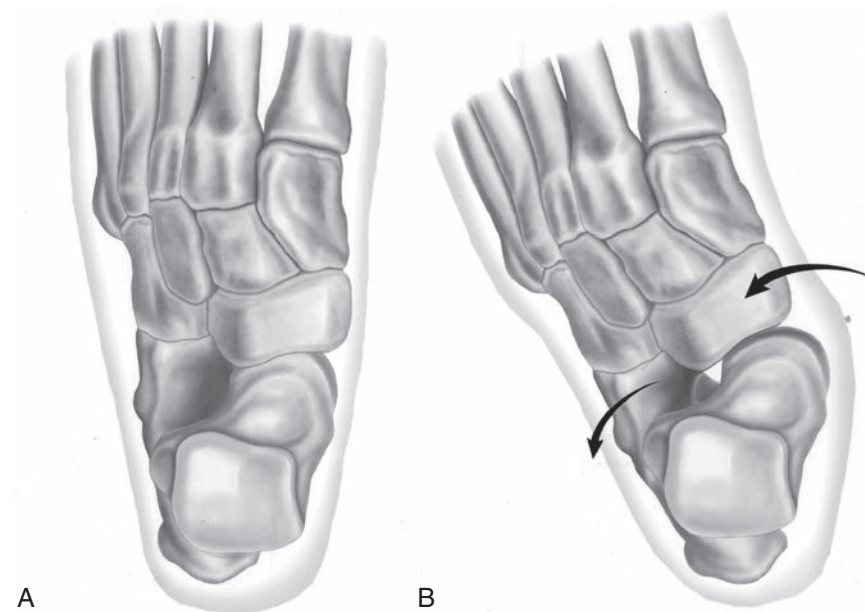


Fig. 10.1 Dorsal-plantar view demonstrating the normal foot (A) and the posterior tibialis tendon incompetent foot (B). With external rotation or abduction of the forefoot, the medial talar head becomes more uncovered by the navicular as it rotates externally. The calcaneus also secondarily rotates externally and tilts into more valgus.

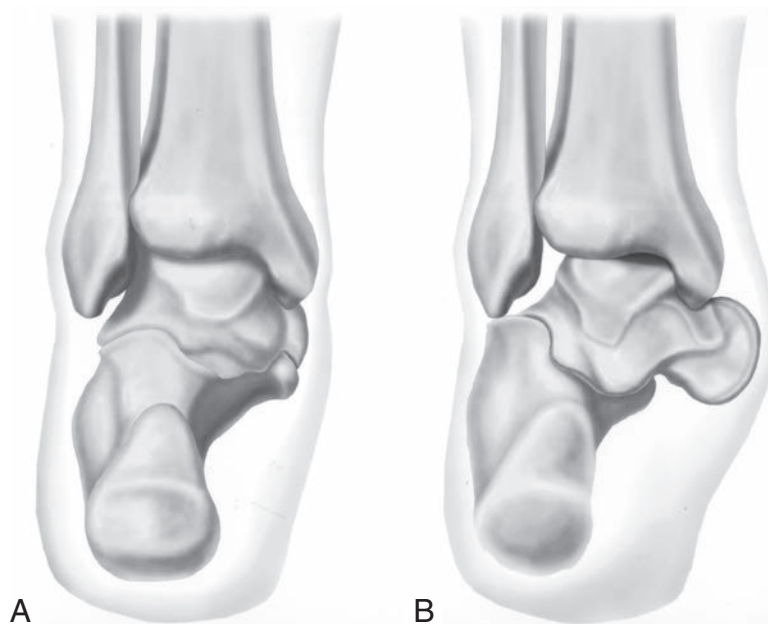


Fig. 10.2 Posterior-anterior view of the normal (A) and posterior tibialis tendon incompetent ankle and hindfoot (B). With external calcaneal rotation, the talar head translates plantarward. This also leads to increased valgus tilting of the calcaneus and subfibular or sinus tarsi impingement.

to a more severe valgus inclination of the hindfoot and external rotation of the calcaneus, also associated with contracture of the triceps surae as it transitions into a hindfoot everter (Fig. 10.2).¹² Clinically, this may result in impinging pain and swelling in the subfibular or sinus tarsi area as the calcaneus abuts against the lateral malleolus. In very severe or neglected cases, a valgus tilt of the ankle may be seen as the deltoid ligament becomes incompetent, and asymmetric valgus arthritis can result.

DIAGNOSIS

Usually, a detailed history, careful physical examination, and weight-bearing radiographs will establish the diagnosis of a posterior tibialis tendon injury. Additional imaging can help to further elucidate the structures involved and extent of disease, but is usually not necessary to reach a diagnosis and classification.

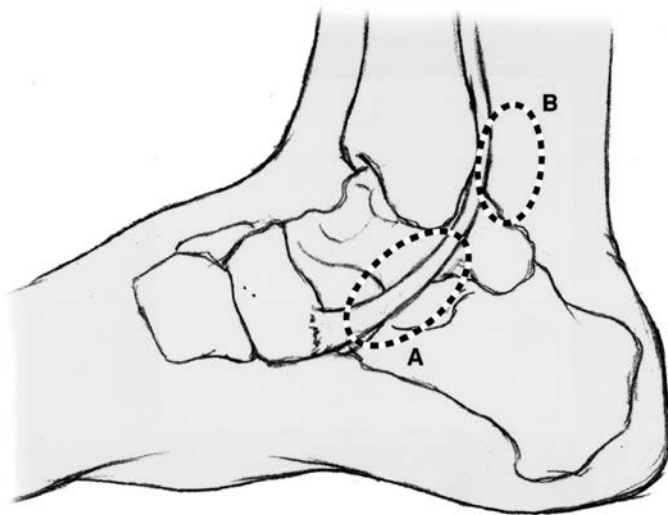


Fig. 10.3 Usual area of maximal tenderness and swelling along the terminal course of the posterior tibialis tendon between the medial malleolus and medial navicular insertion. Occasionally, this can extend to the area posterior to the medial malleolus.

History and Questions to Be Answered

Sometimes the history alone will provide the physician with enough information to suggest the diagnosis. Athletes will complain of pain on the medial aspect of the hindfoot, below the medial malleolus. While the chief complaint is often “ankle pain,” a careful history can isolate the pain to the medial sub-malleolar region. Since conditions of the PTT can occur insidiously, acutely, or a combination of the two, it is important to explore common etiologies.

Acute Pain or Injury

- What was the mechanism of injury?
 - Did the foot sustain an eversion twisting injury, especially on impact from tripping or a fall?
 - Conversely, was there a sudden increase in the level of athletic activity temporally related to the onset of symptoms?
- Were there prodromal symptoms suggesting possible tendon degeneration before the acute injury?

Chronic or Acute on Chronic

- Has the athlete noticed that the arch on the involved side is “flatter,” the foot is “turned out,” the ankle “turned in,” or complained that the injured foot is “weaker”?
- When barefoot on a hard, wet floor surface, such as at bath time, does the patient notice a wider footprint or a “sucking sound” because of a vacuum effect of the collapsed arch on the symptomatic side?
- Has the athlete noticed more medial shoe sole wear or “running over” the medial vamp?
- Does the athlete have any history of gout, pseudogout, or autoimmune disease?
- Are there sensory (dysesthesias or paresthesias) complaints?

Predisposing Factors

- Does the patient have a preexisting and/or progressive pes planus deformity?
- Is there a history of oral steroid use, injected steroids in the area of the tendon?
- Does the patient have a history of, or risk factors for, diabetes mellitus?
- Does the patient have vasculopathy, obesity, or active tobacco use?

Physical Examination and Questions to Be Answered

For comparison, both unclothed and unshod lower extremities from the midhigh distally to the toes should be carefully examined.

- Is there a valgus knee deformity (genu valgus) of the symptomatic side? (This may precipitate or exacerbate posterior tibialis tendon dysfunction, especially if chronic.)
- Is there tenderness and swelling along the PTT, particularly as it courses between the medial malleolus and the navicular insertion, or the posterior aspect of the medial malleolus (Fig. 10.3A–B)?
- In viewing the weight-bearing patient from front and behind, is there increased forefoot abduction or pronation (the “too many toes sign”) (Fig. 10.4)? In addition, is the symptomatic hindfoot in valgus compared to the contralateral side?
- Does the patient have difficulty heel rising with all of his or her weight on the injured side (“single foot heel rise” test), or if he or she is able to heel rise, does the hindfoot fail to invert or invert less than the normal side (Fig. 10.5)? It is important to determine the contributors of pain and weakness.
- Is there tenderness and swelling laterally in the sinus tarsi or subfibular area, suggesting subfibular impingement, especially in the patient with deformity (Fig. 10.6)?
- Is there pain and/or weakness with resisted inversion compared to the asymptomatic side?
- Is the Achilles tendon and/or gastrocnemius contracted? This can be determined with Silfverskiöld’s test (passive ankle dorsiflexion with the knee extended, tests gastrocnemius tightness, passive ankle dorsiflexion with the knee bent relaxes the gastrocnemius and tests Soleus/Achilles tightness).
- Are there abnormal sensory findings that might suggest peripheral neuropathy, especially in the diabetic patient?
- Is there a positive Tinel’s sign over the tibial nerve in the medial ankle or plantar foot sensory deficits that might suggest a tarsal tunnel syndrome?
- Is there a tender and swollen bony prominence in the area of the medial navicular, suggesting an accessory navicular or bone stress injury?
- Is the medial malleolus itself tender or painful with percussion, suggesting a stress fracture?
- Are there any dysvascular findings (absent posterior tibial or dorsalis pedis pulses, delayed capillary refill, cyanosis, toe hair loss, and dystrophic nail changes)?



Fig. 10.4 View from (A) Posterior and (B) Anterior. The positive “too many toes” sign in the posterior tibialis dysfunctional right foot is appreciated when examining the weight-bearing patient from behind. The forefoot is abducted/pronated and the hindfoot is in greater valgus, resulting in more toes seen laterally in the right foot when compared with the left.

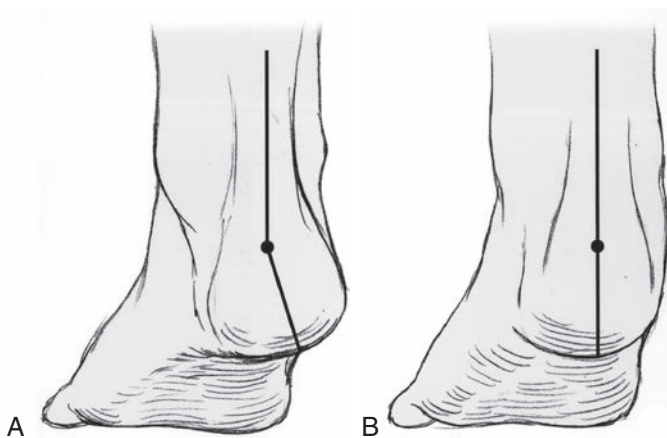


Fig. 10.5 Notice hindfoot inversion in the normal left foot. Although patients with posterior tibialis tendon dysfunction may be able to perform the single-foot heel-rise test, notice that the hindfoot does not invert, or inverts less, than the normal foot. This may be due to some residual function of the posterior tibialis muscle-tendon unit with assistive recruitment of the long toe flexors.

Radiographic Imaging

Ideally, weight-bearing x-rays of the symptomatic foot and ankle should be taken. Also, comparison views of the other foot and ankle often are helpful diagnostically. This includes, at a minimum, the following five views: anteroposterior (AP) foot, lateral foot, oblique foot, AP ankle, and hindfoot alignment (Saltzman) view. If there are concerns about the global alignment of the extremities, a standing x-ray of both legs can be helpful.

Ankle and Foot Radiographs

- Is there flattening of the medial longitudinal arch as measured by calcaneal pitch angle, Meary's angle, and medial arch sag angle (MASA) angle?

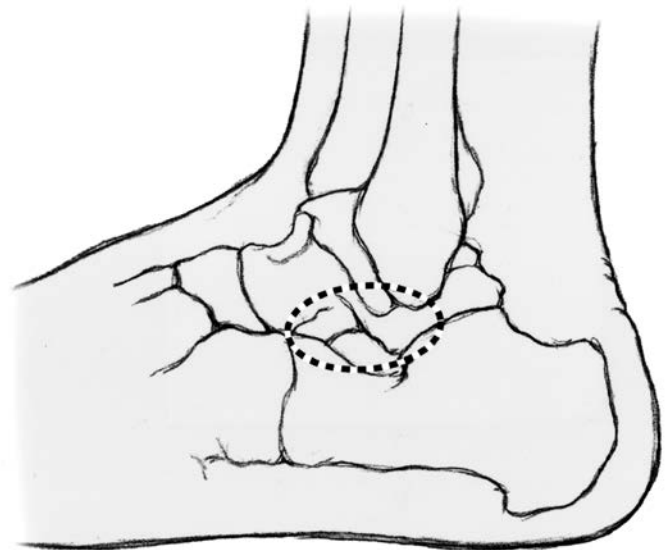


Fig. 10.6 Zone of tenderness and swelling indicative of impingement in the subfibular or sinus tarsi area, often seen with more severe posterior tibialis tendon dysfunction with hindfoot valgus deformity.

- Calcaneal pitch angle: angle measured on a weight-bearing lateral foot radiograph between the supporting surface and a line drawn along the most inferior portion of the calcaneal tuberosity and the most distal and inferior point of the calcaneus at the calcaneocuboid joint. Normal value range is commonly considered 17 degrees to 32 degrees.
- Meary's angle: angle measured on a weight-bearing lateral foot radiograph between the midline axis of the talus and the midline axis of the first metatarsal. Normally Meary's angle is 0 degrees.
- Do films demonstrate a medial or valgus tilt at the ankle ([Fig. 10.7A](#))?

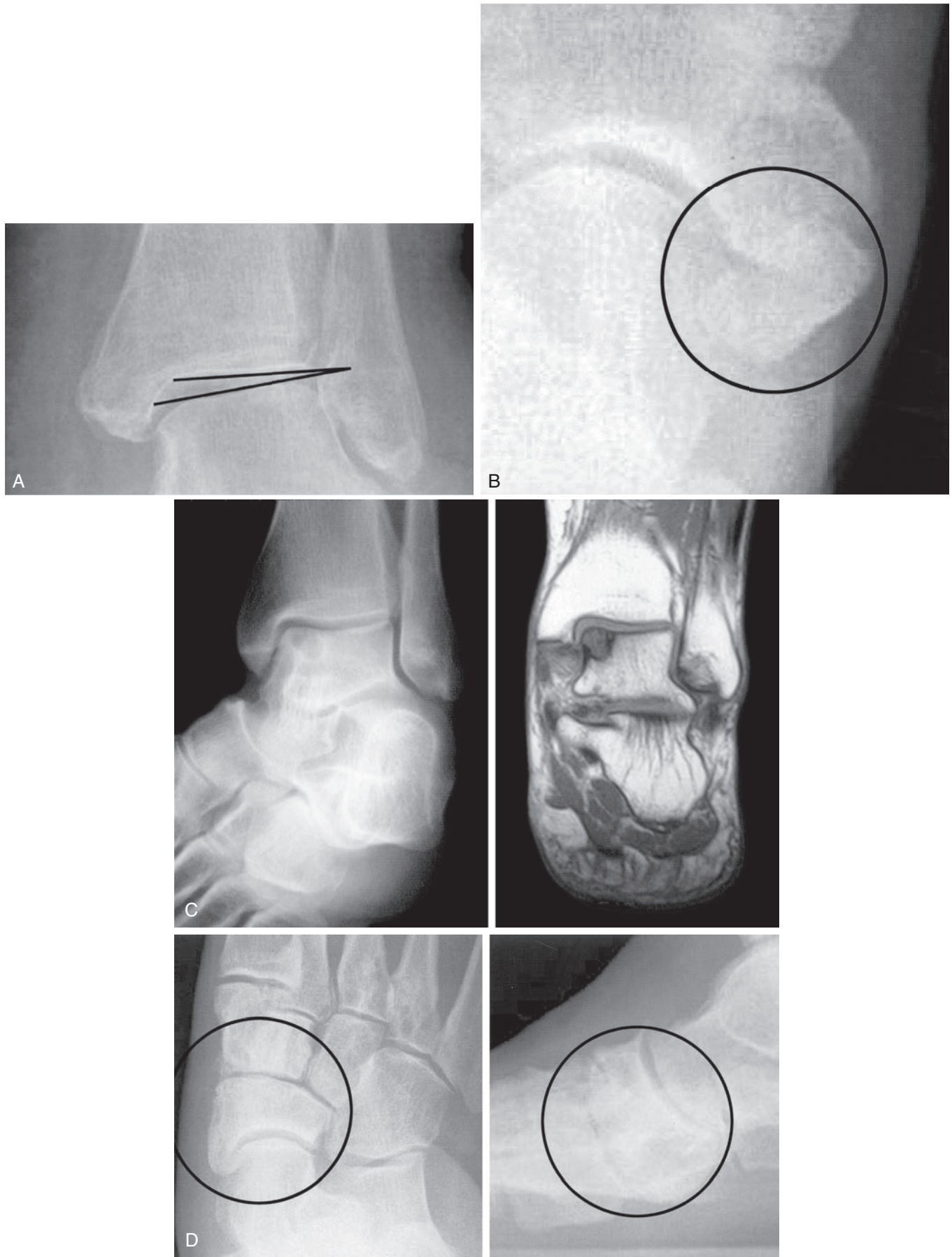


Fig. 10.7 Radiographs of patients with medial ankle or midfoot pain and swelling demonstrating medial talar tilt because of primary deltoid ligament incompetency (A) accessory navicular (B) medial talar dome osteochondritis dissecans with a coronal magnetic resonance imaging (MRI) view (C) and medial column arthritis. (D) All of which can mimic to varying degrees the clinical presentation of posterior tibialis tendon dysfunction.

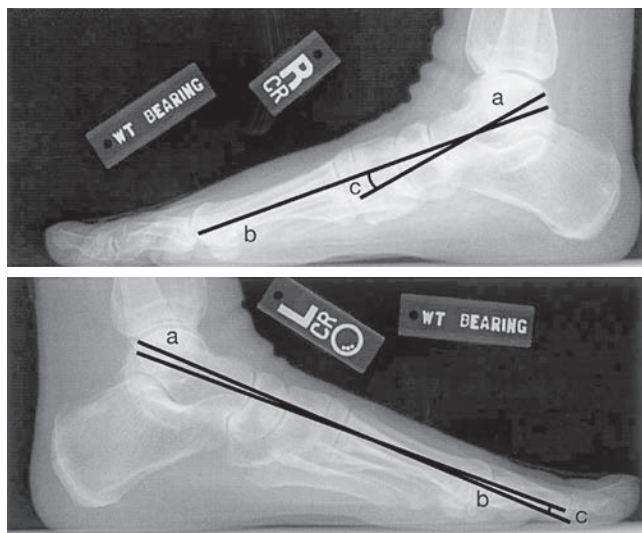


Fig. 10.8 Lateral weight-bearing views demonstrating the midfoot sag of the posterior tibialis incompetent right foot. I prefer this more simplistic measurement of the angle between the long axis of the talus (A) and the I metatarsal (B). The resultant angle (C) is greater in the involved foot. In this case in the normal left foot, these lines are virtually parallel. Also, notice that the subtalar joint is less clearly seen in the symptomatic right foot because of superimposition of the talus and calcaneus from a hindfoot valgus deformity.

- Are there arthritic changes with joint space narrowing, osteophytes, or loose bodies medially? Look at the ankle and all hindfoot and midfoot joints.
- Is there any evidence of a medial malleolar stress fracture or medial talar dome osteochondral lesion (Fig. 10.7C)?
- Is there significant arterial (anterior and posterior tibial) calcification?
- Is an accessory navicular or avulsion fracture visible on the medial navicular (see Fig. 10.7B)?
- Do the films of the foot reveal arthritic changes of the medial subtalar, talonavicular, naviculocuneiform, or medial tarsometatarsal joints (Fig. 10.7D)?
- Is there any evidence of a tarsal coalition, especially in the peri-adolescent athlete?
- On the lateral view, is the talo-first metatarsal angle negative (particularly if different than the contralateral film) (Fig. 10.8)?
- On the AP foot view, is there an increased talo-first metatarsal angle or increased “uncovering” of medial talar head at the talonavicular joint, thus indicating forefoot abduction (compare again with the contralateral foot, Fig. 10.9)?

Magnetic Resonance Imaging

Although not always necessary, if the history, physical examination, and x-rays fail to conclusively determine the diagnosis

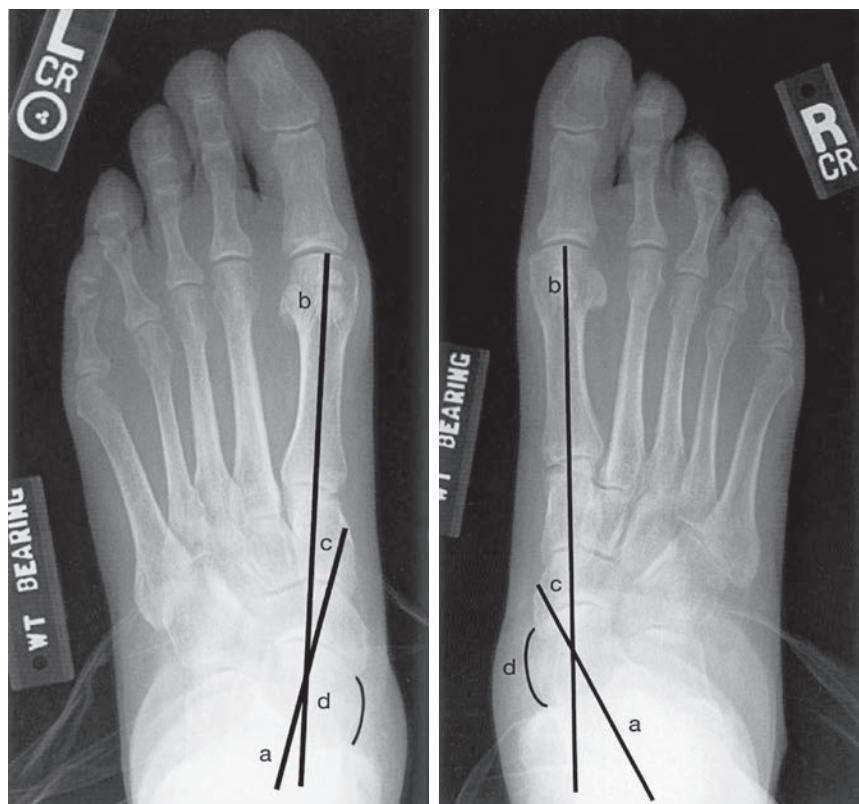


Fig. 10.9 Anteroposterior (AP) weight-bearing views demonstrating abduction deformity resulting from posterior tibialis tendon incompetency in the right foot. Again, I prefer this more simplistic measurement of the angle between the long axis of the talus (A) and the I metatarsal (B). The resultant angle (C) is great in the involved right foot. Also, notice that the medial talar head is more uncovered by the navicular in the involved foot (D).

of a posterior tibialis tendon injury, or to confirm the diagnostician's impression, then a magnetic resonance image (MRI) may be indicated.^{15,16} An MRI can also help determine the extent of acute injury or chronic tendinosis, arthritic changes, and guiding treatment, especially if surgery is planned, and may predict the postoperative clinical outcome.¹⁵ Finally, the MRI may help to determine other conditions that may mimic, be concomitant with, or even contribute to posterior tibialis tendon disease (Table 10.1).^{9,11,14,17,18} Perhaps of historical interest, others have proposed the diagnostic use of

tenography⁸ or ultrasound,¹⁹ but their sensitivity is significantly less than that of a high-quality MRI.

Generally, the MRI will reveal fibrous tendinopathic longitudinal hypertrophy or bulbous enlargement of the tendon, sometimes with cystic or longitudinal voids (Fig. 10.10A). Also, there is typically evidence of degenerative tendinosis and increased tenosynovial fluid within the sheath surrounding the tendon (Fig. 10.10B).^{19,20} These findings are most commonly seen in the inframalleolar region, but also can extend proximally into the posterior medial malleolar area.

DISEASE STAGING

Once the diagnosis is firmly established, the stage of posterior tibialis tendon disease, as popularized by Kenneth A. Johnson's seminal work, is important to determine the proper course of treatment. Johnson initially described stages I to III,⁶ and a stage IV was subsequently added^{12,21} to include degenerative arthritis of the ankle joint (Box 10.1). Stage IV deformities are exceedingly rare in the active athlete, and further discussion is beyond the scope of this chapter. Bluman and Myerson elaborated on the original classification by subclassifying the stage II deformity, as described below.²²

Stage I is essentially peritendinitis and/or tendon degeneration (tendinosis) with a normal tendon length and no deformity. Stage II is characterized by an incompetent or lengthened tendon with a flexible pes planovalgus deformity, meaning that the foot can be manually restored to a corrected neutral

TABLE 10.1 Differential Diagnosis	
Medial ankle arthritis	Medial subtalar joint or medial column arthritis
Medial ankle instability with deltoid ligament rupture/laxity	Symptomatic accessory navicular with synchondrosis disruption
Medial malleolar or talar stress fracture	Medial navicular bony avulsion or stress fracture
Medial talar dome osteochondritis dissecans	Acute injury or tendinosis of the flexor hallucis longus or flexor digitorum longus tendons
Tarsal tunnel syndrome	Peri-insertional anterior tibialis tendon rupture or tendinosis
Tarsal coalition, especially in periadolescent athletes	Medial ankle or hindfoot/midfoot crystalline or autoimmune arthritis

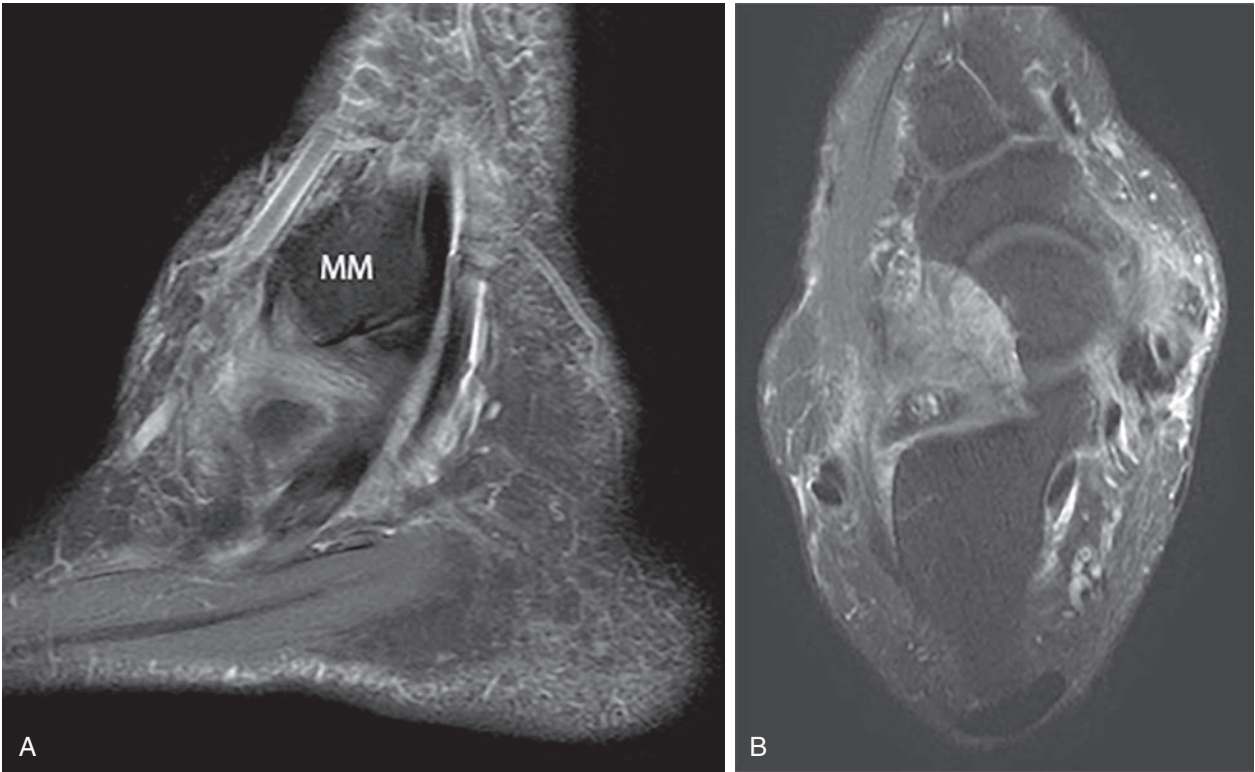


Fig. 10.10 Magnetic resonance imaging (MRI) findings of posterior tibialis tendon dysfunction. (A) Sagittal view at the level of the medial malleolus (MM) demonstrating longitudinal void within the tendon. (B) Transverse view at the level of the talus also demonstrating degenerative tearing and thickening of the PTT with intratendinous voids and increased fluid around the tendon.

BOX 10.1 Disease Stages

Stage I Peritendinitis and/or tendon degeneration (tendinosis)
 No deformity
 Stage II Tendon elongated/incompetent
 Mild flexible deformity
 Stage III Findings of Stage I and II
 Moderate-to-severe deformity that may be rigid with possible subfibular or sinus tarsi impingement
 Radiographic arthritic changes of triple joint complex and/or naviculocuneiform joints
 (Stage IV, which involves a valgus talar tilt and early ankle joint degeneration, also has been described but probably is not applicable to this discussion, given its extreme rarity in the active athlete.)

alignment on examination. Bluman et al.²² further subclassified stage II to include stage IIA (flexible hindfoot valgus), stage IIB (flexible hindfoot valgus with forefoot abduction), and stage IIC (flexible hindfoot valgus with fixed forefoot varus). Stage III encompasses the findings of the preceding stages but with a greater degree of deformity that also may be rigid. X-rays of the stage III foot may further reveal significant arthritic changes in any or all of the triple joint complex (subtalar, talonavicular, and/or calcaneocuboid joints) or naviculo-cuneiform joints, as well as clinical signs and symptoms of subfibular or sinus tarsi impingement.

TREATMENT**Conservative**

In general, conservative treatment is recommended initially, especially for the stage I and II presentation in the otherwise healthy athlete. In the older athlete who has significant comorbid conditions (e.g., diabetes, smoking, vasculopathy, obesity, etc.) that may contraindicate surgical treatment, conservative treatment may be the definitive treatment. In the young, competitive athlete, these comorbidities are uncommon. In general, the focus of treatment for the athlete should be to address: 1) the primary issue of a painful, inflamed and/or dysfunctional PTT; 2) alignment abnormalities that may contribute to attrition of ligaments and PTT; and 3) the deforming force of the triceps surae. If these can be addressed and mitigated, return to sport is feasible for most athletes with stage I and for many with stage II. Stage III deformities are compatible with few sports, but conservative treatment is an appropriate first step in treatment.

Reduction of inflammation. As with any inflammatory condition or injury, rigid immobilization can be therapeutic. Either casting or a rigid boot is recommended for 1–2 weeks to reduce acute inflammatory symptoms. The length of immobilization can vary based on the response to such treatment with reduction of swelling or tenderness. The cast that is well molded to support the arch or the incorporation of an arch support and/or medial wedge if a boot is chosen is further recommended. Prolonged immobilization can be problematic in athletes, since calf atrophy and gait abnormalities can develop. Weight bearing during the immobilization treatment period is

allowed as tolerated. The benefit of a boot, in a compliant athlete, is removal for passive range of motion and application of modalities to reduce swelling. Nonsteroidal antiinflammatory medications also are helpful, but chronic oral steroids should be avoided. Steroid injections also should be avoided, since these can lead to complete rupture^{8,23} or exacerbate a tendinosis.

Support of abnormal alignment. Upon symptom improvement following immobilization, custom-molded arch supports with medial wedging incorporated either in the orthotic and/or on a supportive shoe on the symptomatic side are advised for several months as the athlete integrates back to sport. This can help to support the joints of the medial longitudinal arch, preventing abnormal forces on a healing PTT. Optimal long-term management includes supportive and often-replaced, high-quality athletic shoes. The injured athlete also should avoid any repetitive impact-loading sports or conditioning for several weeks during treatment until symptoms have improved. Cross training (e.g., bicycling, swimming, open-chain weight training, antigravity treadmills, pool running, etc.) can help maintain muscular and cardiovascular fitness during treatment and recovery.

Address the deforming forces. In the athlete with a tight gastrocnemius/Achilles tendon, stretching is helpful,²⁴ especially to avoid reinjury, once he or she has been successfully treated conservatively and returns to the preinjury level of activity. Alvarez²⁵ described a protocol for conservative management of PTT dysfunction and pes planovalgus. In addition, if the athlete is overweight, weight loss is recommended. Other comorbid conditions also should be addressed when applicable, such as smoking cessation, good control of diabetes and autoimmune disease, and management of vasculopathy.

Surgical Treatment

Athletes who fail to improve with appropriate conservative management should undergo an informed discussion about surgical options (Table 10.2). In general, the focus of surgery for the athlete should be to address the same factors outlined for conservative treatment: 1) the dysfunctional or ruptured PTT; 2) progressive deformity; and 3) the deforming force of the triceps surae. It is critical to be mindful of, and address, all planes of deformity. As a hindfoot deformity progresses, the midfoot and forefoot must accommodate, which can alter anatomy and joint stability. Failure to recognize all deformities can lead to undercorrection and persistent symptoms or even failure of the corrective procedure.²⁶

Stage I—Tendon Length Normal

Intraoperative findings include tenosynovitis, often with granulation tissue, increased tenosynovial fluid, and an interstitial longitudinal rupture, usually between the medial malleolar tip and the navicular insertion of the tendon.^{4,10} Fusiform hypertrophy with tendinotic “crabmeat” tissue often is encountered, as well as possible cystic degeneration, especially in a more chronic presentation.

Surgical treatment involves removal of inflammation (tenosynovitis) and removal of degenerative sections of the PTT. The first can be accomplished arthroscopically and is associated with good outcomes.^{27,28} The latter typically requires an open

TABLE 10.2 Surgical management

Technique	Postoperative regimen	Return to sports postoperative
Stage I		
Tenosynovectomy	Cast: 3 weeks	3 months
Repair of interstitial rupture	Rigid brace: 3 weeks	
Possible medial shift calcaneal osteotomy for severe cases?	Supportive shoes with custom-molded arch supports: until 3 months postoperative	
Stage II		
Repair and advance/shorten tendon	Cast: 6 weeks	6 months
Imbricate talonavicular joint plantar medial capsule	Rigid brace: 6 weeks	
Flexor digitorum longus tendon transfer	Supportive shoes with custom-molded arch supports: until 6 months postoperative	
Medial shift calcaneal osteotomy with flexible deformity		
Possible Achilles tendon lengthening or gastrocnemius recession		
Stage III		
Repair/reconstruction of tendon may not be necessary?	Cast: 9–12 weeks	9–12 months
Medial shift calcaneal osteotomy and/or lateral column lengthening if deformity is flexible	Rigid brace: 6–9 weeks	
Arthrodesis if deformity rigid and/or arthritic changes present	Supportive shoes with custom-molded arch supports?	
Possible Achilles tendon lengthening or gastrocnemius recession		

procedure to address degenerative sections of the tendon. If disease is noted proximal to the medial malleolus, then it is important to preserve, if possible, an approximately 1-cm section of the sheath at the medial malleolar level to prevent subluxation of the tendon. (If this is not possible because of extensive proximal disease, then that portion of the tendon should be repaired after the tendon itself is addressed.) Tenosynovitis and granulation tissue are debrided with a small rongeur. The hypertrophied portion with tendinosis within the tendon then is debrided and debulked sharply via a longitudinal incision in the tendon itself. The incision then is repaired with absorbable, interrupted suture with inverted knots.

Postoperatively, splint immobilization is recommended for 2 weeks, followed by rigid bracing, stirrup bracing, or a short articulating ankle foot orthosis (AFO) for another 3 weeks. After immobilization, supportive footwear with a custom-made arch support is recommended for 3 months. In the athlete, repetitive impact-loading sports or conditioning endeavors are avoided until at least 3 months postoperatively. Good results have been noted with both the arthroscopic and open approaches.^{27,29}

Stage II—Tendon Elongated, Deformity Mild and Flexible

Similar but more severe pathologic findings as seen in stage I are encountered in stage II disease. There usually is a longer area of interstitial rupture with accompanying bulbous enlargement of the tendon that may even extend proximal to the medial malleolus. The tendon is found to be elongated, and thus incompetent, resulting in excessive pronation and abduction of the forefoot. (The function of the posterior tibialis is easily compromised with even a small increase in length because the normal

excursion in the healthy tendon rarely exceeds 1 to 2 cm.) There is a progressive pes planus deformity noted on physical examination and radiographs, but the deformity is passively correctable. The association of a significant gastrocnemius equinus with stage II disease entails that manual passive compression of the supple hindfoot deformity results in a rigid plantarflexed ankle. As described in for conservative management above, the surgical approach to stage II requires addressing 1) the painful, inflamed and/or degenerative PTT; 2) correction of bony alignment abnormalities that contribute to attrition of ligaments and PTT; and 3) the deforming force of the gastrocnemius or triceps surae.

The attenuated, degenerative posterior tibial tendon. The PTT is debrided and repaired as described for stage I. Shortening of the tendon is advised by removing degenerative tissue and advancing the healthy tendon to its plantar medial insertion on the navicular. It usually is necessary to detach the medial insertion and excise excess peri-insertional tendon before securing it to the decorticated plantar medial aspect of the navicular with nonabsorbable sutures incorporated in bone anchors¹⁰ or through drill holes (Fig. 10.11F–I).^{5,6,10,12,14,20,30,31} Other options include excising a transverse segment and imbricating the attenuated plantar medial capsule of the talonavicular joint torn (Fig. 10.11B, D, and G).^{12,30}

In more severe stage II cases, and especially if the tendon is ruptures or extensively degenerated, a flexor digitorum longus tendon transfer is recommended (Fig. 10.11A).^{4-6,8,10-12,14,20,30,31} The flexor digitorum longus (FDL) is in close proximity to the posterior tibialis tendon (Fig. 10.11C and E). Within the same surgical incision, the flexor digitorum longus is tenotomized sharply as distally as possible and is secured to the navicular or

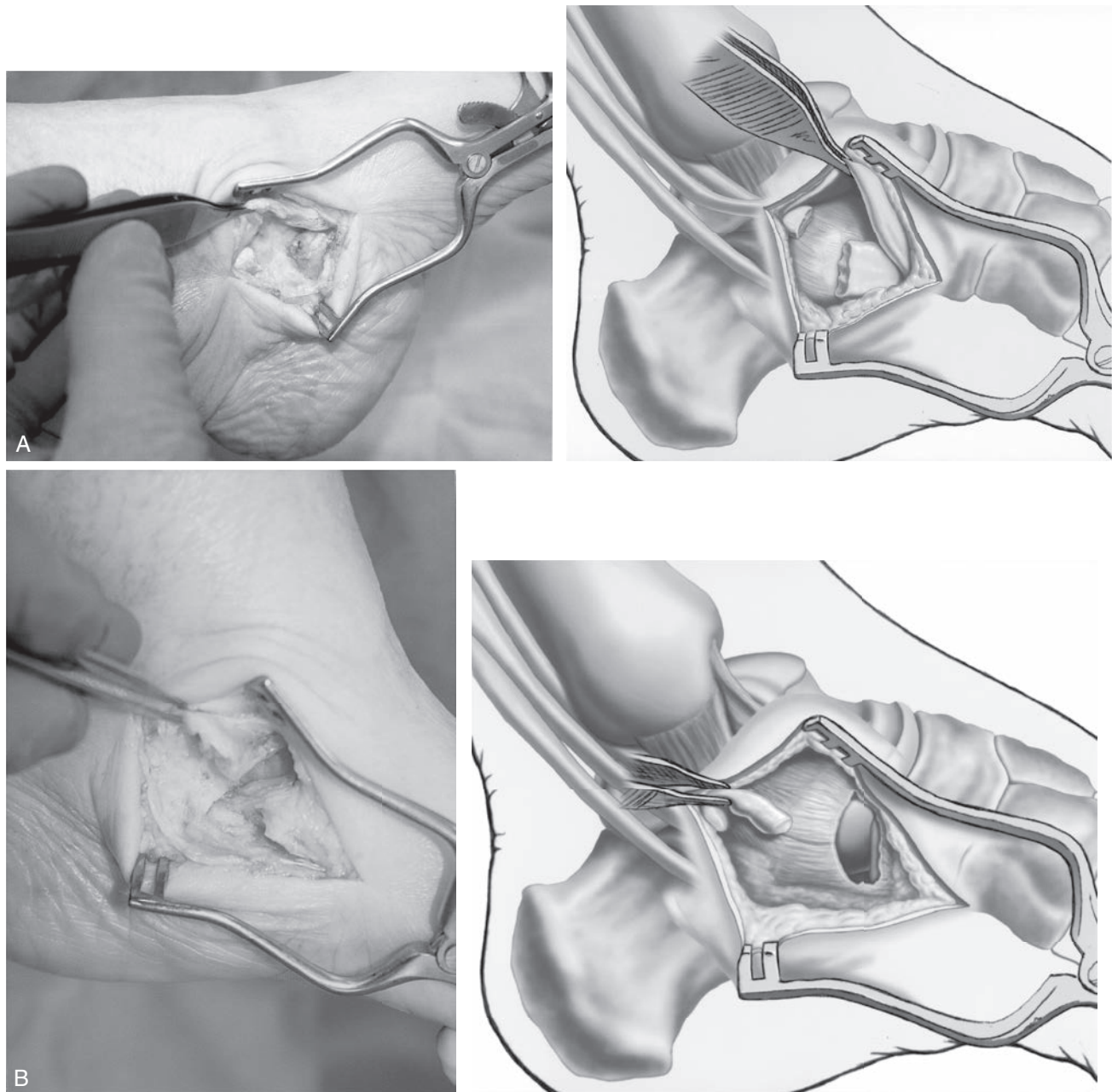


Fig. 10.11 Intraoperative photographs and corresponding schematic drawings demonstrating my preferred method of reconstruction of a complete rupture of the posterior tibialis tendon. This patient had a flexible deformity without degenerative triple joint arthritis. A medial shift calcaneal osteotomy was added to the medial soft tissue reconstruction. (A) Complete rupture of the posterior tibialis tendon. The two ends could not be approximated because of proximal migration of the proximal end. (B) Subsequent to debridement of the distal end of the posterior tibialis tendon, the plantar medial talonavicular joint capsule was incised and an elliptical segment removed to later imbricate it.

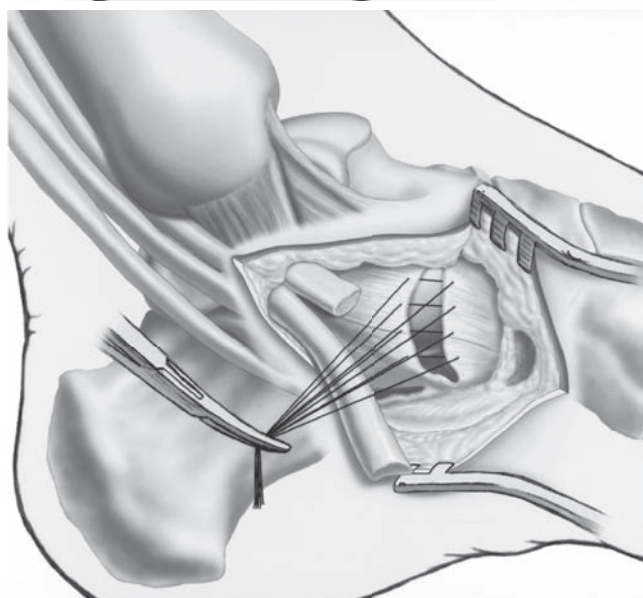
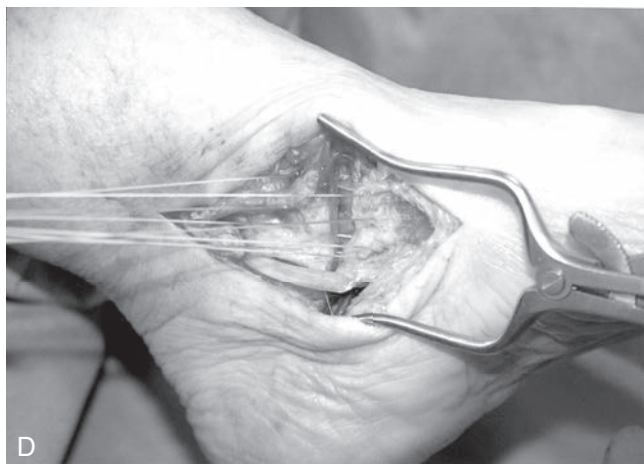
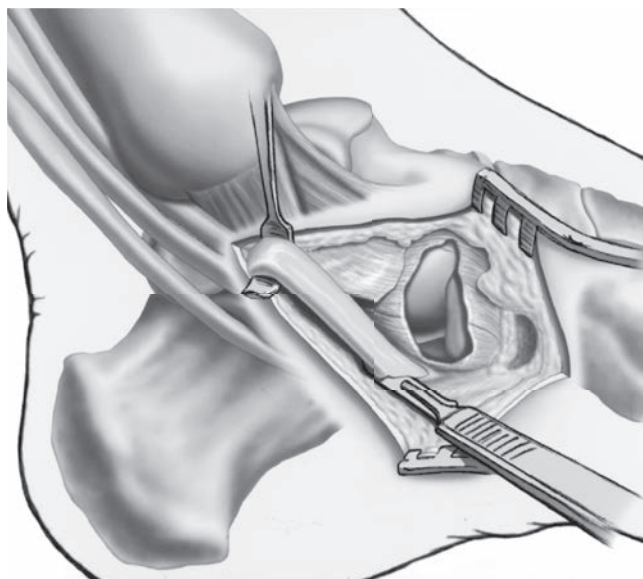
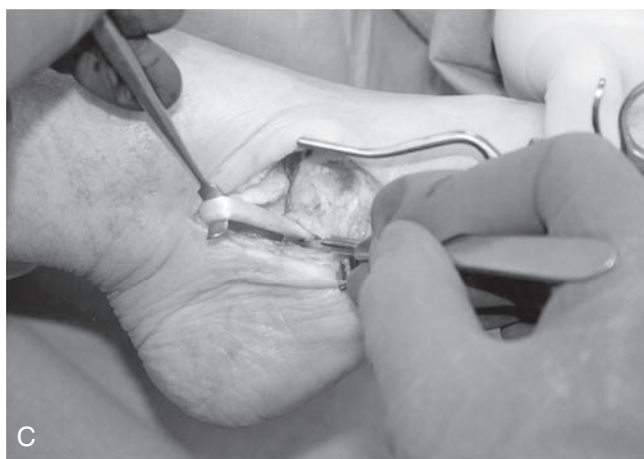


Fig. 10.11, cont'd (C) The flexor digitorum longus tendon is harvested as distally as possible. It is not necessary to tenodesse the distal end of the flexor digitorum longus of the flexor hallucis longus tendon to maintain adequate lesser toe flexor function. (D) Heavy absorbable stay sutures are placed and tagged in the plantar medial talonavicular joint capsule.

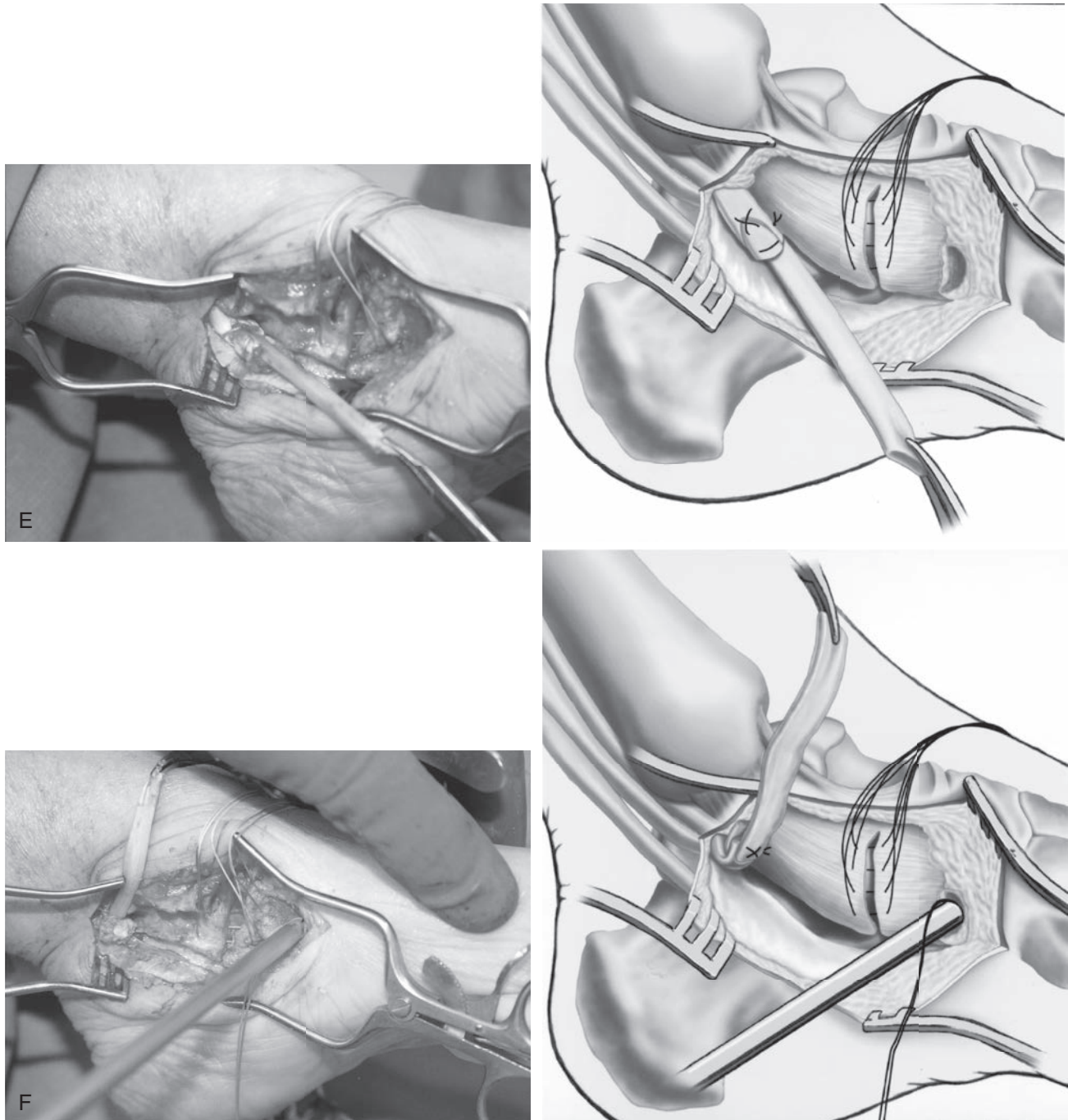


Fig. 10.11, cont'd (E) The flexor digitorum longus tendon is passed through and tenodesed with maximal tension to the proximal end of the posterior tibialis tendon. (F) A bone anchor is placed in the decorticated medial aspect of the navicular.

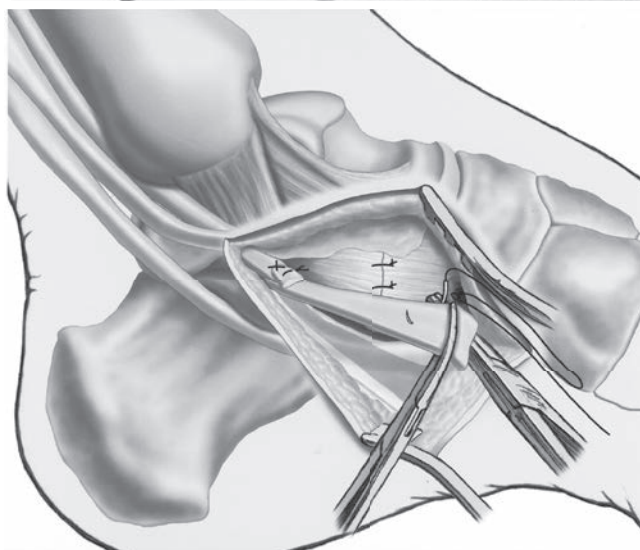
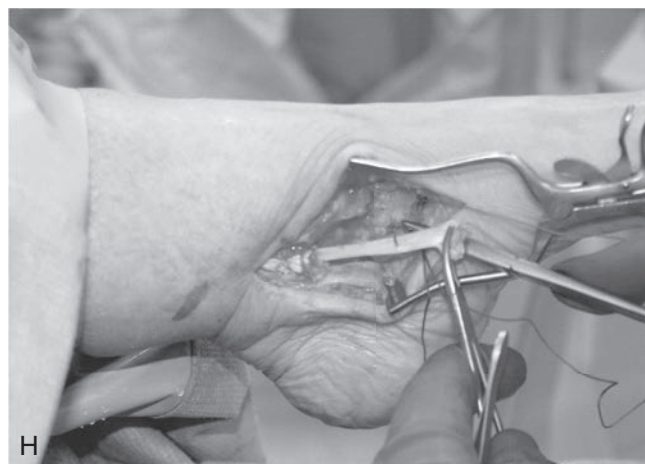
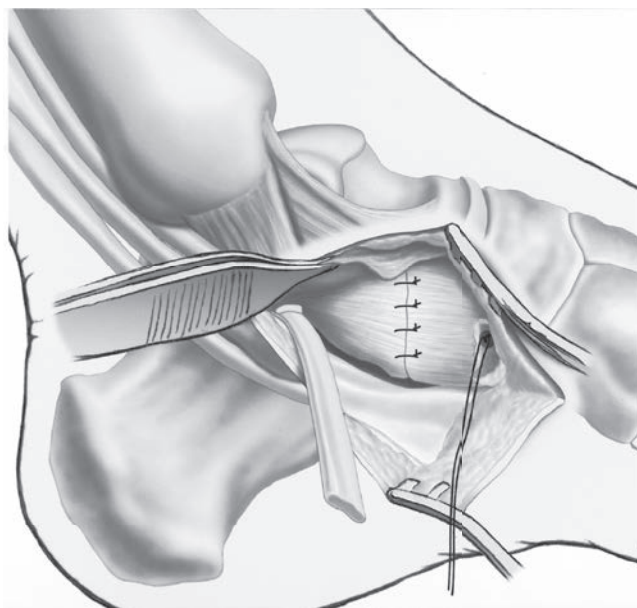


Fig. 10.11, cont'd (G) The plantar medial talonavicular joint capsular stay sutures are tied. (H) The flexor digitorum longus tendon is secured to the medial navicular under tension with the nonabsorbable sutures from the bone anchor.

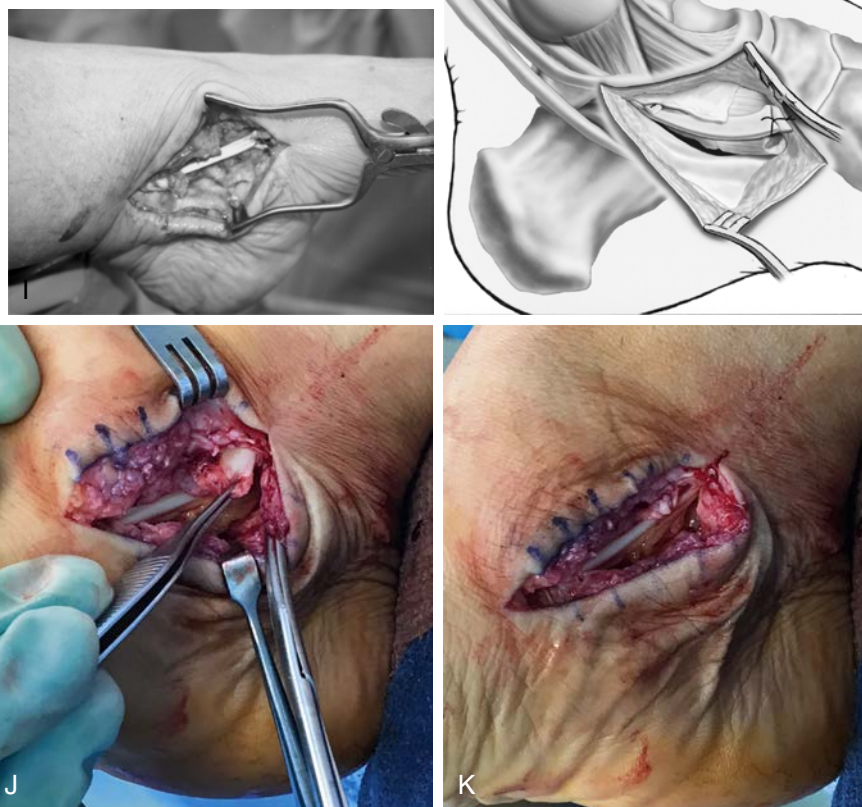


Fig. 10.11, cont'd (I) Final appearance of the reconstruction. Heavy absorbable sutures also are used to further secure the flexor digitorum longus tendon transfer to the fibrous tissue of posterior tibialis tendon sheath and the surrounding periosteum. (J) Posterior tibial tendon stump at proximal extent of incision, (K) tenodesis of PTT to transferred FDL with distal tenodesis of FDL to navicular.

as a side-to-side transfer to the repaired and advanced posterior tibialis tendon. Other tendons have been suggested for transfer if the flexor digitorum longus is not healthy or available. Although the flexor hallucis longus has a stronger muscle than the flexor digitorum longus, its transfer is not recommended because the dissection associated with its harvesting is technically challenging and risky, since Flexor Hallucis Longus (FHL) transfer requires crossing the neurovascular bundle. Also, the resultant weakness of great toe flexion may be a significant problem for a high-performance athlete.

Correction of bony alignment. Since the early 1990s, a standard component of the surgical correction of stage II deformity includes osteotomies to correct mechanics and alignment, and support the soft tissue repair. A medial displacement calcaneal slide osteotomy (MDCO) was popularized by Myerson,^{12,30} and described by others,^{11,20,24,32} for stage II disease. The effect of this osteotomy is to correct the hindfoot valgus deformity, translate the pull of the gastrocsoleus muscle via the Achilles tendon more medial to the axis of the subtalar joint, thus enhancing varus force on the hindfoot, and partially reestablish the medial longitudinal arch. The long-term benefit includes a reduction in load stress on the reconstructed posterior tibialis tendon or FDL transfer.

The MDCO is a straight cut from the lateral hindfoot at an angle of approximately 45 degrees to the plantar surface of the heel roughly equidistant between the posterior facet of

the subtalar joint and the posterior plantar edge of the calcaneal body, thereby avoiding the insertion of the Achilles tendon and the plantar fascia origin.^{12,20,30,31} An oblique, slightly curved incision is made, and great care is taken to protect the sural nerve in the lateral approach to the calcaneal wall (Fig. 10.12A). The periosteum is incised and preserved for later closure. A power saw can be used until the surgeon approaches the medial calcaneal wall, but an osteotome is recommended to complete the osteotomy medially to prevent injury to the neurovascular bundle (Fig. 10.12B–C). A medial shift of approximately 8–10 mm is recommended depending on the extent of deformity.^{12,20,30,31} Provisional fixation is achieved with two percutaneous pins in the sinus tarsi area until definitive internal fixation is placed (Fig. 10.12D). A partially threaded cannulated cancellous screw via a separate plantar posterior heel incision can be used for internal fixation. The proper placement of this screw should be guided fluoroscopically to avoid penetration of the subtalar joint and medial or lateral calcaneal wall. Countersinking the screw head is advised to prevent symptomatic hardware.

Several fixation methods are available. Dynamic compression nitinol step staples or contoured plates can be used for definitive internal fixation (Fig. 10.13A–C). This eliminates the need for a second incision associated with screw placement, is technically less challenging, and theoretically reduces the likelihood

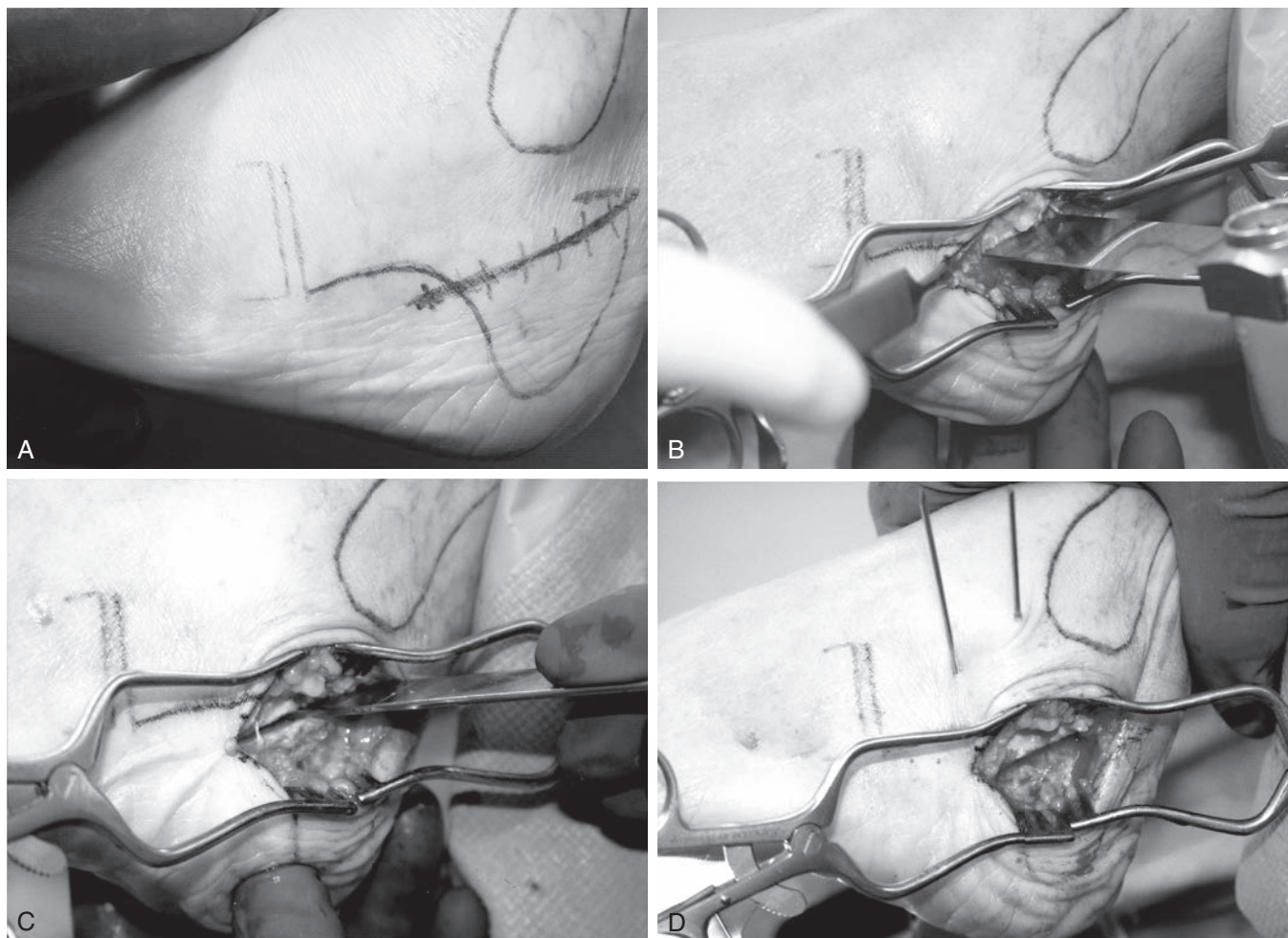


Fig. 10.12 Medial shift calcaneal osteotomy. (A) Recommended location of lateral incision. (B) Power saw used initially for osteotomy. Multiple retractors are recommended to protect the surrounding soft tissues. (C) Osteotomy completed through the medial calcaneal wall using an osteotome to minimize any potential damage to the neurovascular bundle. (D) Temporary percutaneous smooth pin internal fixation until definitive internal fixation is placed.

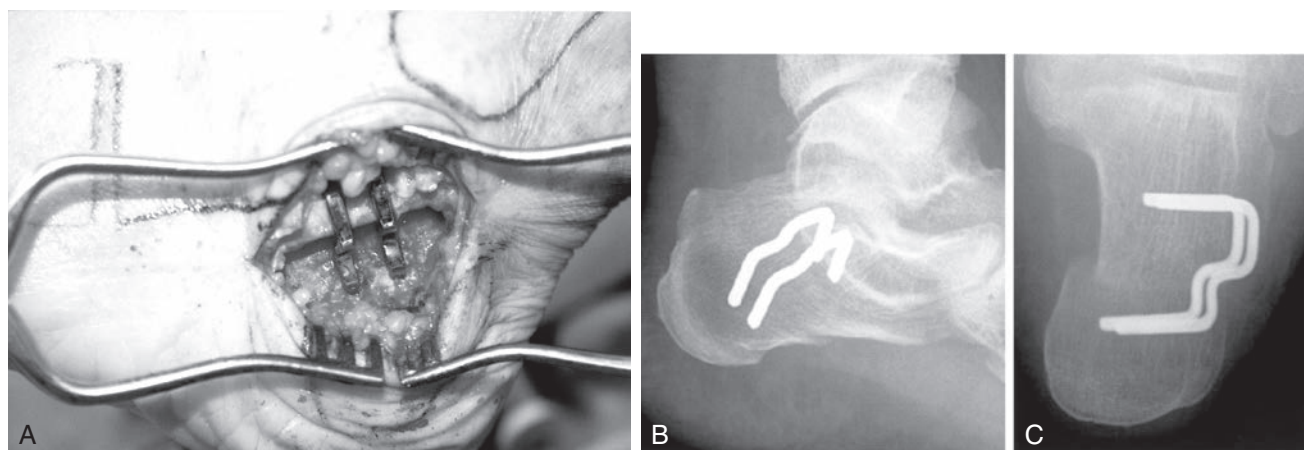


Fig. 10.13 Intraoperative photograph (A) and lateral (B) and axial (C) heel views demonstrating nitinol step staple internal fixation of the medial shift calcaneal osteotomy of approximately 10 mm for patients with primarily stage II and III posterior tibialis tendon dysfunction.

of symptomatic hardware. Alternatively, a single compression screw can be placed through a percutaneous incision on the heel achieving compression across the osteotomy site (Fig. 10.14A–C). The size and number of screws are a matter of some debate and is at the discretion of the surgeon.³³

In most cases of stage II deformity, there is a residual equinus contracture which prevents the foot from passive dorsiflexion to at least 10 degrees with the knee fully extended. In this event, a percutaneous Achilles tendon lengthening or gastrocnemius recession is indicated.²⁴ This procedure serves to allow sufficient

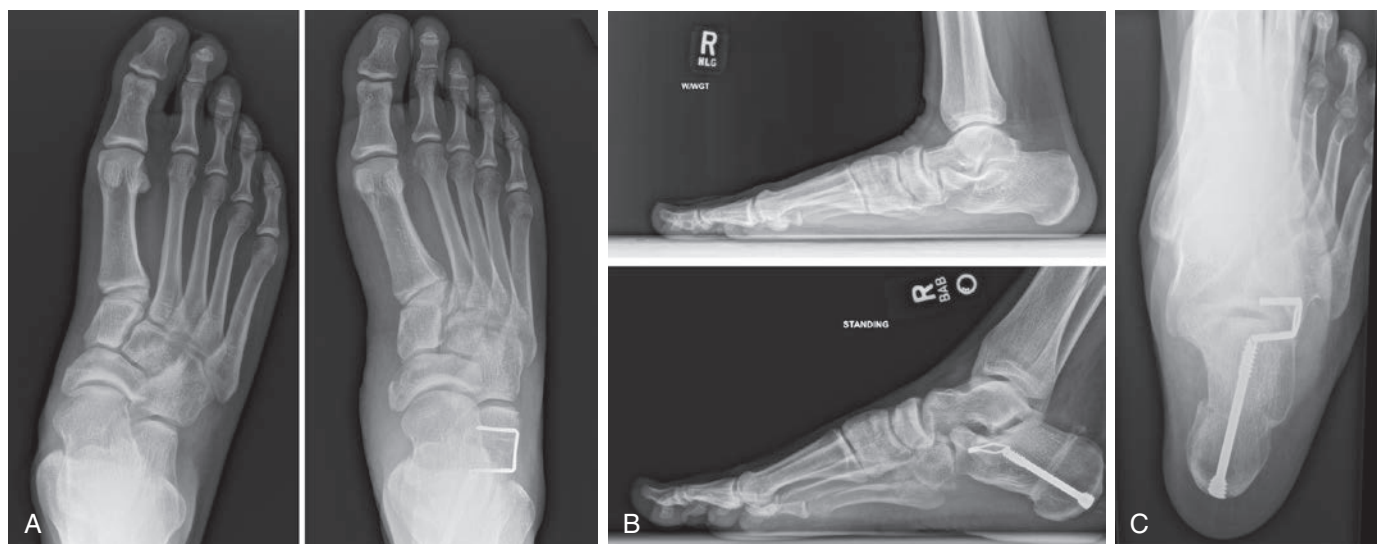


Fig. 10.14 Radiographs of double calcaneal osteotomy to correct flatfoot, including medializing calcaneal osteotomy with compression screw fixation and lateral column lengthening procedure. (A) Pre- and postoperative anteroposterior views, (B) pre- and postoperative lateral views, and (C) postoperative axial view.

ankle range-of-motion for normal gait, and to diminish the stress on the reconstruction and may help to prevent recurrent rupture and deformity postoperatively.

Postoperative care. Postoperatively, non-weight bearing and casting is advised for 6 weeks, initially with the foot in plantarflexion and inversion for the first 2 weeks. The foot is brought to a neutral position gradually by the end of the fourth week, when partial weight bearing is permitted. If a medial shift calcaneal osteotomy is performed, then serial axial and lateral x-rays of the heel are taken to monitor healing. Following casting, rigid bracing with an arch support is recommended for 6 weeks, with progression to full weight bearing as tolerated. The patient may remove the brace for bathing, sleeping, and active ankle range of motion exercises. Supportive footwear with a custom-made arch support is worn until 6 months postoperatively. Return to repetitive impact-loading sports or conditioning is ill advised before 6 months postoperatively. The majority of athletes are able to return to their previous sport and activity level following this procedure.³⁴

Stage III—Tendon Elongated, Flexible or Rigid Moderate to Severe Deformity

Stage III presentation is rare, especially in the younger athlete. Intraoperative pathologic findings often eclipse those of stage II, and the tendon is grossly incompetent even if still intact or may be completely ruptured with retraction of the proximal end of the tendon in the distal medial leg. Surgical repair or reconstruction of the tendon as described in stage II disease may not be necessary when a bony stabilization procedure (fusion or opening wedge osteotomy) is performed in stage III disease. A soft tissue reconstruction alone, even with a medial shift calcaneal osteotomy, is likely inadequate to prevent recurrent deformity and associated symptoms, especially in the heavier athlete.

If the deformity, although moderate to severe, is still flexible and there are no significant degenerative arthritic changes, then the surgeon may consider a medial shift calcaneal osteotomy

combined with a lateral column lengthening, with either a calcaneocuboid joint distraction arthrodesis or anterior calcaneal lateral column lengthening with bone graft can be effective to correct the deformity (Fig. 10.14).^{21,24} A calcaneocuboid distraction arthrodesis is suboptimal for the athlete, as this will limit hindfoot motion significantly and unduly stress the articulations of the synchronous function of the talonavicular and subtalar joints, possibly leading to early degenerative arthritis, which may be the result of diminution of circulating synovial fluid delivery of nutrition to the cartilage of these unfused, but now stiffer, joints. Theoretically, a lateral column lengthening with opening wedge osteotomy has the advantage of preserving more motion and thus preventing long-term arthritic disease.²⁴ An opening wedge plantarflexion osteotomy with bone graft of the dorsal medial cuneiform also has been described²¹ to further correct residual forefoot varus deformity with restoration of a more balanced, “tripod,” weight-bearing foot. In addition, step-cut osteotomies of the anterior process of the calcaneus may improve bone contact area and improve outcomes.³⁵ These procedures are technically challenging, and overcorrection can be a problem. Also, if autogenous iliac tricortical bone graft is chosen, the surgeon must consider the associated morbidity. In cases with stage III deformity and degenerative arthrodesis is indicated. This can be limiting for an elite field or court athlete. There is evidence of success with arthrodesis of the subtalar^{6,21,36} or talonavicular joint alone, calcaneocuboid and talonavicular joint arthrodesis (double arthrodesis),¹⁰ or triple arthrodesis.³² Perhaps it may not make much difference which type of arthrodesis is chosen, because, again, the fusion of even one of these joints severely limits the motion of the other two, thus maintaining the desired correction. However, long-term pain and eventual arthritis may develop because of the limited motion in the remaining unfused joints for the same reason as an isolated calcaneocuboid distraction arthrodesis as previously described. A triple arthrodesis with deformity correction would, of course, prevent this, but over time it may lead to usually

valgus ankle instability and arthritis.^{12,24} This is the result of long-term attenuation of the deltoid ligament usually resulting from undercorrection of hindfoot valgus that is rigid and therefore incapable of inversion/eversion torque conversion, thus translating those forces to the medial ankle. On rare occasions, especially if the hindfoot is overcorrected to varus, or in the pre-existing cavovarus foot that is undercorrected, just the opposite can occur, with lateral talar tilt, instability, and arthritis. It is generally wise to limit the arthrodesis to joints that absolutely need it due to arthrosis or significant deformity. It is not uncommon to fuse just the subtalar joint to correct hindfoot valgus, or just the talonavicular joint (if arthritic) to correct abduction. Fusions can be augmented with osteotomies to correct a deformity. As is the case with stage II, a percutaneous Achilles tendon lengthening or gastrocnemius resection²⁴ is further indicated if the foot cannot be passively dorsiflexed beyond 10 degrees with the knee fully extended.

Postoperatively, cast immobilization generally is in the 6- to 12-week range, depending on the progress of healing of the osteotomy or arthrodesis. Some limited weight bearing usually is allowed at 6 weeks postoperatively, depending on the level of healing on serial x-rays. Rigid bracing after casting is advised for 6 to 12 weeks. Supportive shoes with custom-molded arch supports may not be necessary, especially if a fusion is performed. Return to athletic activity is allowed 9 to 12 months postoperatively, pending complete bony and clinical healing.

PERILS AND PITFALLS

Underdiagnosis or misdiagnosis of posterior tibialis tendon injury in the athlete can be avoided with a conscientious history, physical examination, and weight-bearing x-rays. The differential diagnostic possibilities (see Table 10.1) also should be kept in mind when encountering the athlete with a suspected posterior tibialis tendon injury to prevent the perils and pitfalls of misdiagnosis and to aid selection of the proper treatment. If the presenting patient's diagnosis then is still in question, then an MRI is recommended. However, even the most astute diagnostician can fail to determine the proper diagnosis. MRI can also help the clinician identify other contributing or masking injuries, such as spring ligament tear, accessory navicular, stress fracture, etc. It is important to remove all degenerative PTT tissue, but tenodesis of healthy PTT to FDL proximally can contribute additional muscle to the tendon repair (Figs. 10 and 11J–K).

SUMMARY

Injury to the tibialis posterior tendon is not uncommon in athletes. Acute ruptures are rare, but tendinopathy can occur, impacting athlete performance, and creating risk of progressive deformity. Prompt diagnosis and identification of injury and classification of foot alignment is critical to managing this problem in athletes. Most cases will respond to conservative treatment, particularly in the patient without deformity (stage I). Surgical treatment is advised if conservative treatment fails in any stage, but especially if there is significant deformity (stages II and III). Prompt treatment, whether conservative or surgical, may prevent

the development of fixed deformity or degenerative arthritis and the potential need for an arthrodesis, which is highly problematic for repetitive, impact-loading conditioning and sports.

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Functional Nerve Disorders and Plantar Heel Pain

David A. Porter

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INTRODUCTION

Tarsal tunnel syndrome (TTS) is classified as a focal compressive neuropathy of the posterior tibial nerve (PTN) or one of its associated branches individually or collectively within the tarsal tunnel.^{1,2} The tarsal tunnel is a fibro-osseous space that is defined by the medial malleolus (superiorly), tibia (anterior border), posterior process of the talus (posterior border), calcaneus (lateral border), abductor hallucis (inferior border), and flexor retinaculum (lacinate ligament), which lies over the tibial nerve to create an enclosed space.² Within the tarsal tunnel lies the long flexor tendons and posterior tibial artery and vein along with the tibial nerve and its branches.³ Because of the inelasticity of the tunnel, any enlargement of the structures inside the tunnel can increase pressure leading to nerve compression.⁴ In general, nerve injury in this area is secondary to another pathology.⁴ The intrinsic and extrinsic etiologies of TTS primarily stem from posttraumatic, biomechanical, inflammatory, and morphological conditions; however, it can also be classified as an idiopathic and iatrogenic syndrome.¹ Characteristic clinical manifestations of TTS include paresthesia, dysesthesia, and hyperesthesia radiating from the retro-malleolar region to either the sole, heel, digits of the forefoot, or any combination of these areas.^{1,5,6} In some cases, pain has been shown to radiate proximally up the calf.⁴ The patient may report foot weakness as well.⁴ In addition to the symptoms described above, swelling over the tibial nerve may be palpable.⁴ Symptoms are typically made worse with increased activity such as standing, walking, or running.² The varying location and degree of symptoms depends on the location and the degree of compression of the tibial nerve or nerve branches.⁴

While rare in the general population, TTS has become increasingly more common in athletes. Neurologic conditions account for 10% to 15% of all exercise-induced leg pain among running athletes.⁷ Although the exact incidence of TTS in athletes is unknown, a study conducted by Kinoshita et al. identified surgically treated TTS patients, and found that between 1986

and 2002, 2.7 patients were treated annually with TTS. While this number is small, the percentage of sports-related cases among them is relatively high (39.1%).⁶ As mentioned above, TTS typically due to a secondary pathology. The increased frequency in athletes could be related to direct contusion, ill-fitting shoe wear, space occupying pathology (e.g., venous stasis, varicosities, accessory muscles, and ganglion cysts), or lower limb malalignment.⁸ Specific examples include tenosynovitis, accessory muscle (accessory soleus, accessory flexor tendons), fracture, or, more rarely, ligament fibrosis from chronic medial/deltoid ankle sprains.⁴ Repetitive trauma in the setting of predisposing malalignments is commonly seen in athletes.⁷ Researchers believe that injury to the PTN, because of its location and vulnerability at the tarsal tunnel, is due to the repetitive nature of running accompanied with abnormal or excessive pronation.⁹ Valgus deformity or flat feet can also exacerbate the problem.^{4,6,10,11} Additionally, sports that require repetitive hyper dorsiflexion may predispose athletes to TTS because dorsiflexion increases the overall pressure within the tarsal tunnel.⁸

Athletes with TTS will experience symptoms similar to non-athletes. Individuals with TTS may report medial ankle pain with cramping, burning, and tingling that radiates into the plantar arch of the foot and is exacerbated by activity, such as running, jumping, and prolonged standing.⁸ The pain can be sharp, shooting, or dull.⁴ Swelling, as mentioned previously, may also be present. Individuals may get relief from symptoms with rest, elevation, and removal of tight shoe wear.⁸

Medical causes of leg/ankle-pain have many similar presenting features, which makes TTS frequently misdiagnosed or underdiagnosed. Underdiagnoses is one possible reason why the incidence of tarsal tunnel in the general population and athletes seems low. Clinical presentations may appear similar, but in fact have a different pathophysiology relating to separate vascular abnormalities, compartment syndrome or neurological dysfunction.¹² For example, Sanger et al. reviewed the case of a 19-year-old female collegiate soccer player complaining of

unilateral foot pain that later transformed into bilateral foot pain with numbness, tingling, and cramping. This led to the diagnosis of TTS; however, the initial diagnosis was plantar fasciitis because of the location of pain and the description of symptoms. The misdiagnosis subsequently delayed her return to sport.¹³ Early diagnosis is key because a delay in diagnosis can lead to worsening of symptoms, inferior athletic performance, missed competition, and delayed return to sport.⁸ Clinicians should consider several general principles to facilitate the diagnosis and management of neurologic symptom: (1) maintain high level of index of suspicion for neurologic syndromes; (2) recognize common presentations of neuropathic pain; (3) perform a meticulous physical examination with detailed palpation of individual anatomic structures; (4) consider a broad differential diagnosis (neurologic and non-neurologic); (5) use diagnostic testing appropriately—electromyography/nerve conduction velocity test (EMG/NCV) and magnetic resonance imaging (MRI); and (6) make rational clinical decisions, including referral for second opinion when indicated.⁷

Tarsal tunnel syndrome diagnosis greatly relies on an in-depth history and physical examination because of the many possible causes of leg/ankle-pain. The aim of the history in a patient with possible neurologic leg/ankle pain is to gather occupational and sporting history, to determine the location and timing of the pain, its mechanism of onset, degree of irritability, aggravating and relieving factors, presence of other joint pain or swelling, previous local trauma, and presence of neurological signs or symptoms.¹² The physical exam combines the use of many investigations to properly diagnosis TTS. The Hoffmann-Tinel sign is the most commonly used clinical test of TTS.¹ A positive Tinel sign or nerve percussion/compression test will cause paresthesia either locally or radiating along the course of the nerve when the suspected site of compression is percussed.¹ It is suggested that greater than 50% of patients with compression neuropathy of the tarsal tunnel will demonstrate a positive Tinel's sign of the PTN.¹ Patients may also exhibit loss of two-point discrimination⁴ or occasionally, an abnormal Semmes-Weinstein test for pressure. Dorsiflexion-eversion test has been shown to increase strain on the tibial nerve and illicit symptoms also.⁴ Kinoshita et al. report that the dorsiflexion-eversion test reproduces or aggravates symptoms in 36 out of 44 feet in their study, with no replication evident in the control group.⁶ However, it is this author's opinion that local pain with palpation specific to the PTN nerve and the medial plantar nerve (MPN) is still the hallmark of TTS and its diagnosis.

Radiological imaging modalities such as isotope bone scanning, ultrasound, and magnetic resonance imaging (MRI) relate to the detection of bone and soft tissue lesions rather than neurological disease for the etiology of TTS.¹² MRI, the gold standard in identifying suspected compression of the tarsal tunnel caused by obstructive masses, can be used to confirm space-occupying pathology.^{1,14} MRI has been shown to be effective in 88% of patients with a firm clinical diagnosis of TTS.⁷ Not only does it confirm a suspected lesion, but it defines the depth, extent, and margins of the lesion for accurate characterization.¹ Ultrasounds can also effectively visualize structures within the tarsal tunnel and accurately identify compressive lesions and/or



Fig. 11.1 Axial T1 MRI scan of left ankle showing accessory muscle tarsal tunnel. Arrow points to accessory flexor muscle in tarsal tunnel against lateral planter nerve of posterior tibial nerve. Can be source of plantar lateral foot pain with tarsal tunnel variant involving only the lateral plantar nerve.

focal changes in the tarsal tunnel cross-section area.⁷ Diagnostic evaluation should also include weight-bearing foot and ankle radiographs to evaluate for deformity, talocalcaneal coalition, fracture malunions, and osteophytes.^{6,8,10,11}

We have noted an increasing occurrence of an accessory muscle that acts to compress the PTN in the tarsal tunnel in the athlete. This can present as either an accessory soleus, which is outside the tarsal tunnel but still causes pressure on the PTN, or an accessory flexor tendon within the tarsal tunnel. The accessory flexor tendon is unnamed but is thought to be an accessory flexor hallucis tendon (Fig. 11.1).

In some circumstances, the accessory flexor tendon in the tarsal tunnel will only affect the lateral plantar nerve. In this isolated lateral plantar nerve compression, the athlete complains only of pain in the lateral plantar foot (see case study). This presentation can be more confounding, and we have seen athletes misdiagnosed as fifth MT stress fractures, peroneal tendon injury, and cuboid stress fracture. Failure to improve with nonweight bearing (NWB) or immobilization is often a clue to nerve pain in the athlete.

In addition, electrodiagnostic investigations, which include nerve conduction studies and EMG, can assist in the diagnosis of TTS.^{1,6} While it has been concluded that sensory nerve conduction is more sensitive, yet less specific in identifying nerve injury than motor nerve conduction, it is recommended that both motor and sensory nerve conduction studies are evaluated.⁹ Unfortunately, these studies often yield an unacceptable level of false-negative results with inexperienced electromyographers, and should be utilized as an adjunctive assessment to confirm physical examination findings.¹ Because of the poor sensitivity and specificity for TTS, these investigations may be most useful

in ruling out proximal nerve pathology.⁸ We do believe, however, that with experience, detection of TTS with EMG/NCV can be more sensitive and specific. We therefore recommend developing a relationship with an interested lower extremity electromyographer to enhance their capabilities diagnosing the condition with the technique.

Researchers have recently recommended that a firm diagnosis of TTS be made only when the following triad exists: (1) foot pain and paresthesia, (2) positive nerve percussion sign/Tinel sign, and (3) positive electrodiagnostic studies. If only two exist, then the term *probable TTS* is recommended, and if only one exists, the diagnosis should be reconsidered.⁷

Nonoperative, conservative treatments are the preferred initial treatments even though they are often unsuccessful.⁸ Treatment includes activity modification, immobilization, oral or topical nonsteroid antiinflammatory drugs (NSAIDs), neuromodulator medications (e.g., Gabapentin, Pregabalin), physical therapy, biomechanical management, and steroid injections.^{2,7,12} Physical therapy includes strengthening the foot intrinsic and medial arch supporting muscles, Achilles stretching in subtalar neutral, lower limb kinetic chain rehabilitation, and proprioception-enriched rehabilitation in cases of ankle or subtalar joint instability.⁷ Special attention should be paid to Achilles tendon flexibility and foot intrinsic and ankle support muscle strength and coordination.⁸ Biomechanical management varies with clinical presentation and can be assisted with the use of motion control shoe, medial heel wedge, medial sole wedge/medial buttress, ankle stirrup brace, heel lift, or fixed ankle walking brace.⁷ In severe cases, medial wedges and arch supports may exacerbate symptoms, and more rigid immobilization via molded hindfoot orthosis, ankle-foot orthosis, or walking boot/cast may be needed.⁷ Tarsal tunnel injections may assist with treatment by reducing swelling in the tarsal tunnel providing relief of symptomatic nerve compression.¹² However, for those infrequently injecting the tarsal tunnel or if there is a poor pulse to palpate, ultrasound guidance should be considered when doing steroid injections to minimize risk of neurovascular injury.⁸

In the result of failed conservative treatments, surgery should be considered. Surgical procedures involve decompressing the tibial nerve or involved branches, while also repairing the pathology responsible for increased tunnel pressure.⁴ This may involve dissection of the flexor retinaculum, neurolysis of the affected nerve, tenosynovectomy, excision of bony fragments, stretching of the neurovascular bundle around foot deformities, or removal of space-occupying masses.⁴ While pain relief can be immediate with surgery, neuropathic symptoms generally improve 6 weeks postoperatively, but maximal recovery time may take 6 months or more.⁷ Kinoshita and colleagues reported a complete return to sports in 12 of 18 athletes post-surgery.⁴ Many studies have revealed that surgical release may improve or resolve the overall symptoms of TTS in 85%–90% of cases.¹ Alternatively, several studies have also reported little to no improvement of symptoms from surgical intervention.¹ Researchers believe that this could be due to continual clinical dichotomy between objective and subjective surgical outcome parameters, making definitive results highly variable.¹ While

failure is described to be less than 5%, initial releases often fail because of failure to control hemostasis, which leads to scarring and neuritis or nerve damage.² Therefore, we advise meticulous hemostasis after tourniquet release before definitive skin closure. Failures of surgical intervention often result from incorrect initial diagnosis, incomplete release of the tarsal tunnel (releasing the PTN, without releasing the MPN in the abductor canal), adhesive neuritis following initial decompression measures, intraneural damage associated with direct neural trauma or systemic disease, presence of space-occupying lesion, or double crush syndrome.^{1, 15}

CASE STUDY 11.1

The athlete was a 21-year-old white female senior basketball player who plays the 4-5 position on a Division I program majoring in Sports Management, who was originally from Serbia. She was evaluated for right lateral foot pain. She presented with a chronic 3-year history of right foot pain that was intermittent, achy, throbbing, sharp, and burning in nature. She states that she does not remember a specific injury but has a history of “recurrent stress fractures/stress reactions in the right foot” that never improved with immobilization, rest, even NWB status. Because of her lateral foot pain we were concerned for a peroneal tendon or abductor digitii quinti minimi (ADQM) injury. An MRI was normal and particularly showed no stress fractures and normal peroneal tendons. We tried an injection in the ADQM with no relief. We then ordered an EMG/NCV test, and it showed changes in the lateral plantar mixed nerve only with decreased amplitudes (essentially absent) but near-normal medial plantar mixed nerve amplitudes. There were severe changes on the needle exam to the ADQM with severe motor unit drop-out and severe loss of recruitment consistent with lateral plantar nerve involvement. The needle exam to the MPN was essentially normal.

The exam was consistent with pain over the PTN in the tarsal tunnel. There was some sharp tenderness locally and radiation into the lateral plantar foot that reproduced her symptoms. The plain x-rays were normal.

We performed an injection into the right tarsal tunnel placing some local anesthetic with steroid around the PTN and the MPN. We were able to confirm we were near the nerve by some mild radiation into the distal nerve distribution with the injection. It took 10 days to 2 weeks to get relief (a little longer than typical), but the athlete reported near-complete relief with her lateral foot pain. Since we were still in the off-season, she elected to proceed with a complete *tarsal tunnel release*. We followed a typical postoperative protocol that included cold compression therapy intermittently; boot immobilization for 2–4 weeks; then gradual return to biking, Stairmaster, and elliptical; and then running with a lace-up and velcro ankle brace. A functional progression program followed after she could run comfortably for 30–40 minutes on a treadmill. She returned to practice at 3 months and led the team in scoring and rebounding her senior year. She returned overseas after the season and pursued professional basketball. She obtained complete relief of her preoperative pain.

AUTHOR'S APPROACH TARSAL TUNNEL SYNDROME

We have taken a conservative approach to evaluation and treatment of TTS in the athlete. Many athletes can have nerve pain that is not amenable to surgical release. We are careful and cautious regarding our indications for surgical release of the posterior tibial and MPN. We have seen the effects of operative treatment of nerve pain that is not entrapment, and often noted that the patient can be worse.

Therefore, we have taken the following approach. If other injuries and ailments have been ruled out—e.g., posterior ankle impingement, anteromedial ankle impingement, posterior tibial tendon problems, medial dome osteochondral lesion (OCL), etc., **and** the athlete has pain over the posterior tibial and/or the MPN suggesting TTS (radiation into the tarsal tunnel nerves with local palpation, reproduction of their symptoms with local palpation of the nerves, +/- burning pain)—we will obtain an EMG/NCV test with a skilled and experienced electromyographer (often many neurologist or physical medicine specialists are competent in the upper extremity but not experienced in tarsal tunnel evaluation). If the test is positive for “tarsal tunnel,” we then proceed with a tarsal tunnel injection in the office to see if these electromyography changes are indeed reversible and specific to the athlete’s pain. We also like to see if the injection in fact alleviates the athlete’s symptoms at least temporarily. We inject both the traditional tarsal tunnel area around the PTN **and** around the MPN in the medial midfoot. We ensure we are around the appropriate nerves by confirming with the athlete that they experience some radiating symptoms corresponding to the nerves being injected. Others may want to do the injection with the aid of an ultrasound (as mentioned above) to navigate the location of the needle injection. We mix 0.5 cc of 1% lidocaine without epinephrine, 0.5cc of 0.25% Marcaine without epinephrine, and 3–6 cc of betamethasone all in one syringe so we know if the patient gets good relief in the office, the steroid injection is in the proper location. We ask the athlete to report the percent of relief in the office and then have the athlete call and report their pain relief 7–10 days after the injection. Relief in the office indicates relief from the local anesthetic and confirms at least that the nerve is the source of pain. Relief 7–10 days later indicates the nerve changes are reversible and surgical release may be beneficial. We believe the relief with the steroid must be 75% relief of pain or greater and be for greater than 1–2 weeks to be a positive result.

We have noted 75%–90% chance of good results with a complete TTR if there is a positive EMG/NCV test for tarsal tunnel **and** there is greater than 75% pain relief with the steroid injection for greater than 2 weeks (Fig. 11.2).

Some of our most satisfied athletes are those that have been misdiagnosed and found to have TTS and have had good relief with surgical decompression. We also still caution: some of our most unhappy athletes can be those who have had poor results from nerve releases and likely did not have entrapment, or had some form of iatrogenic worsening of their nerve pain.

MEDIAL PLANTAR NERVE ENTRAPMENT “JOGGER’S FOOT”

The “Jogger’s foot” can be more common than treating physicians realize. Don Baxter, who has treated numerous elite runners, commented on this entity in [Chapter 1](#) of our second edition. He noted that the MPN gets entrapped at the knot of Henry and mentioned that it can be an isolated entrapment irrespective of any more proximal TTS. Abnormal range of motion may lead to a “squeezing effect by the hypertrophied abductor hallucis



Fig. 11.2 Well-healed tarsal tunnel release incision left ankle. Note extension of incision into medial midfoot for good release of medial nerve.

muscle.” In an isolated entrapment, a minimal incision releases the MPN; because it is relatively deep, care must be taken to avoid damage to other nearby structure. We also routinely release this area with our TTR unless there are no symptoms referring to the MPN.

CASE STUDY 11.2

A 16-year-old female elite middle distance runner from New Mexico was seen in consultation for chronic left foot pain. She complained of mainly sharp pain with running in the left midfoot just below the navicular. She had mild pronation deformity restricted to the left foot and normal midfoot and hindfoot alignment on the right. She had seen several other health care providers who had diagnosed her with left hindfoot collapse. She had reported a moderate left foot injury 3–4 years ago but had not sought out treatment for that injury. She had recovered but had this mild collapse in her left foot ever since. She had been told she would not run competitively again and would need involved hindfoot and midfoot reconstruction.

However, her symptoms seemed much more localized to the MPN just underneath the navicular. She reported some radiation into the left forefoot with palpation of the MPN. An EMG/NCV test was undertaken and was suggestive of distal TTS with primary involvement of the MPN. An in-office injection of the MPN and distal PTN gave 100% relief for several weeks. She returned for an attempt at distal TTR. At the time of surgery, she had prominent scarring of the adductor canal around the MPN and the distal PTN. The distal PTN and the MPN were released with the open procedure and complete release of the MPN was undertaken down to the bifurcation of the MPN into the common digital nerves.

She underwent a traditional TTR rehab protocol with a full-length soft orthotic to support but not overly pressure the MPN under the midfoot. She returned to win the state championship in her primary race. She received a Presidential scholarship to an Ivy League school and ran track for 2 years in college before deciding to discontinue her competitive career. She remained pain free at last contact.



Fig. 11.3 Lateral radiograph of a dorsal osteophyte on naviculum caused a deep peroneal neuralgia.

ANTERIOR TARSAL TUNNEL SYNDROME

Anterior tarsal tunnel is a compression neuropathy of the deep peroneal nerve under the extensor retinaculum, dorsal talonavicular joint, or more distally over the Lisfranc ligaments and underneath the extensor hallucis brevis muscle and tendon. Symptoms include anterior ankle or dorsal foot pain with radiation to the first webspace that worsens with plantar flexion.¹⁶

The hallmark is pain to palpation over the affected nerve. Treatment includes release of the inferior aspect of the extensor retinaculum and debridement of any dorsal talonavicular spurs (Fig. 11.3). The lateral branch of the deep peroneal nerve supplies motor innervation to the extensor digitorum brevis (EDB), and entrapment can cause sinus tarsi–like symptoms. We avoid transecting the nerve as a treatment, especially proximal to the lateral branch to the EDB. This will denervate the EDB and lead to its atrophy. In dancers, hypertrophy of the extensor hallucis brevis has been shown to cause bilateral anterior TTS.¹⁶

CONCLUSION

In conclusion, TTS is an entrapment of the tibial nerve within the tarsal tunnel due to increased pressure typically secondary to another pathology. Local or radiating numbness, tingling, and pain over the medial heel, arch, medial sole, or sole of the foot can be described by patients with TTS. A sensory deficiency may also be observed or reported. These symptoms have much overlap with other causes of leg pain, so a broad differential diagnosis is crucial. Tarsal tunnel syndrome is increasing in frequency among the athletic population. Because of this, it is important that sports medicine physicians have a high index of suspicion of lower extremity neuropathy when dealing with exercise-induced leg pain. A detailed patient history and meticulous physical exam is of great importance when diagnosing TTS. Together, the physical exam, radiographic imaging, and nerve conduction studies in conjunction with selected tarsal tunnel injections create a reliable method of diagnosis for TTS. A quick diagnosis will aid in appropriate treatment and a more rapid return to sport. Generally, an initial conservative treatment is recommended over surgical

intervention. If the symptoms persist and become debilitating, surgery should be considered. There is still much debate on the positive outcomes of surgery because of the objective and subjective parameters used. More research needs to be done in this area to determine the true effectiveness of surgical intervention. Also, further evaluation needs to be directed toward the incidence of accessory muscle involvement with TTS in the athlete. Jogger's foot is a focal MPN entrapment that is in the continuum of TTS.

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Arthritic, Metabolic, and Vascular Disorders

Gregory Rowdon, David Taylor

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INTRODUCTION

Foot and ankle problems are common complaints to the physician who cares for athletes. Most of these complaints can be attributed directly to the athlete's training and competition with their sport. However, athletes are not immune from disease. Most of these athletes will present to the sports medicine physician assuming their complaints are related to their participation, and many will try to explain their complaints as being secondary to some aspect of their training. Although the vast number of complaints evaluated by the sports medicine physician is directly attributable to a primary musculoskeletal source, the physician must maintain an appropriate differential diagnosis to include those disease states that also can affect the musculoskeletal system. The foot and ankle are common sites for these disease states to present as they mimic sports injuries.

Many of the individuals who present to a sports medicine clinic are not "highly competitive" athletes but are athletes nonetheless. These individuals are commonly referred to as "recreational athletes" and generally are older. They strive to maximize their abilities in their own chosen activity while attempting to reap the myriad of benefits of a healthy lifestyle. This group of athletes may have concurrent disease states that must be taken into account as they attempt to maintain their healthy, active lifestyle. Diseases such as diabetes, gout, thyroid conditions, osteoporosis, and so forth can present with musculoskeletal complaints. The purpose of this chapter is to review those disease states, which may mimic a primary

musculoskeletal problem in both the competitive and recreational athlete. Knowledge of these conditions is essential to the physician caring for athletes.

INFLAMMATORY/ RHEUMATOLOGIC

Still's Disease (Adult Onset)

Still's disease is a seronegative polyarthritis that usually affects young adults. It is characterized in its initial manifestation as a spiking fever and a red/salmon-colored rash, usually over the trunk and extremities. The rash is transient and appears at the time of the fever spikes. The inflammatory arthritis is a polyarthritis or oligoarthritis. It commonly affects the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints, as well as the wrists, knees, hips, and shoulders. Occasionally, the cervical spine, intertarsal joints, temporomandibular joints (TMJ), and the distal interphalangeal (DIP) joints are affected. It may lead to fusion of the carpal-metacarpal and the intercarpal joints. Laboratory evaluation commonly shows an elevated white blood cell count as well as an elevated erythrocyte sedimentation rate (ESR). Anemia of chronic disease is commonly present. Secondary nonmusculoskeletal findings include lymphadenopathy, hepatosplenomegaly, pericarditis, and carditis. The disease is treated with nonsteroidal antiinflammatory medications (mild disease). Often, oral corticosteroids are required to control moderate disease along with biologic/nonbiologic disease modifying antirheumatic drugs (DMARDs) for severe disease. Overall, Still's disease has a good prognosis.

Ankylosing Spondylitis

Ankylosing spondylitis is an insidious-onset seronegative inflammatory condition affecting young individuals, that is, generally younger than 40 years old. It has a uniform sex distribution, but the disease seems to be milder in females. Also, females have more peripheral involvement rather than spine involvement. Ankylosing spondylitis affects the sacroiliac (SI) joints, followed by the spine and peripheral joints. There usually is symmetric loss of spine movement. The peripheral joint involvement occurs in 20% to 30% of ankylosing spondylitis patients and has a predilection for the lower extremities. Achilles' tendinitis, plantar fasciitis, and costochondritis also are associated with the disease process. It is common to have fatigue, weight loss, low-grade fever, and, in more severe cases, uveitis, pulmonary fibrosis, and cardiac abnormalities. Laboratory findings include an elevated ESR. The natural history of ankylosing spondylitis is poorly defined, with some patients experiencing minimal disease and some patients experiencing severe disease. Treatment usually involves physical therapy and antiinflammatories. Refractory cases may respond to antitumor necrosis factor (TNF) agents.

Reiter's Syndrome

Reiter's syndrome involves the triad of arthritis, uveitis, and conjunctivitis. It commonly occurs following an episode of either genitourinary or gastrointestinal (GI) infection. It has associated features of inflammatory eye lesions, balanitis, oral ulcers, and keratodermatitis. Reiter's syndrome has a male-to-female occurrence of 5:1. The arthritis experienced in Reiter's syndrome is a reactive arthritis rather than an infectious arthritis. It usually occurs 2 to 6 weeks following the onset of an infectious episode. It is asymmetric and mainly affects knees and ankles. It is usually of acute onset. There may be diffuse swelling of fingers and toes, that is, sausage digits. There is commonly inflammatory change at both the Achilles' tendon insertion and the plantar fascial origin. There also may be associated low-back pain with involvement of the SI joints, making it difficult to distinguish it at times from ankylosing spondylitis. The conjunctivitis in Reiter's syndrome may be either unilateral or bilateral. It usually is mild and transient and is a noninfectious source like the arthritis. Common skin lesions in Reiter's syndrome are small, shallow, painless penile ulcers called balanitis circinata. Another associated skin lesion is keratoderma blenorrhagica, which represents hyperkeratotic skin lesions mainly involving the soles of the feet, but they also can be found on the palms and the scrotum. Radiographic findings may demonstrate erosions or periosteal changes, particularly at the Achilles' tendon insertion or plantar fascial origin. Also, an asymmetric sacroiliitis may be present that is in contrast to the symmetric involvement of ankylosing spondylitis. Reiter's syndrome also is seronegative but usually demonstrates an elevated ESR and white blood count. Treatment for Reiter's syndrome involves antiinflammatory medications and intra-articular steroid injections as well as physical therapy. Systemic oral steroids have been shown to be of minimal benefit except in refractory cases. Nonbiologic DMARDs may be necessary. Topical steroids are used for the skin lesions and for the conjunctivitis. The prognosis for Reiter's

syndrome is generally good, with typical duration of 3–5 months with either complete remission or little active disease after 6–12 months. However, 15%–20% of patients may develop a chronic persistent arthritis.

Psoriatic Arthritis

Psoriatic arthritis is the combination of psoriasis and inflammatory arthritis. To make a definitive diagnosis of psoriatic arthritis, skin or nail changes of psoriasis must be present at some point in the course of the disease. The arthritic changes can be present before skin changes develop. The joint pattern in psoriatic arthritis is variable but commonly includes a pauciarticular asymmetric arthritis involving the peripheral joints. It is common to have the spine involved in combination with peripheral joints as well as inflammation of tendon and insertion points of tendons, that is, enthesitis. Digits may become sausage like. There often are associated eye changes, including conjunctivitis, iritis, and episcleritis. Psoriatic arthritis has an equal sex distribution and usually has onset in the 30- to 40-year-old age group. Laboratory results are often normal, but some athletes will present with an elevated ESR and/or a normocytic normochromic anemia. Synovial fluid evaluation typically reveals a mild inflammatory process. Radiographs often reveal DIP erosive disease, sacroiliitis, and enthesopathy and/or periostitis. Treatment of psoriatic arthritis involves the use of antiinflammatory medications (mild disease), physical therapy, and intra-articular corticosteroids to treat the inflammatory arthritis. Oral glucocorticoids are generally avoided due to the increased risk of developing erythroderma or pustular psoriasis. The focus of treatment, however, involves treating the athlete's skin lesions. Oral methotrexate (a nonbiologic DMARD) is a common therapeutic choice because it treats both the skin lesions and the arthritis. Severe disease may require a biologic DMARD or TNF agent. Psoriatic arthritis was once considered a mild disease with a good prognosis. Now, however, it is considered a more severe disease, and early referral to rheumatology is recommended.

Enteropathic Arthritis

Enteropathic arthritis is arthritis associated with inflammatory GI conditions including ulcerative colitis and Crohn's disease, and infectious GI conditions, including *Shigella*, *Salmonella*, *Campylobacter*, *Yersinia*, and Whipple's disease. The arthritis, when associated with ulcerative colitis or Crohn's disease, usually is one of a peripheral arthritis with associated sacroiliitis and less often enthesopathies. It often is a transient, oligoarticular, migratory, nondestructive arthritis associated with the bowel disease activity. The knees and ankles are most often involved. Synovial fluid from the affected joints contains mild to severe inflammation. There are a variety of associated cutaneous lesions with the disease, and mucosal, serosal, and ocular lesions may occur. The arthritis with ulcerative colitis and Crohn's disease often resolves with medical (glucocorticoids or TNF agents) or surgical treatment of the intestinal disease.

The arthritis associated with enteropathic infection often comes on a few weeks following the bowel symptoms. The arthritis, in this case, is a reactive arthritis and, again, affects

mainly knees and ankles. There also may be axial joint involvement. Enthesopathies, although not common in association with ulcerative colitis and Crohn's disease, are common in association with infectious GI conditions and typically involve the plantar fascia and Achilles' tendon insertions. The arthritis is usually self-limited, resolving weeks to months after the bowel infection. Treatment is symptomatic, involving the use of anti-inflammatory medications, physical therapy, and intra-articular corticosteroid injections.

Rheumatoid Arthritis

Rheumatoid arthritis is a chronic, systemic inflammatory disease characterized by significant joint involvement. It affects multiple systems extensively, and thus a full detailed description of the disease is beyond the scope of this chapter. It involves symmetric upper extremity, knee, and foot destructive changes, sparing the DIP joints of the hands and feet. It results in progressive joint destruction and deformity. Again, there are multiple extra-articular features, including rheumatoid nodules, arteritis, neuropathies, scleritis, and pericarditis. Lymphadenopathy and splenomegaly are common. The incidence in females is two to three times greater than in males. It may occur at any age and increases in frequency with increasing age. Hand, wrist, knee, and foot are most commonly involved, but any diarthrodial joint can be affected. The elbows, shoulders, sternoclavicular (SC) joints, hips, ankles, and TMJ are less commonly involved. Spine involvement is limited to the upper cervical spine.

Feet and ankle changes are similar to those seen in the hands. Cocking up of the toes may occur secondary to subluxation of the metatarsal heads. This gives the digits a claw-like appearance. Fibular deviation of the first through fourth toes may occur. Bursal inflammation about the foot/ankle also occurs with the retrocalcaneal bursa being most common. Laboratory evaluation usually shows a normocytic, normochromic, or hyperchromic anemia. There often is an elevated ESR and positive rheumatoid factor. Joint fluid evaluation reveals mild inflammation. Treatment involves referral to a rheumatologist and the use of DMARDs in all patients diagnosed with rheumatoid arthritis. The use of anti-inflammatory medications, physical therapy, and intra-articular corticosteroid injections for symptomatic joints are adjuncts to treatment.

Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a chronic, multisystem inflammatory disease affecting bone, joints, tendons, skin, kidney, heart, lungs, GI tract, and central nervous system (CNS). Again, a full and detailed description of the disease process is beyond the scope of this text. SLE has a 9:1 female-to-male ratio. The arthralgias and arthritis are a common presenting complaint. The arthralgia/arthritis often is symmetric. Joint capsule, ligamentous, and tendon involvement can be prominent in the disease, and hand or foot deformities may develop. There often are marked laboratory abnormalities, including a normocytic, normochromic anemia, leukopenia, thrombocytopenia, elevated ESR, and positive antinuclear antibody (ANA) and double-stranded DNA. Because of the great heterogeneity

of the disease, treatment is highly individualized. In general, antiinflammatories, topical/oral corticosteroids, antimalarials, and immunosuppressive agents are used.

Gout

The pathogenesis of gouty arthropathy involves tissue deposition of uric acid crystals from a supersaturated extracellular fluid. Gout involves recurrent attacks of severe articular or periarticular inflammation. Late involvement of the disease involves crystal deposition of uric acid within articular, osseous, soft tissue, and cartilaginous structures. These tophi occur late (>10 years) in the disease. There may be renal impairment with or without uric acid urinary calculi. Hyperuricemia may be demonstrated in individuals without gout and uric acid levels may be within the normal range in individuals showing clinical gouty arthropathy. Gout is a disease of middle-aged men and postmenopausal women. It increases in frequency with age.

An acute, gouty, arthritic flare most commonly involves the great toe metatarsophalangeal (MTP) joint but also commonly involves the ankle. It usually involves a single joint with an acute onset, often during the evening hours. The joint often appears warm, red, and swollen and usually is exquisitely tender. The flare may subside spontaneously 3 to 10 days following onset without treatment. Individuals often are symptom free following an acute attack, but over time, if untreated, the attacks may increase in frequency, in the number of joints affected, and in duration of symptoms when flared. The flares may be triggered by trauma, alcohol, drugs, stress, or medical illness. Tophi when present occur most commonly in the synovial tissue, subchondral bone, olecranon bursa, patellar and Achilles' tendons, subcutaneous tissue on the extensor surface of the forearms, and overlying joints. Radiographic findings in gout usually are negative. Often they are obtained to rule out other joint processes, such as a septic joint, or to evaluate for the presence of chondrocalcinosis. More chronic cases can show periarticular erosions and frank degenerative changes, especially in the great toe MTP joint. The gold standard for diagnosis is monosodium uric crystals demonstrated in joint fluid. The white blood cell count from a symptomatic joint usually reveals moderate inflammation. Treatment in the acute setting may involve anti-inflammatory medications, oral corticosteroids, or colchicine. Treatment in the chronic setting may also involve the use of colchicine the most commonly involves the use of allopurinol or probenecid.

Pseudogout/Calcium Pyrophosphate Dihydrate Crystal Deposition (CPPD)

Pseudogout involves acute, gout-like, inflammatory attacks that occur secondary to calcium pyrophosphate dihydrate crystal deposition (CPPD) within joints. The incidence of clinically symptomatic pseudogout is one-half that of true gout. Calcium pyrophosphate dihydrate crystal deposition may occur as an incidental finding in a symptom-free joint with radiographic evaluation. The term "chondrocalcinosis" is used to describe this x-ray appearance. The male-to-female ratio of pseudogout is 1.4:1 and is in marked contrast to the distribution in gout. The pseudogout flare usually involves one or more joints lasting

for several days. It usually is abrupt in onset but self-limited. Findings may be as severe as in true gout, but typically the attacks of pseudogout are less painful. The knee is the most commonly affected joint, but all joints are susceptible, including the first MTP joint. The flare may be triggered by trauma, surgery, stress, or medical illness. Individuals usually are symptom free between flares. Treatment is with antiinflammatory medications and intra-articular steroid injections. Recurrent disease may be treated with prophylactic colchicine.

PEARL

Suspect inflammatory disease in a joint that has no history of trauma and that is swollen and warmer than expected for the history.

PEARL

Suspect inflammatory disease if there is a history of multiple joint involvement or other systemic complaints that is, skin, GI, constitutional, and so forth.

CASE STUDY 12.1 Gout

A 46-year-old, male runner awakens with a swollen, warm, red right ankle, which is exquisitely painful. He denies injury but did go for his usual 3-mile run 1 day ago. The rest of his history is noncontributory. On physical examination he demonstrates an effusion to the ankle with the joint erythemic and warm. The ankle is diffusely and significantly tender. The rest of the examination is noncontributory. X-rays are normal. Laboratory studies show a normal complete blood count (CBC), ESR, renal function, and uric acid. Joint aspiration demonstrates a mild to moderate inflammatory response and is positive for monosodium urate crystals. The patient was treated with indomethacin and demonstrated a complete response over the next few days.

CASE STUDY 12.2 Reiter's Syndrome

A 24-year-old, professional basketball player presents with a left ankle that is painful, swollen, red, and warm. He also notes several toes that are swollen and right heel pain. His past medical history and family history are noncontributory, except that he was treated for a *Chlamydia* infection 1 month ago. He is on no medications except for Visine for "irritated" eyes. Physical examination demonstrates an erythemic, warm left ankle with mild effusion. Several sausage digits are noted. The right plantar fascia origin is tender. Both conjunctiva are injected. The rest of the examination is noncontributory. X-rays are normal. Laboratory studies are negative, including an inflammatory workup, except that the ESR is elevated and the white blood cell count is at the upper limits of normal. The athlete was treated with nonsteroidal antiinflammatory drugs (NSAIDs) and physical therapy. The athlete returned to baseline and there were no recurrences.

OTHER

Lyme Disease

Lyme disease is a multisystem illness caused by the tick-borne spirochete *Borrelia burgdorferi*. The disease is characterized by a rash at the bite site (erythema chronicum migrans), constitutional symptoms, neurologic abnormalities, cardiac involvement, musculoskeletal complaints, and a reactive arthritis. Early

in the disease course, there often is migratory pain without specific inflammation to the joints. Tendon, bursal, and muscular inflammation is common. The reactive arthritis usually occurs in intermittent attacks. It can be monoarticular to oligoarticular and has a preference for large joints, especially the knees. It can last for months, with chronic flares over several years. The treatment for Lyme disease early in its course is tetracycline, amoxicillin, or cefuroxime. With disseminated or late course disease, intravenous penicillin usually is the treatment of choice.

Sarcoidosis

Sarcoidosis is a multisystem illness characterized by noncaseating epithelioid granulomas in affected tissues. It has a tendency to affect young adults of either sex. It most often begins as bilateral hilar lymphadenopathy, pulmonary infiltrates, and skin and eye lesions. However, there may be bone lesions, localized muscular granulomas, and acute inflammatory arthritis. The arthritis is the most common rheumatologic manifestation and can be the initial complaint. The arthritis most commonly affects the ankles and knees. The most severe attacks usually occur during active disease. These flares usually last for 2 to 3 weeks. Chronic arthritic changes are much less common. The prognosis in sarcoidosis is favorable. Treatment usually is antiinflammatory medication or a short course of oral corticosteroids.

METABOLIC DISEASE

Metabolic diseases are an uncommon cause of concern in the athletic foot and ankle. The most common metabolic disease that may present with foot and ankle issues is diabetes mellitus. The neuropathy and microvasculopathy in the extremities, especially the foot and ankle, can result in a wide range of sequelae. Metabolic bone disease is another common metabolic disease that uncommonly affects an athlete's foot and ankle. In cases of recurrent stress fractures, metabolic bone disease such as osteoporosis may be the underlying cause. Medications and/or supplements can cause metabolic bone disease or can cause other conditions that are risk factors for metabolic bone disease. Examples include steroid use (or abuse), which causes drug-induced osteopenia, or vitamin B12 deficiency, which can cause a neuropathy that may present with diabetes-like complications.

Diabetes Mellitus

Diabetes mellitus is a common disorder. Younger athletes are more likely to be type I, but many type II diabetics are involved with athletics, especially on a recreational or fitness level. The most important factor is achieving optimal control of the athlete's diabetes. Tighter control usually equates with fewer complications. In the setting of the foot and ankle, the most important complication is peripheral neuropathy, which usually occurs in a long-standing diabetic. Peripheral neuropathy leads to the possibility of skin breakdown and subsequent ulceration and infection. In an athlete's foot and ankle, skin integrity can be a concern, regardless of diabetes. Callus formation, blisters, abrasion, and fungal infections are very common in athletes. In the setting of diabetes, these conditions can lead to ulceration and bacterial infection and potentially may develop a serious complication faster than in a nondiabetic athlete.

Skin ulceration is a significant concern for all diabetic athletes. Cellulitis can develop quickly. Even worse is the possibility of osteomyelitis. Left untreated, these complications could be career altering or even career ending. Most plantar wounds or ulcers in a diabetic are polymicrobial. Superficial skin infections on the dorsum of the foot or around the ankle may be less likely to be polymicrobial, but if empiric treatment is warranted, standard regimens to cover typical pathogens for diabetic ulcerations should be used. Proper wound care is essential, and weight-bearing activities may have to be restricted temporarily. One special note is that deep foot ulcers with signs of cellulitis may be infected with *Pseudomonas* because athletic shoes may harbor these bacteria. Lastly, deep ulcers need debridement and/or other investigation to search for osteomyelitis, although this would be unusual in the athlete.

Diagnosis and testing of diabetes is beyond the scope of this chapter, but it is important to note that monofilament tactile and vascular examinations are essential for the evaluation and monitoring of diabetic neuropathy. Routine diabetic care is essential for tight control of glucose levels and prevention of complications. It also is important to note that sports participation should be encouraged in the diabetic population because physical activity can have beneficial effects on the disease as a whole. Simply keep in mind that more attention must be paid to lower-extremity skin care in the athlete. In individuals with foot alignment prone to callus formation, such as a cavus foot, professional callus shaving may be warranted. Orthotics may be useful in spreading out load-bearing surface of the foot and may help to alleviate pressure spots before they can ulcerate.

Diabetics have other complications that can affect the athlete's performance and general health, but one that can have specific foot and ankle relevance is the fact that diabetics have a higher incidence of osteoporosis and may have an increased rate of stress fractures. The key is focusing on the foot and ankle but remembering to see the athlete as a whole person.

Metabolic Bone Disease

Metabolic bone disease encompasses any disorder that changes the mineralization of the normal skeleton. Osteoporosis is the most common metabolic bone disease that could affect the foot or ankle. This is a concern primarily in the mature or elderly athlete.

Osteoporosis and osteopenia are common disorders, especially in postmenopausal women. However, they do not usually affect the foot and ankle. The most common sites of fracture in osteoporosis are the spine, wrist, ribs, pelvis, hip, and humerus. Osteoporosis is a concern in mature or elderly athletes because weaker bones may lead to an increased fracture rate or recurrent fractures. Osteoporosis and osteopenia are abnormalities of the bony matrix, where bone is less dense but of normal architecture. Other metabolic bone diseases may not have normal bony architecture, such as osteomalacia.

Bone densitometry (dual energy x-ray absorptiometry [DEXA] scan) is the test of choice for diagnosis of osteoporosis. Standard radiographs are unreliable. DEXA scanning will differentiate osteoporosis from osteopenia. A DEXA score of 2.5 standard deviations below the mean is diagnostic of osteoporosis. Scores of 1.0 to 2.5 are diagnostic of osteopenia.

Treatment of osteoporosis is beyond the scope of this chapter (see also [Chapter 4](#)), but a brief summary follows. The best

treatment is prevention. Calcium intake should be at least 1000 mg/day in an adult, and vitamin D (800 IU per day) is needed to aid in the absorption of the calcium. Weight-bearing resistance exercise also is important in building and maintaining strong bones. Once osteoporosis has been diagnosed, several treatment options exist. Calcium and vitamin D need to be taken, but they will not adequately increase bone density. At this time bisphosphonates (e.g., alendronate and risedronate) are a first-line treatment for increasing bone density. Estrogen increases bone density in postmenopausal osteoporosis but has other significant tissue effects that need to be taken into account before use. Selective estrogen-receptor modulators (raloxifene and tamoxifen) can prevent bone density loss and decrease fractures. Parathyroid hormone actually can stimulate osteoblastic activity if the concentration is not too high. Follow-up DEXA scanning is important to monitor therapy.

Most cases of osteoporosis are idiopathic, age-related, or postmenopausal. There are many secondary causes that are not as common but need to be kept in mind. Please see [Box 12.1](#) for a list of these secondary causes. The majority of patients with osteoporosis will be older recreational athletes, but bone loss can occur in a younger athlete. The classic scenario in a younger patient would be a college-age, female runner with recurrent stress fractures and an eating disorder and who is anovulatory. This is the classic female athletic triad (see [Chapter 28](#)). The results of the female athletic triad syndrome include metabolic bone disease and can lead to an increased rate of stress fractures. Any patient with recurrent stress fractures or problems healing existing fractures must be evaluated for possible metabolic bone disease. Clinical judgment is needed to determine when to test an athlete for metabolic bone disease in the setting of recurrent stress fractures. There are no established guidelines for the number or frequency of fractures that necessitate further investigation. In our opinion,

BOX 12.1 Secondary Causes of Osteoporosis	
Nutritional	Marfan's syndrome
Inadequate calcium intake	Ehlers-Danlos syndrome
Malabsorption	Osteogenesis imperfecta
Bulimia or anorexia nervosa	Endocrine Disease
Exogenous Substances	Diabetes mellitus
Glucocorticoids	Hyperparathyroidism
Some chemotherapeutic agents	Hyperthyroidism
Excessive alcohol	Cushing's syndrome
Some anticonvulsants	Vitamin D deficiency (rickets/osteomalacia)
Cyclosporine	Hypogonadism
Tacrolimus	Growth hormone deficiency
Thyroxine	Functional
Bone Marrow Disease	Prolonged immobilization or disuse
Leukemia	Miscellaneous
Lymphoma	Postsurgical
Myeloma	Subtotal gastrectomy
Metastatic carcinoma	Celiac disease
Bone cysts	Renal
Rheumatologic/Connective Tissue Disease	Renal tubular acidosis
Rheumatoid arthritis	Hypercalciuria

there is no specific number of fractures needed to prompt workup for metabolic bone disease, but if there is enough clinical evidence to suggest metabolic bone disease, a workup is warranted (i.e., two to three stress fractures within a 2-year period).

Workup for metabolic bone disease is directed toward the suspected cause. For example, in a mature fitness athlete with recurrent stress fractures the cause is most likely to be a result of idiopathic or primary osteoporosis, and initial workup would start with a DEXA scan. A significantly different approach would be the case for a teenage girl with recurrent stress fractures and would include a more detailed dietary and menstrual history, as well as laboratory workup.

Medications/Supplements/Deficiency States

Several medications, supplements, or deficiencies can result in disease-like states that can result in foot and ankle issues in an athlete. Most cases concern medications or supplements that result in metabolic bone disease. Table 12.1 has several examples of medications that can cause osteoporosis. Also, deficiencies can result in metabolic bone disease. The obvious is calcium deficiency, but other states can lead to osteoporosis as well. Examples include growth hormone deficiency, thyroid hormone deficiency, and hypogonadism. Please see Table 12.1 for more examples. In some cases, excess states can lead to metabolic bone disease. Hyperparathyroidism and Cushing's disease would be examples. In addition, medications, supplements, or deficiency states can lead to other conditions that can affect the foot and ankle. Vitamin B12 or folate deficiency can lead to a peripheral neuropathy, which in turn could lead to some of the same concerns that a diabetic athlete may have. The bottom line is to search for clues to the underlying cause and, if possible, correct the disorder, discontinue the medicine or replete the deficiency.

CASE STUDY 12.3 Female Athlete Triad

A 19-year-old, female, college freshman, cross-country/track athlete presents with a 2-week history of gradually worsening left foot pain. The pain initially was present at the start of her runs and became worse as she tried to run through the pain. Now the pain is present with activities of daily living (ADLs). Over the last 24 hours, her pain has worsened significantly. She has noted some mild swelling in the area of her dorsal midfoot/forefoot. About 1 month ago she added some runs outside of her usual training runs/practices. She has concern for a possible stress fracture as she has a history of prior stress fractures (three fractures during her senior and junior years of high school). The rest of her history of present illness is noncontributory. She is on no medications but admits to the use of over-the-counter (OTC) diet pills. She has a history of "spotty" periods and has not had a period since she was a sophomore in high school. The rest of her past medical history is noncontributory. Physical examination demonstrates a height of 5 feet 6 inches and a weight of 105 lb (BMI = 17), minimal erosions of the enamel of the teeth and fine hair on the arms but is otherwise noncontributory. X-rays show a completed fourth metatarsal stress fracture. Laboratory studies including CBC, electrolytes, thyroid, and hormonal status tests are noncontributory. DEXA testing shows bone mineral density 2.5 standard deviations below the mean of young adults. A multi-team approach was used to treat the athlete and involved the team internist, a dietician, and a sports psychologist. Treatment included a walking boot for the stress fracture with activity modification, increased caloric intake and calcium supplementation to 1500 mg per day, hormonal supplementation, counseling, and involvement of the athlete's family for emotional support.

VASCULAR/ LYMPHATIC DISORDERS

Arterial Disease

Arterial disease represents decreased blood flow to the lower extremity. The most common cause is occlusive disease secondary to atherosclerosis and associated embolic phenomenon. It is uncommon in young healthy athletes unless there is a genetic predisposition or severe risk factors. It is most common in middle-aged to older-aged recreational athletes, especially those who have concurrent disease, that is, diabetes or elevated triglycerides or cholesterol. It presents as claudication of the lower extremities, which is defined as exercise-related pain. Evaluation at rest, unless late in the disease, may be entirely normal, although decreased lower-extremity pulses may be present. The disease usually is progressive, causing increased pain at lesser workloads. Evaluation may include arteriography, and definitive treatment may require vascular surgery.

Claudication in a young athlete may be caused by popliteal artery entrapment (see Chapter 23). Its cause is either an entrapment of the popliteal artery in the popliteal fossa secondary to an anatomic variation of the popliteal artery and surrounding myofascial structures or a functional entrapment compressing the artery by the exercising muscles and surrounding bone. It has an 85% male preponderance and usually occurs in the second or third decade. It is bilateral in 25% of cases. The athlete usually complains of cramping to the calf and foot with associated numbness or paresthesias. In 10% of patients, there are acute or chronic ischemic changes of the lower extremity, including skin and temperature changes as well as rest pain and possible tissue necrosis. Physical examination usually is normal, but the diagnosis may be suspected if pulses diminish in the affected extremity with maximal ankle dorsiflexion or with active plantarflexion with the knee fully extended. However, these examination findings also are found in normal individuals who have no lower-extremity complaints. Evaluation usually includes noninvasive vascular studies, including lower-extremity Doppler, preexercise and postexercise ankle/brachial blood pressure indices, continuous wave Doppler ultrasound with provocative maneuvers, mentioned previously, and a duplex ultrasound that combines anatomic evaluation with quantitative and qualitative analysis of arterial blood flow. The gold standard for evaluation, however, is arteriography. Treatment involves surgical release of the entrapped artery. Although few long-term studies exist regarding the prognosis of popliteal artery entrapment syndrome, studies suggest that the prognosis is most favorable if no arterial damage has occurred at the time of diagnosis and treatment.

Raynaud's phenomenon is manifested by pallor and cyanosis of the digits in response to some type of stressor, usually exposure to the cold but also possibly secondary to an emotional distress. It can present at any age but is most common in women between the ages of 20 and 40 years. It has an unknown etiology. Patients usually have no findings at the time of physical examination. Occasionally, some bluish discoloration of the tips of the digits may be present between attacks. A typical attack causes the digits to become pale and cyanotic with a sharp demarcation of these findings with the skin more proximally. Raynaud's may be associated with other diseases such as scleroderma, and when this occurs, it is referred to as Raynaud's phenomenon. When just the Raynaud's findings are present without concurrent other disease, then it is called Raynaud's disease.

The prognosis for Raynaud's patients generally is good. For athletes who are exposed to cold weather conditions, protective clothing is usually sufficient. More severe cases may require a pharmacologic treatment, which may include calcium channel blockers, alpha-adrenergic blockers, or vasodilators.

Another condition that may affect the foot during cold weather outdoor activities is chilblain or pernio. Chilblain is an inflammatory disorder of the skin induced by cold temperature. It often affects women in the second or third decade of life. The etiology of chilblain is unknown. It presents as bluish red edematous areas of the skin overlying the lower extremities. Patients may complain of itching/burning to the areas of skin change. Repeated exposure may cause the lesions to become chronic and ulcerative. The lesions generally resolve with avoidance of the cold. Often, however, there will be a permanent area of hyperpigmentation at the prior site of the lesions.

Venous Disease

Thrombophlebitis is uncommon in a young, healthy athlete. It may occur from direct trauma from a contact sport, especially in association with postgame travel in an away team returning to the home location or following limited activity after a significant injury or elective surgery. A previous history of thrombophlebitis may predispose an individual to a second episode. Three factors as part of Virchow's triad may lead to the formation of a thrombosis, and these include venous stasis, injury to the venous wall, and a hypercoagulable state. Any unexplained swelling associated with lower-extremity erythema and increased temperature should raise the suspicion of a venous thrombus.

The main concern in detecting a venous thrombus is to determine whether the lesion occurs within the superficial venous system or the deep venous system. Superficial lesions are treated symptomatically and may present as tender, erythemic, palpable cords within the subcutaneous tissue. However, because of the potential serious complications of a deep venous thrombus, definitive study should be obtained to rule out any deep system involvement if there is any question regarding the presentation. Testing involves noninvasive, lower-extremity Doppler examination that provides an approximate 90% accuracy. If deep venous thrombosis is discovered, treatment involves rest and initiation of anticoagulation therapy. Anticoagulation therapy usually is instituted for 3 to 6 months for the first episode and may require chronic anticoagulation therapy for repeated episodes. Anticoagulation reduces the likelihood of further formation of the thrombus and lessens the potential complications of embolic phenomenon. Measures aimed at correcting any underlying risk factors such as minimizing immobilization and treating any cause for the hypercoagulable state also are recommended.

Varicose veins are prominent, abnormally distended, tortuous, superficial veins of the lower extremities that occur in approximately 20% of adults. The cause is usually one of defective valves within the veins or congenitally absent valves. They are more common in females and often are associated with a family history of varicosities. Any condition that decreases venous outflow from the lower extremities, that is, pregnancy, also may cause varicosities.

Normal venous return from the lower extremities usually is accomplished by contraction of the lower-extremity musculature

to pump the blood back up the venous gradient. Intact/competent venous valves prevent back flow. When the valves are incompetent or absent, pooling blood distends the veins, leading to further obstruction that causes worsened flow from the lower extremities. An exercising athlete with varicose veins further worsens this condition because of increased arterial flow into the exercising lower extremities. Usually this worsening of the venous return during exercise has little effect on exercise tolerance. Some athletes, however, may complain of a nonspecific heavy sensation to the extremities with exercise. This vague, exercise-related discomfort is known as "venous claudication."

If venous congestion of the superficial system progresses, it may lead to involvement of the deep venous return. This may then result in chronic edema, venous dermatitis, and/or stasis ulcers. Treatment is initially symptomatic using elevation and support stockings. Surgical vein stripping also may be an option for persistent problems, which do not respond to a more conservative approach. Proper skin care to treat the chronic dermatitis and any ulcers that may develop also is necessary.

Lymphatic Disease

Other sources of edema of the lower extremities, but usually not associated with pain, are abnormalities of the lymphatic system. Lymph vessels serve to transport lymph fluid back to the venous system through the thoracic duct at the left jugular vein. At lymph node junctions along the lymph system, immunologic and filtering is done to the lymph fluid. Lymphatic channels, which normally follow the venous tree, are susceptible to many of the same forces that affect the venous system and include trauma, mechanical obstruction, and surgical removal of lymph nodes as well as venous hypertension.

Primary lymphedema is a disease of the lymph systems with an unknown cause. It is most common in females and often is unilateral. It usually has onset before the age of 40. The diagnosis may be confirmed with either lymphogram or contrast lymphangiography. Treatment is symptomatic and aimed at reducing the lower-extremity edema with elevation, support stockings, and, occasionally, diuretics. Chronic lymphedema may cause recurrent skin infections, which in turn lead to an overload of the lymph system, causing further edema. Rarely, surgical intervention may be necessary.

CASE STUDY 12.4 Popliteal Artery Entrapment

A 17-year-old, high school senior cross-country runner presents with a several-month history of exercise-related left calf and foot pain. The pain is described as cramp-like in quality and has become progressively worse with time. The pain has become more intense and has onset earlier in his runs. There are no associated paresthesias, increased tension to the calf musculature, or loss of foot or ankle control during the runs. The pain will resolve after several minutes of rest, but with return to running after resolution, the pain will return almost immediately. There are no symptoms noted on the right or symptoms outside of activity. Past medical history and family history are noncontributory. Physical examination is noncontributory except that with the knee in full extension and forced dorsiflexion or active plantarflexion, the dorsalis pedis and posterior tibialis pulses diminish. X-rays are negative. Superficial and deep posterior chronic exertional compartment testing is negative. Arteriography demonstrates entrapment of the artery at the knee. The athlete is treated surgically with release of the artery and makes a gradual return to running.

CONCLUSION

In summary, inflammatory metabolic, vascular diseases are common in the general population but uncommon causes of foot and ankle concerns in athletes. However, being attuned to the possibility of these disease processes complicating an athlete's ability to perform his or her chosen sport can allow the physician to address these issues and enhance the athlete's performance or enjoyment of sport.

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Dermatologic, Infectious, and Nail Disorders

Andrew M. Tucker

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INTRODUCTION

The majority of sports and fitness activities place extraordinary stresses on the feet. In addition to the potential for musculoskeletal injury, the sport participant's foot is at risk for a number of dermatologic and infectious conditions that range from incidental to potentially disabling. Direct pressure from shoe or surface, shear stresses from running/jumping/cutting activity, and the increase in moisture from perspiration are predisposing factors to these commonly seen conditions. The increase in moisture at the outer layer of skin (stratum corneum) can enhance the passage of microorganisms through the skin. Certain anatomical issues of the foot or lower leg, and extreme participant size in some sports, increase the stressors on the skin structure. Cuts and abrasions are less common in the feet than other sites on the athlete, but can occur in certain settings and also increase the risk of infection. In addition, the athlete's environment, including playing surfaces, locker rooms and shower areas, may contribute to risk of certain types of infection.

This chapter will review the most common dermatologic and infectious issues that present to the medical staff in the training room and clinic setting, and focuses on the presentation, examination findings, and treatment strategies with the goal of maintaining optimum health and performance.

SKIN CONDITIONS CAUSED BY MECHANICAL STRESSES/TRAUMA

A myriad of trauma-related skin conditions occur commonly in athletes, and the foot is a common site for these typically benign findings that usually do not interfere with participation and training, but can be problematic in certain circumstances if left

untreated. Also, identification and education to the athlete can help alleviate fear of a more ominous disorder.

Blisters

Blisters are caused by repetitive friction between skin and shoe and/or sock. Moisture from perspiration, increased heat, and poorly fitting shoes increases the risk of blister formation. Underlying bony abnormalities of the foot may also increase the risk of formation. Blisters are formed from a split between the epidermis layers that fills with transudate. Blisters are among the most common complaints in surveys of runners and other athletes and typically appear in toes, plantar surfaces of the metatarsals heads, and heels.^{1,2}

Blisters appear as areas of erythema followed by formation of the vesicle or bullae (Fig. 13.1). Formation is often accompanied by sensation of burning or stinging. The fluid filled lesions may appear dark when blood fills the blister cavity. Data has demonstrated that blisters are best treated with sterile drainage using a blade or needle without disturbing the roof. The roof of the blister can adhere to the base of the lesion, providing protection and decreasing risk of infection.³ After drainage, petroleum jelly and occlusive dressings may be used to protect the healing lesion. Commercially available products, such as hydrocolloid pads, are available that can be applied daily to promote healing (2nd Skin). Athlete and clinician should continue to monitor the affected area for secondary infection.

Prevention of blisters includes properly fitted footwear, moisture wicking socks, and consideration of extra padding over bony prominences. Athletes have often used two pairs of socks to help distribute the frictional forces to the foot. Drying powders, such as aluminum chloride, may be used to decrease moisture around the foot. Agents such as 10% tannic acid soaks may assist in hardening the skin.⁴



Fig. 13.1 Blister formation on plantar surface of toe.



Fig. 13.2 Callus on plantar aspect of foot, with hyperkeratosis and prominence of skin lines.

Calluses

Calluses are defined as hyperkeratotic plaques created by repeated friction and pressure on the skin (Fig.13.2).⁵ Most calluses are painless, but can become symptomatic when extremely thick and place excessive pressure on underlying structures. The differential diagnosis includes plantar warts and corns. The diagnosis can be confirmed with paring of the lesion. Warts contain the characteristic black dots of tiny thrombosed vessels, while corns contain a central core. When treatment is required, gentle paring with a blade or pumice stone will debulk the lesion and provide relief. Prevention of calluses can include synthetic socks, lubricant jelly, and properly fitting shoes in order to minimize friction and pressure across the skin. In symptomatic cases, where underlying anatomical abnormalities are causative, surgical treatment may be indicated.

Corns

Corns, similarly as calluses, may present in two ways. Hard corns (clavus durus) are typically found on the sole or dorsum of the toes (Fig.13.3). Soft corns (clavus mollis) are often found in the interdigital area due to pressure from one toe on another

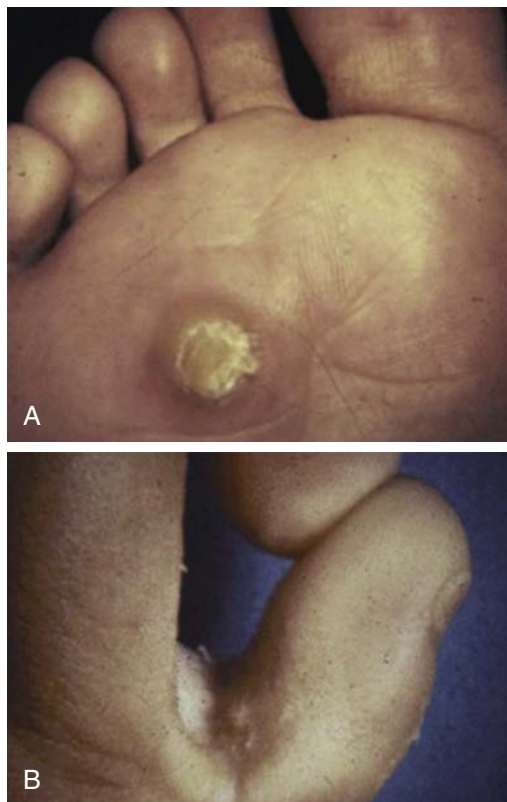


Fig. 13.3 (A) Hard corn characterized by well-demarcated hyperkeratotic skin. (B) Soft corn located in interdigital space.

and are often painful.^{5,6} Diagnosis is confirmed by paring of the lesion. Corns are characterized by a central core, while calluses show normal skin lines and plantar warts the characteristic black dots. Paring will provide pain relief, and pads may help redistribute and minimize pressure. In addition to mechanical or sharp debridement, electrosurgery has been used successfully as an alternative treatment.⁷ Nonsurgical treatment options include warm water soaks and keratolytics (salicylic acid pads), topical or intralesional steroids, or topical retinoids.⁴ Preventive measures are similar to those for calluses and blisters.

Nodules

Athlete's nodules have been classically described as small, flesh-colored lesions, usually occurring at the dorsum of the toes, foot, or anterior ankle and lower leg due to repetitive pressure and friction of tight fitting shoes, skates, or boots. Comparable lesions have been described on the hands of boxers and laborers.⁸ The lesions typically present insidiously and are often painless. Occasionally, the lesion may reach a critical size and cause some pressure-related discomfort that may require attention. The histology of these lesions often will show increase in collagen density in the dermis with relatively normal epidermis.⁸ The differential diagnosis can include ganglion, granuloma annulare, rheumatoid nodule, gout, foreign body reaction, and elastoma.⁶

Treatment is often not necessary but, when symptoms dictate, can include medical treatment with topical keratolytics (salicylic acid), or intralesional injection of corticosteroid to address the thickened collagen in the dermis.⁴ Surgical removal is an option for recalcitrant cases, and would be reserved for the athlete's off season if possible.

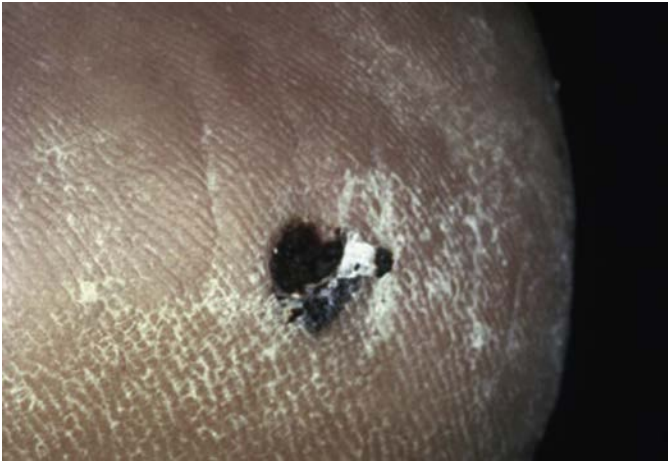


Fig. 13.4 Black heel (talon noire) – painless black macule on heel, can resemble melanoma. (From Williams JD, et al. *Dermatoses Resulting From Physical Factors*. In: Williams JD, *Andrews' Diseases of the Skin* 13th edition. 2020, Elsevier. 18-45.e3. Fig. 3.36)



Fig. 13.5 Flesh colored painless papules of the medial heel consistent with piezogenic papules. (From Smith, ML. *Environmental and Sports-Related Skin Diseases*. In: Bologna, J ed: *Dermatology* 4th edition. Philadelphia, Elsevier. 1569-1594. Fig. 88.17)

Black Heel

Black heel, also called “talon noire”, has been described in multiple sports including basketball, football, lacrosse, soccer, and tennis.⁹ Sheering force caused by the athlete’s rapid stops and starts is the cause, resulting in tiny blood vessels rupturing in the papillary dermis. The presentation is a painless black macule that is well defined, usually less than 1 cm, on the posterior heel or plantar aspects of the toes (Fig. 13.4). The differential diagnosis includes melanoma, melanocytic nevus, and tinea nigra. Paring of the superficial layers of the stratum corneum removes the hemorrhage, though no specific treatment is necessary for this asymptomatic lesion. Hemocult testing of the material can confirm blood and give reassurance. Punch biopsy is appropriate if melanoma is suspected.

Piezogenic Pedal Papules

This entity is characterized by multiple small flesh or yellow appearing papules that are painless and found in the posterior, medial, or lateral surface of the calcaneus (Fig. 13.5). The cause is subdermal fat herniation and their appearance is most noticed with prolonged weight-bearing and has been observed



Fig. 13.6 Repetitive trauma of the second toe resulting in subungual hematoma (“tennis toe”). (From Smith, ML. *Environmental and Sports-Related Skin Diseases*. In: Bologna, J ed: *Dermatology* 4th edition. Philadelphia, Elsevier. 1569-1594. Fig. 88.16)

in runners and other sports.¹⁰ When symptomatic, multiple treatments have been tried with limited success including injectable corticosteroids, compression, and heel cups and padding.¹¹ However, in most athletes, benign neglect after educating the athlete is the treatment of choice.

NAIL CONDITIONS CAUSED BY TRAUMA

A number of conditions have been described that share a common mechanism of acute or repetitive trauma to the nail, including “tennis toe” or “jogger’s toe” (Fig. 13.6).¹² The longest toes (first or second) are usually affected. Acute stress from a sudden stop/start or repetitive stresses from long distance walking or running can be causative as the affected nail is pushed against by the toe box. The nail may become acutely painful due to subungual hemorrhage, or may be asymptomatic. The differential diagnosis includes fungal infection, melanoma, or psoriasis. Fungal infections tend to cause yellowish, hyperkeratotic changes to the nail. Culture may be needed to confirm the diagnosis. Melanoma may be difficult to distinguish and requires a biopsy. Hutchinson’s sign, pigmentation of the nail and the nail fold, suggests melanoma (Fig. 13.7).¹³

Treatment may include simple observation, and the traumatic nail changes resolve with rest from the offending activity. For acute subungual hematomas, drainage with a heated paper clip or needle boring through the nail will provide quick relief. The nail should be covered with sterile dressing until the nail heals. Prevention relies on properly fitted footwear and trimming of the nails. Toe caps can provide protection inside the shoes.

Onychocryptosis (Ingrown Toenail)

Onychocryptosis typically occurs when a curved great toenail causes excessive force and stresses on the adjacent soft tissue, producing pain and inflammation. Predisposing factors include



Fig. 13.7 Pigmentation of the nail and nail fold suspicious for melanoma (Hutchinson's sign).

trimming the nail excessively short and curving the edges of the nail.⁶ Treatment can include removal of the offending portion of the nail versus nonoperative treatment of frequent soaks and modification of footwear.¹⁴ The area should be monitored for development of more serious secondary bacterial infection. Prevention includes trimming the nail straight across without rounded edges, properly fitted footwear, and foam toe caps. In rare circumstance, repeated onychocryptosis recalcitrant to partial nail excision, can require partial or complete nail bed ablation.

INFECTIONS OF THE SKIN AND NAILS

Infections of the feet are common in athletes and fitness participants due to increased moisture from perspiration that facilitates growth and entry of microorganisms through the skin, and to the athlete's environments that may harbor increased concentrations of bacteria and fungi.

Fungal Infection of the Skin

Tinea infections are caused by the dermatophytes trichophyton, microsporum, and epidermophyton.¹⁵ Tinea pedis, fungal infection involving the foot, may present in three ways. The most common, caused by *Tinea rubrum*, presents as mild to moderate erythematous scaly plaques in a moccasin distribution (Fig. 13.8). The interdigital type, also caused by *Tinea rubrum*, is characterized by macerated plaques between the toes. A third presentation causes pruritic erythematous vesicles and plaques on the instep, and is caused by *Tinea mentagrophytes*.⁶

The differential diagnosis of tinea pedis includes contact dermatitis, psoriasis, and pitted keratolysis. The diagnosis can often be successfully made on clinical grounds. However, potassium hydroxide slide exam or culture may be used to confirm the diagnosis. The clinician must be aware that secondary infections with bacteria, such as staphylococcus or pseudomonas, commonly accompany fungal infections, complicating both diagnosis and treatment. Culture may be helpful to confirm co-infection.



Fig. 13.8 Diffuse scaling of tinea pedis (moccasin type). (From Vega-Lopez F. Dermatological Problems. In: Farrar J, et al. *Manson's Tropical Diseases*. Philadelphia, Elsevier. 2014. 995-1026.e1. Figure 68.22)

Treatment for mild to moderate fungal infection of the feet usually involves a topical antifungal cream for 2 to 4 weeks. The allylamine creams are fungicidal and are preferred.¹⁵ Ciclopirox is both fungicidal and antibacterial. For severe fungal infections, oral therapy with terbinafine or itraconazole may be used for 2 weeks, in addition to the topical treatment. These oral antifungals may be contraindicated if the athlete is taking a number of common medications, including quinidine, benzodiazepines, and statin drugs. Blood counts and liver function tests are recommended for athletes taking oral antifungals for a month or more.⁶

Additional treatment may include drying agents (Domeboro solution, aluminum chloride) to assist in the treatment of weeping lesions. Extreme pruritis may require use of mild to moderate-potency corticosteroids, in addition to the appropriate antifungal medication.

Prevention of these common infections includes use of moisture-wicking socks, the immediate removal of wet socks after activity, thorough cleaning and drying of feet, and proper cleaning of locker room, showers, and pool decks to minimize the growth of common fungi. Studies have shown that regular use of topical or oral antifungal medication can reduce the incidence of infection in susceptible athletes.¹⁶

Fungal Infections of the Nail

Tinea unguium, also called onychomycosis, is common among athletes, as the predisposing factors for fungal skin infections translate to increased risk for infection of the nail. Dermatophytes from the genus *Trichophyton* are the most common causes, specifically *Tinea mentagrophytes* and *Tinea rubrum*.⁶

The clinical presentation is that of a thickened distal nail, usually yellowish in color (Fig. 13.9). The patient usually complains of the cosmetic appearance of the nail rather than pain; however, the nail may be thickened to the point of causing discomfort. The differential diagnosis includes the previously described traumatic nail conditions (subungual hematoma) and psoriasis.



Fig. 13.9 Onychomycosis of the great toe with yellow, thickened nail.

Diagnosis is usually made on clinical grounds, but may be confirmed by KOH slide test or culture. Treatment resulting in cure is difficult. Oral terbinafine or itraconazole is recommended for 12 weeks, but resolution is difficult to attain.¹⁷ Alternative treatments include laser therapy. Extended use of oral antifungal medication necessitates monitoring of blood counts and liver function tests before and after drug regimen. Prevention involves the same measures as those for skin fungal infections.

Bacterial Infections of the Skin

Bacterial skin infections are common in athletes, though perhaps more likely at sites where abrasions and minor lacerations occur in the exposed skin of the head, neck, and extremities. Bacterial skin infections likewise can occur in the feet as a result of the same predisposing factors as fungal infections, including excess moisture from perspiration facilitating microorganism entry through the skin. Skin breakdown from blisters or trauma from ingrown toenails may predispose to infection. Secondary bacterial infections can occur at the site of fungal infections, and require specific treatment.

Bacterial skin infections may be caused by a number of gram positive or gram negative bacteria. *Streptococcus* and *staphylococcus* species are common causes of skin infections. Outbreaks of bacterial skin infections, including methicillin resistant *staphylococcus aureus* (MRSA), have been documented in several team sports in recent years (Fig. 13.10).¹⁸ MRSA infections have become more prevalent in the outpatient setting and account for over half of skin and soft tissue infections (SSTIs) in U.S. emergency departments.¹⁹ Clinicians are encouraged to consider MRSA as a cause for any purulent soft tissue infection, including those of the foot. In general, treatment for localized infection without systemic signs or symptoms is usually incision and drainage, with or without oral antibiotics.¹⁵ In the sports medicine setting, mild to moderate MRSA infections are most typically treated with clindamycin, trimethoprim-sulfamethoxazole, or doxycycline, though local variations in microbial sensitivity may drive antibiotic choice.



Fig. 13.10 Evolving staphylococcal infection with redness, swelling, and purulent drainage.

Impetigo

Impetigo is a common infection of the superficial epidermis and is usually caused by *staphylococcus aureus* and group A beta-hemolytic *streptococcus pyogenes*.²⁰ Infections most commonly affect young children, though may occur at any age. Face and upper extremities are more commonly involved than the feet and lower extremities. The classic lesions are characterized by erythematous plaques and papules with honey-colored crust. Pustules may or may not be present. Diagnosis is typically made clinically and can be confirmed by culture. Treatment includes warm water soaks and topical mupirocin twice daily for 10 days is as effective as oral antibiotics for limited disease. Oral antibiotics are necessary for more widespread disease, and selection is based on culture results and patient tolerance. Systemic antibiotic choices include beta-lactamase resistant penicillins (cloxacillin, dicloxacillin), broad spectrum penicillins, cephalosporins, or macrolides. Treatment duration is typically 10–14 days.²⁰

Folliculitis

Folliculitis may affect the lower legs and generally is caused by *staphylococcus aureus*, *streptococcus*, or *pseudomonas*, though mechanical or chemical causes are also possible.¹⁵ Association with exposure to water or hot tubs has led to the common reference of “hot tub folliculitis.”²¹ Fungal etiology is uncommon but should be considered in resistant cases. These discrete erythematous pustules are seen in association with hair follicles, and are most common on the back and extremities (Fig. 13.11). Diagnosis is generally made clinically, but culture may be necessary in certain resistant cases. Treatment of limited lesions is with mupirocin topical twice a day. More severe disease requires treatment with cephalexin, dicloxacillin, or flucloxacillin.

Cellulitis

Cellulitis is a bacterial infection of the subcutaneous tissue and presents as a rapidly expanding, painful, indurated area with overlying erythema and warmth (Fig. 13.12).¹⁵ In addition, systemic symptoms of fever, chills, and malaise may be present.



Fig. 13.11 Folliculitis with small erythematous pustules associated with hair follicles.



Fig. 13.12 Cellulitis of the leg characterized by expanding erythematous border, tenderness, and warmth of the skin.

Group A beta-hemolytic streptococcus and staphylococcus aureus are the most common causes and methicillin resistant staphylococcus aureus must be considered.²² Diagnosis is clinical, and Gram stains and cultures from infected material may assist in identification. Blood cultures are obtained when the patient presents with systemic symptoms. Treatment should not be delayed. If MRSA is considered, sulfamethoxazole-trimethoprim, clindamycin, or doxycycline is recommended. Patients with systemic symptoms or unstable vital signs require immediate hospitalization and parenteral antibiotics.¹⁵

Furunculosis (Boils)

Furunculoses are uncommon in the lower legs and feet and present as tender erythematous lesions that can progress from small (1 cm) to several centimeters over the course of 1 to 2



Fig. 13.13 Characteristic pits of keratolysis are caused by enzymatic breakdown of the stratum corneum.

days.¹⁵ The lesion may become fluctuant and drain. Any draining or fluctuant lesion should be cultured. Antibiotic selection depends on culture, but empiric treatment covers MRSA and trimethoprim-sulfamethoxazole, doxycycline, or clindamycin can be used until culture results are available.

Pitted Keratolysis

This infection is typically seen in adolescent and young adult basketball players, tennis players, and runners. Excessive perspiration and occlusive footwear are risk factors. A number of bacteria have been implicated, including corynebacterium, actinomyces, and streptomyces.^{23,24} The well-defined crater-like pits on the sole of the foot are due to enzymes that break down the stratum corneum, and a distinct foul odor is characteristic (Fig. 13.13). Topical therapies including clindamycin, mupirocin, and erythromycin topical solutions are effective treatment. Drying agents can also be helpful for treatment and prevention.

SUMMARY

The active patient and sports participant is at risk for a number of dermatologic and infectious conditions of the foot due to a number of predictable and modifiable factors. This chapter provides the sports medicine provider with an overview of the most common diagnoses, their presentation, clinical findings, and treatment options, with the goals of maintaining health and limiting interference with performance.

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Nonsurgical Treatment of Acute and Chronic Ankle Instability

Mary Hastings, Devon Nixon, Jeremy McCormick

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INTRODUCTION

Ankle sprains are common injuries in the athletic population, with an estimated 85% involving the lateral ligament complex.¹ The lateral ligament complex consists of the anterior talofibular ligament (ATFL), posterior talofibular ligament (PTFL), and the calcaneofibular ligament (CFL).² Cumulative data from a systematic review and meta-analysis estimated the incidence of ankle sprains at 12 per 1000 exposures based on the highest-quality studies reviewed, with athletes participating in indoor and court sports at greatest risk of injury.^{3,4} Moreover, a recent study of collegiate athletes found that at least 1 in 28 collegiate athletes sustains a lateral ankle sprain during each academic year, with the highest incidence in basketball and soccer.⁵ Duration of symptoms (acute versus chronic) helps treatment decision making; broadly, ankle instability can be managed nonsurgically (through short-term immobilization with bracing or casting and then early functional rehabilitation) or with surgery. This chapter will focus exclusively on the nonsurgical management of both acute and chronic ankle instability.

ACUTE LIGAMENT INJURIES

Diagnosis

Acute lateral ankle injuries most commonly occur following an excessive inversion and internal rotation of the hindfoot while the leg is in external rotation.¹ Accurate diagnosis is achieved through careful history and physical examination including anterior drawer and talar tilt ankle ligament testing (with comparison to the uninjured side) (Fig. 14.1 and Video 14.1). Acute ankle injuries are commonly graded I to III, with higher grades indicating more injury to the lateral ankle ligament complex. However, there are multiple grading schemes offered in the

literature, and grading may be difficult to assess in the acute setting, as specialized tests are poorly tolerated by patients. Fractures need to be identified when evaluating a painful foot or ankle. Ottawa ankle rules guide when radiographs are necessary, including when the patient is unable to tolerate weight bearing on the injured extremity or when there is tenderness at the medial malleolus, lateral malleolus, base of the fifth metatarsal, or navicular.⁶ Furthermore, the role for stress imaging in the acute setting to assess for excessive radiographic talar tilt has been debated. We do not routinely employ them in clinical practice.¹

Acute-Phase Treatment

The hallmark of treatment for an acute ankle injury involves early mobilization⁷ and a protocol of rest, ice, compression, and elevation (RICE).^{8,9} Rehabilitation after injury to the lateral ankle ligaments and surrounding tissues follows a continuum of care that is sensitive to the severity of injury and the injured tissues' ability to tolerate stress.¹⁰ During the early phase of rehabilitation, injured tissues poorly tolerate stress. The goal of acute ankle sprain treatment is to optimize tissue healing by protecting injured tissues and providing an environment that promotes tissue healing.

Role for Immobilization

Ankle sprain severity helps to determine the type and duration of immobilization and weight-bearing limitations.¹¹ Short-leg casts and nonweight-bearing restrictions are typically reserved for severe sprains or sprains that have impacted the integrity of the weight-bearing joint. Injuries that may require strict immobilization and nonweight bearing include grade III ankle sprains or perhaps more mild sprains in less compliant patients. In general, cast immobilization and/or

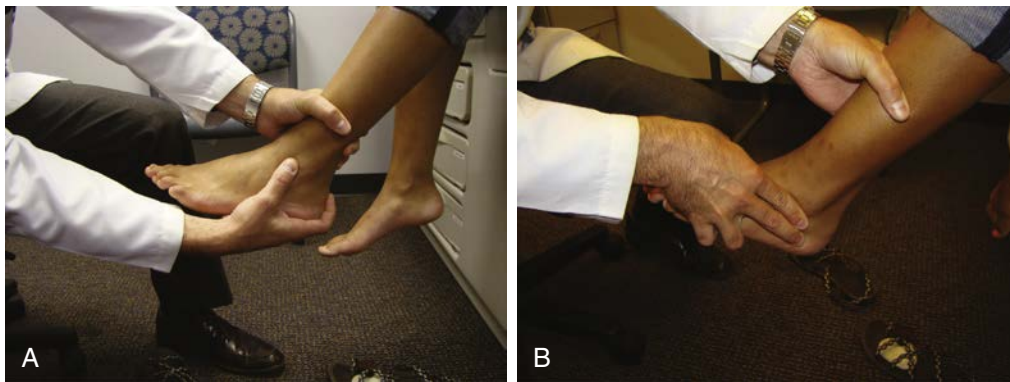


Fig. 14.1 (A) Anterior drawer test. The foot is translated anteriorly with a grasp of the heel with the foot in slight plantarflexion. Counterpressure is applied to the anterior leg with the opposite hand. (B) Talar tilt test. The foot is inverted with a grasp of the hindfoot with the foot in neutral dorsiflexion. By placing a finger on the lateral talar process one can more clearly discern ankle motion from subtalar motion. (From Irwin TA, Anderson RB, Davis WH. Principles of the physical examination of the foot and ankle. In: Coughlin MJ, Saltzman CL, Anderson RB eds. *Mann's Surgery of the Foot and Ankle*. 9th ed. Philadelphia, PA: Saunders; 2014:37-60, [Fig 2.29])



Fig. 14.2 Lace-up ankle brace for ankle sprain. (From Bret C. Jacobs, Justin A. Lee, Durable medical equipment: types and indications, *Medical Clinics of North America* 2014;98(4):881-893, Fig. 10.)

protected weight bearing should be brief for acute ligamentous injuries—some authorities even recommend that it not exceed 10 days.¹² However, immobilization duration differs in the presence of a fracture. Use of a walker boot and full weight bearing may be a reasonable consideration for patients with less severe ligament injuries and who might benefit from boot removal for range of motion (ROM) and active edema management. Rigid ankle braces are also suitable alternatives for immobilization (Fig. 14.2). Assistive devices (i.e., crutches, walker, and knee scooters) might be indicated if patients cannot ambulate without a limp. Once patients can ambulate without a limp in either a walker boot or ankle brace,¹¹ they then proceed to the next phase of rehabilitation.

As patients move to the next phase of rehabilitation, they may choose to maintain use of a supportive ankle brace or use ankle taping as they work through recovery.¹³ Taping has been shown in normal subjects with below-average proprioceptive scores to add proprioceptive feedback to ankles.¹⁴ Extrapolating this might suggest that a patient may feel better early in the recovery period after an acute ankle sprain when some of their proprioceptive feedback has been slowed relative to normal. Taping and bracing, however, should not be used as a substitute for the phases of rehabilitation reviewed below. These supportive tools can be used in conjunction with the rehabilitation protocol and should be weaned as a patient progresses through recovery and demonstrates return of proprioception and balance.

Edema Management

Edema management is critically important during the early phase of rehabilitation and is best managed with a multi-modal approach that includes:

- Rest (immobilization and weight-bearing restrictions as discussed above)¹¹
- Ice (potential regimen may include icing for 10 minutes on, 10 minutes off, 10 minutes on and then waiting 2 hours before repeating)¹⁵
- Compression (with compression garments and/or pneumatic compression devices)
- Elevation¹⁶
- Antiinflammatory medications

Educating patients to avoid gravity-dependent positions is crucial for maintaining edema reduction. Research demonstrates that ankle edema can return after only 5 minutes in the gravity-dependent position.¹⁷

Mobility

For uncomplicated ankle sprains, a randomized control trial demonstrated that mobility exercises initiated during the first week after injury can improve function and activity level without increasing pain, swelling, or risk of reinjury.¹⁸ The accelerated rehabilitation protocol proposed by Bleakley et al.¹⁸ was completed twice daily and included: 1) active ankle dorsiflexion/plantarflexion (20 repetitions); 2) active ankle clockwise and counter clockwise circumduction (20 repetitions); 3) active, combined hip, knee, and ankle flexion followed by full extension (30 repetitions); 4) resisted isometric dorsiflexion, plantarflexion, inversion, and eversion contractions (5 repetitions of each exercise, held for 10 seconds); and 5) standing gastrocnemius ankle stretching (3 repetitions, held 20 seconds each). Avoiding extremes and forced motions replicating the direction of injury (which is typically plantarflexion and inversion) will help to reduce tissue disturbances during the healing process. For more severe ankle sprains, foot and ankle ROM may need to begin following a brief period of immobilization.

Progressive-Phase Treatment

As patients advance through the acute treatment phase, additional exercises should be instituted to gradually increase stress on the injured tissues in a controlled manner. The use of activity monitors (with step goals and limits) as well as patient journals (to detail the type of new activities performed) help to guide the patient and therapist during treatment progression. Minor, transient increases in symptoms are expected during this rehabilitation phase; however, symptom exacerbation should not persist for more than a day and should not exceed a level that results in new restrictions on function.

Postural Control

Perhaps the most important component of rehabilitation following an ankle sprain is postural control (Fig. 14.3).^{19,20} These exercises will challenge the musculoskeletal, somatosensory, and cardiovascular systems, particularly as exercises increase in difficulty. The visual system can compensate for limitations in the somatosensory system – therefore, postural control exercises should progress from eyes open to eyes closed. Exercises should

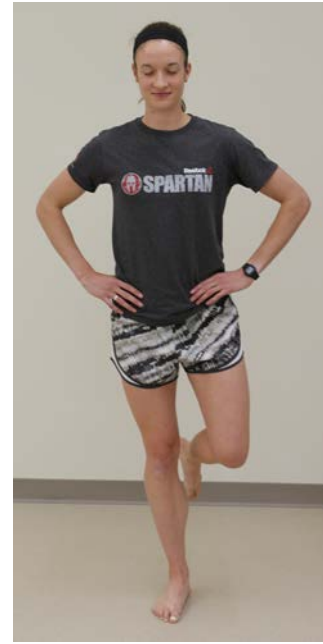


Fig. 14.3 Patient performing a single leg stance, eyes closed. Occluding visual input challenges postural control and is an important component of rehabilitation after an ankle ligament injury.

also progress from solid, predictable surfaces to softer, less predictable surfaces. Finally, the exercises should also incorporate external perturbations (e.g., being bumped) and unexpected challenges (e.g., responding to sport-specific needs). Treatment progression should also consider sport-specific environments. For example, soccer players may have ball-specific drills introduced into their rehabilitation protocol to assess their functional recovery and to identify deficits to be addressed in treatment. The Star Excursion Balance Test, a measure of targeted reach distance while balancing on the involved leg, can be a sensitive marker for identifying postural control deficits and tracking progression with rehabilitation (Fig. 14.4).^{21,22}

Strengthening

Ankle strengthening should progress from isometric exercises to resistance band exercises. If the targeted muscle is not fatigued during resistance, resistance should be increased. Both concentric and eccentric phases should be observed. Often eccentric motion has poorer control with a cogwheel appearance, so exercises should be tailored to address these impairments. If there is an ankle plantarflexion strength deficit, functional activities should progress to unilateral heel rises as well as bilateral and unilateral hopping. Careful attention should also be paid to eversion strength, as peroneal deficits can exacerbate feelings of ankle instability.²³ A complete rehabilitation plan should address strength of the uninjured limb and any hip abductor or external rotator strength deficits to improve poor lower extremity positional control noted during weight-bearing activities (i.e., single leg stance, jumping, or hopping).²⁴

Loading Tolerance

The overall volume and intensity of weight-bearing activities can be gradually increased as symptoms abate. Activity monitors and patient journals should be continued to aid in the safe

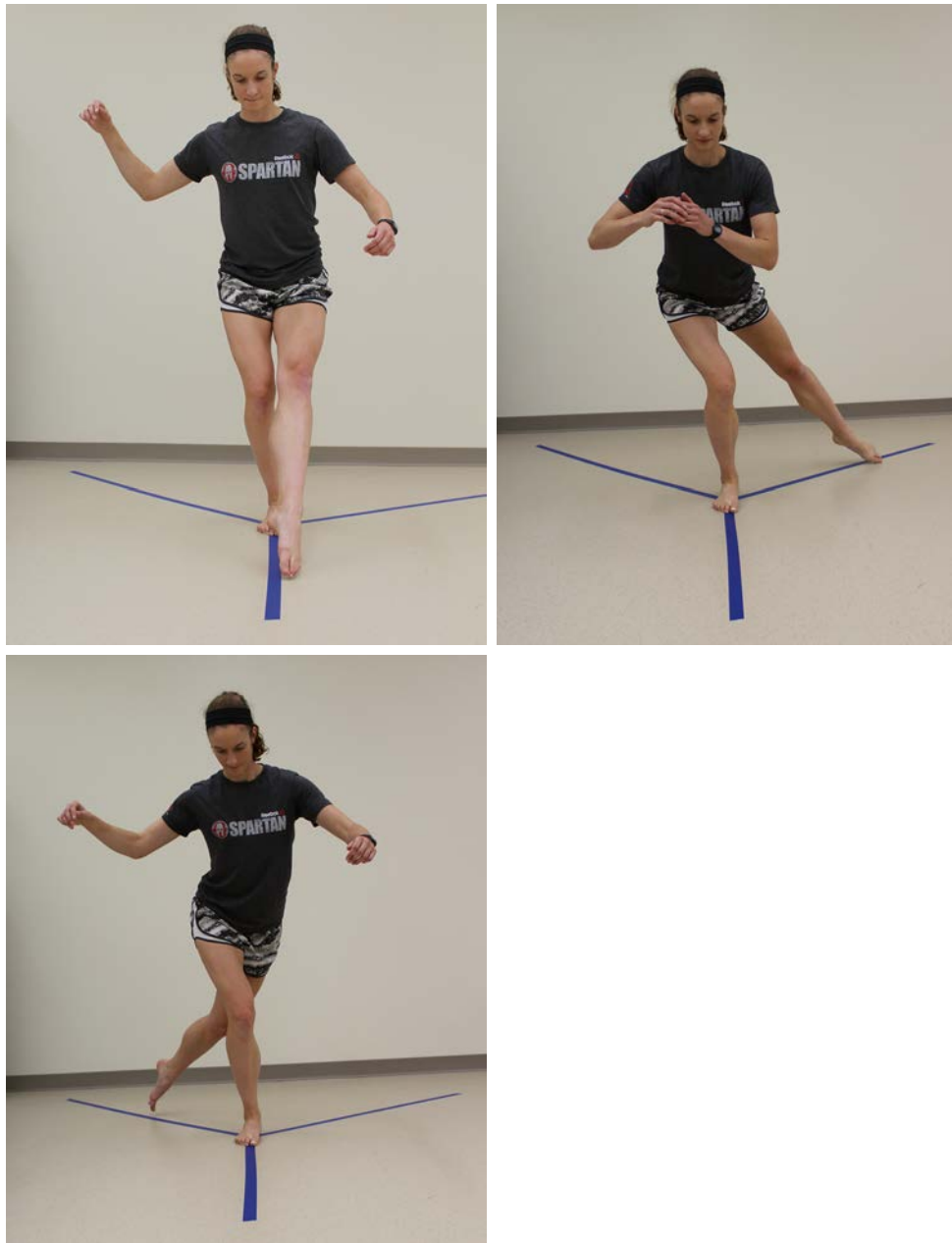


Fig. 14.4 The modified star excursion test (Y-balance test). The patient stands on the involved side and reaches as far forward, postero-laterai, or postero-medial as they can. The toe gently touches down and the patient returns to the upright single leg standing position. Distance reached is recorded.

progression of rehabilitation. Running can be slowly introduced and gradually increased using a run/walk ratio. An example walk/run rehabilitation protocol can be found in [Table 14.1](#). Rehabilitation protocols should be tailored to the individual needs of the athlete: for example, baseball players need to execute short sprints over straight or curved distances on uneven surfaces, whereas volleyball players need to jump, pivot, and cut quickly.

Return to Sport Testing

To assess an athletes' readiness to return to sport, several tests can be used to quantify function, including the Star Excursion Balance Test,^{22,25} shuttle run test,^{26,27} single leg hop for distance test,²⁸ triple hop test for distance,²⁸ and crossover hop test for

distance.²⁸ Poor performance on these tests has been associated with incomplete functional recovery and residual symptoms; however, these tests are not uniformly used in the clearance of athletes back to sport.

Authors' Preferred Treatment Approach for an Acute Ankle Injury

For patients presenting with an acute ankle sprain, we obtain a thorough history and perform a detailed physical examination. For less severe injuries and patients able to tolerate weight bearing, we initiate the above-mentioned protocol early in the rehabilitation to aid in patients' functional recovery. For more severe injuries requiring immobilization and protected weight bearing, we typically limit immobilization to approximately

TABLE 14.1 Run/Walk Rehabilitation Protocol

Run/Walk Ratio	Number of Cycles
15 seconds/45 seconds	4
30 seconds/60 seconds	4
1 minute/1-2 minutes	4 (building up to 4-10 minutes of total running)
2 minutes/1 minute	5 (building up to 10-24 minutes of total running)
3 minutes/1 minute	8 (24 minutes of total running)
4 minutes/1 minute	6 (24 minutes of total running)
6 minutes/1 minute	4 (24 minutes of total running)
8 minutes/1 minute	3 (24 minutes of total running)
12 minutes/1 minute	2 (24 minutes of total running)
15-20 minutes continuous running*	

*Increase 1-3 minutes per run until at goal distance

1 week. Patients requiring this are then reevaluated, and if there is no improvement in symptoms, we explore alternative diagnoses (i.e., high ankle sprain, lateral process of the talus fractures). If no other diagnosis is identified, immobilization will continue until the patient is able to tolerate weight bearing in a protective boot. For those patients that are able to enter into a rehabilitation protocol (either initially after the injury or after a period of immobilization and protected weight bearing), we reassess their rehabilitation progression at approximately 4 weeks from injury. If there has not been sufficient progression, we consider advanced imaging such as magnetic resonance imaging (MRI) to identify other pathology that may be limiting recovery (i.e., osteochondral lesions of the talus). A comprehensive list of potential diagnoses for the continually painful ankle is beyond the scope of this chapter.

CHRONIC ANKLE INSTABILITY

Approximately 40%–55% of patients with an ankle injury suffer from residual symptoms including persistent pain, recurrent

sprains, and repeated giving-way episodes.^{29,30} Further, 20% of patients continue to report symptoms of instability and pain at even 5 years following an acute ankle sprain.³¹ In general, symptoms are categorized as chronic if they persist beyond 6 months from index injury and have failed appropriate nonoperative management.³²

Like acute ankle sprains, a careful history is fundamental to an accurate diagnosis for those patients presenting with chronic symptoms. For patients presenting with long-standing complaints, it is important to carefully understand the chief complaint, mechanisms of injury (in particular those that cause continued instability), current level of activity and disability, and the presence or absence of mechanical symptoms. In addition, it is imperative to understand what treatments patients have previously received: specific note should be made to the duration of rehabilitation and the types of modalities and exercises employed during therapy. Studies indicate that, for many patients, chronic ankle instability is likely the result of poor initial rehabilitation following an acute injury.¹ Physical examination, again, should be systematic and detailed. Ligamentous laxity—as assessed by the anterior drawer and talar tilt tests—can be more readily identified in patients with chronic instability given the lack of ecchymosis and swelling seen in acute injuries. Furthermore, the exam should note deformities^{33,34} including hindfoot varus, plantar-flexion of the first ray, and midfoot cavus as well as those patients with generalized ligamentous laxity as calculated by the Beighton score (Fig. 14.5).³⁵ Weight-bearing radiographs should be obtained, with attention paid to alignment to assess for the presence of a radiographic cavovarus posture that may predispose to lateral ankle instability. MRI can be informative to identify alternative sources of ankle pain including chondral lesions, peri-articular tendon tears, degeneration, sinus tarsi injury, and impingement syndromes. Characteristic findings of chronic ankle instability on MRI include abnormal ligament thickness and ligament discontinuity, with wavy, attenuated ligaments being the most common appearance.³⁶

Traditionally, chronic ankle instability has been attributed to either mechanical or functional instability.³⁷ Functional

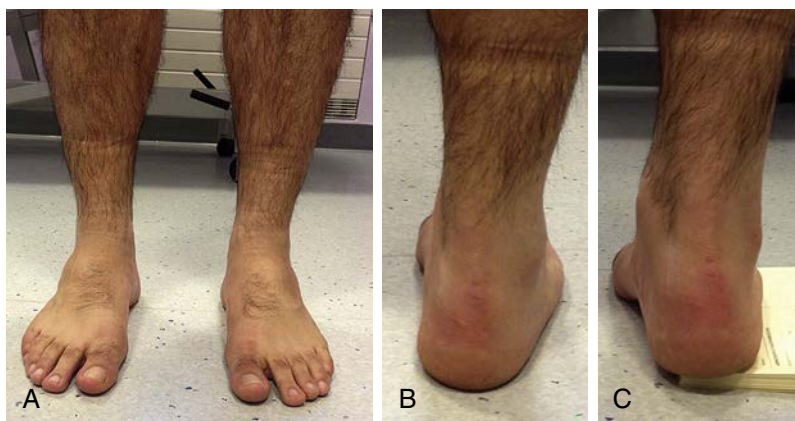


Fig. 14.5 Patient with bilateral subtle cavus deformity. (A) The peek-a-boo heels and the prominent extensor digitorum brevis muscle on the dorsolateral aspect of the foot seen on the frontal view (Δ). (B) varus posture of the heel on the rear view. (C) Correction of the varus on the Coleman block. (From Ali Abbasian, Gregory Pomeroy. The idiopathic cavus foot—not so subtle after all, *Foot and Ankle Clinics* 2013;18(4):629-642, Fig. 2.)

instability is viewed as the patient-reported sensation of instability that can be due to a variety of causes including impaired proprioception and sensation,³⁸ impaired neuromuscular firing patterns,^{39,40} or impaired postural control. Mechanical instability, conversely, may be the result of pathologic ligament laxity, impaired arthrokinematics, synovial inflammation, and degenerative changes.³⁷ While chronic instability, historically, has been viewed as a dichotomous process (mechanical versus functional), it is likely more a spectrum of disease with a subset of patients displaying features of both mechanical and functional instability. Tools like the Star Excursion Balance Test may aid in the assessment of patients with multidimensional instability.

In general, chronic ankle instability due to predominantly functional instability can be best managed initially through nonoperative measures.⁴¹ Patients with functional instability are more likely to benefit from a structured program of rehabilitation than patients with mechanical instability. Such conservative management includes similar principles to those outlined previously for acute ankle injuries; immobilization is typically not required, though. Evidence even suggests that through a structured functional rehabilitation program, 50% of patients with chronic ankle instability can achieve satisfactory functional stability.⁴²

As an adjunct to a rehabilitation protocol for ankle instability, many patients will benefit from the use of an orthotic.⁴³ While orthotics are frequently referred to as “arch supports,” an orthotic best suited to help a patient with chronic lateral ankle instability will have more lateral hindfoot support and less “arch support” so as to reduce varus force through the hindfoot and ankle. Ideally the orthotic will provide more of a pronation effect to the foot and resist excessive supination. There may need to be a cutout for the first metatarsal head to allow the pronation from the lateral heel wedge. This type of orthotic will be most helpful in a patient with underlying hindfoot varus. The orthotic should not be used as a stand-alone method of treatment for ankle instability but in combination with the established rehabilitation protocol.

Patients with chronic lateral ankle instability may also consider taping or bracing as an additional mode of treatment. Taping has been shown to improve proprioception in normal subjects with baseline decreased proprioception,¹⁴ and bracing has been shown to reduce the incidence of acute ankle injuries in high school football and basketball athletes.^{44,45} However, a meta-analysis by Raymond et al. suggested that in patients with chronic lateral ankle instability, taping and bracing provided no benefit to proprioception. While studies are not in complete agreement regarding the efficacy of taping or bracing, patients often like it as an adjunct to treatment as they progress through their rehabilitation protocol. There is often concern that reliance on taping or brace wear over time can weaken an ankle, leaving it more prone to injury. Corodova et al., however, suggested that the concern is not well founded when they demonstrated that consistent use of a brace did not change the latency to inversion of the peroneus longus.⁴⁶

As with use of an orthotic, taping and bracing should not be used as a stand-alone treatment for chronic lateral ankle instability. The National Athletic Trainers' Association do, though, recommend that athletes with a history of previous ankle

sprains wear prophylactic ankle support (tape or brace) for all practices and games.^{8,26,47,48} They also suggest that lace-up braces, semi-rigid ankle braces, and traditional ankle taping are all effective in reducing the rate of recurrent ankle sprains in athletes.^{8,47,49,50}

For those patients who fail nonsurgical management, operative interventions can then be considered. Consensus opinion on the definition of failure of nonsurgical management for chronic ankle instability is not clearly defined in the literature.

Authors' Preferred Treatment Approach for Chronic Ankle Instability

Patients who present with long-standing ankle instability require a detailed history, as discussed previously, to better understand what treatments they have undergone prior to presentation for care. Physical examination is fundamental to understand if there is evidence of mechanical instability versus functional instability (or a combination of both). Alignment evaluation is key to uncovering underlying deformity that may be exacerbating instability. Advanced imaging is most helpful in understanding associated pathologies. For those patients with predominantly functionally based instability without deformity or associated pathologies, we recommend initially a rehabilitation program as described above to target areas of deficit. If, after a reasonable course of therapy and consideration of other nonoperative adjuncts such as orthotics, taping, or bracing, patients continued to report ankle complaints, we then discuss surgery. For those chronic patients with predominantly mechanically based instability, we may be more inclined to discuss the role for surgery earlier in the treatment algorithm. While a trial of nonoperative treatment is a reasonable consideration, patients with significant mechanical instability should be educated on the potential for surgery. An informed discussion is key to these evaluations to devise a treatment plan that follows the patients' preferences.

Prevention

Given how common ankle sprains are, injury prevention continues to be a topic of great interest. Ankle injury prevention is of particular concern with the increase in sport specialization among adolescent athletes. Recent data from McGuine et al. revealed that high school athletes with moderate and high sport specialization were more likely to sustain a lower extremity injury than athletes with low sport specialization; ankle injuries were the most common injury in this cohort.⁵¹ Further, ongoing prospective trials are assessing the impact of preventive interventions on musculoskeletal injuries including ankle sprains.⁵² The role of ankle taping in managing ankle instability has long been a feature of treatment and injury prevention. While there is data to suggest that ankle-taping programs may restrict extremes in ROM and improve muscle reaction time,⁵³ there is mixed evidence regarding its efficacy in ankle injury prevention.⁵⁴ However, there is reasonable evidence to suggest that ankle bracing⁵⁵ and postural control exercises⁵⁶ can decrease ankle sprain recurrence. More recently, the use of mobile applications to aid in ankle injury prevention have been proposed,⁵⁷ but clearly further work is necessary to determine how to best reduce the burden of ankle injuries.


CONCLUSION

Acute lateral ankle injuries are common and best treated initially with an early rehabilitation program. A brief period of immobilization may be necessary in the initial treatment phase. This is followed by a regimented program of physical therapy geared toward efficient recovery and return to sport. For those patients with continued ankle instability and chronic symptoms, understanding how much of the instability is functional versus mechanical may help predict the likely effectiveness of further rehabilitation. However, this can be challenging, as some patients may present with features of both functional and mechanical instability. Fortunately, most patients improve nonoperatively after acute and chronic ankle sprains, and if they do not, there are surgical procedures that can be helpful for these patients.

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 **Video 14.1** <https://www.kollaborate.tv/link?id=5c9d1c7f4da0c> Title: Clinical and Radiographic Evaluation of Lateral Ankle Instability. Legend: Clinical evaluation of the right ankle lateral ligaments utilizing

the anterior drawer and talar tilt exam. Radiographic evaluation of chronic lateral ankle instability showing instability on anterior drawer and talar tilt test.

Ankle Sprains, Ankle Instability, and Syndesmosis Injuries

Thomas O. Clanton, Jess Mullens, Jonathan Backus, Norman Waldrop, III, Ana Robinson

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INTRODUCTION

Epidemiology

Despite attempts to reduce the incidence of ankle sprain injuries worldwide, ankle sprains continue to account for a large proportion of sport-related injuries as reported consistently in epidemiological studies.¹⁻⁶ In the US population in general, the incidence of ankle sprains was reported to be 2.15 per 1000 person-years with the peak incidence in ages 15–19 (7.2 per 1000 person-years). Almost 50% of those ankle sprains happened in athletic endeavors, with basketball, football, and soccer having the highest percentage of ankle sprain injuries among sports in the United States.⁶ Hootman et al. reported that ankle sprains accounted for 15% of all injuries in 15 sports analyzed using National Collegiate Athletic Association (NCAA) injury surveillance data from 1988–2004. They found ankle sprains to have an incidence of 0.83 per 1000 athlete exposures (AEs) compared with 0.15 per 1000 AEs for anterior cruciate ligament (ACL) injury and 0.28 per 1000 AEs for concussions.⁷ Thus, an ankle sprain is approximately five and a half and three times as common in occurrence as an ACL or concussion, respectively!

In an epidemiological study of ankle injuries in 255 countries, Fong et al. reported on 70 sports, with aeroball, wall climbing, and indoor volleyball having the highest prevalence of ankle injury, with ankle sprains accounting for over 50% of the ankle injuries in 30 of the 43 studies where injury type was defined.² A systematic review and meta-analysis of prospective epidemiological studies was reported by Doherty et al. and concluded that higher risk for ankle sprain occurred in (1) females over males, (2) children over adolescents and adults, (3) court sports and indoor sports, and (4) lateral sprains over syndesmosis and medial sprains.¹

Combination Injuries

Ankle sprains include those to the lateral ligament complex, the deltoid complex, and the syndesmosis. Sprains of the lateral ankle ligament complex generally comprise up to 85% of all sprains to the ankle.⁸ It is important to realize that while this chapter is separated for discussion of these specific areas of injury, combination injuries occur and include multiple sprains, as well as other injuries including fractures, contusions, strains, tendon tears, cartilage injury, and tendon dislocations. Clearly, this requires a thorough evaluation of each patient to understand the mechanism of injury, the severity and nature of the symptoms, and any history of ankle problems. Coupling this with a detailed physical examination and appropriate imaging allows determination of the best treatment for the individual patient. Postinjury follow-up is also important, since persistent symptoms can exist in 32%–74% of individuals with a history of lateral ankle sprain, and these symptoms may continue beyond 10 years.^{9,10} A checklist of potential sources of continuing symptoms is valuable (Table 15.1).

LATERAL ANKLE SPRAINS

Acute Lateral Ankle Sprain

Assessment

Approximately 70% of all lateral ankle sprains involve isolated anterior talofibular ligament (ATFL) injury.² The ATFL is the weakest of the lateral ligament structures and is put under increased stress in the most common mechanism of injury, which is foot supination with heel inversion while the ankle is plantarflexed. Pain and swelling are present followed by bruising, loss of motion, and difficulty walking in more serious cases. The anterior drawer and talar tilt tests signify instability when positive, and this may vary from mild to severe. A classification of lateral ankle sprains related to the

severity of injury and magnitude of the above findings has been published and validated for time to recovery (Table 15.2).

Acute lateral ankle sprains are typically treated nonoperatively (see Chapter 14) in almost all circumstances.¹¹ Exceptions to this would include open injuries, large avulsion fractures, or other associated pathology such as dislocating peroneal tendons, an osteochondral fracture or loose body, or a bimalleolar fracture equivalent, which would essentially be a dislocated ankle that tears both medial and lateral ligaments. Among the remaining controversies related to acute lateral ankle sprains is whether to operate when faced with an athlete who clearly has a severe injury with major instability. Varying opinions can still be heard on this topic.^{8,12–14} Another situation that stimulates discussion over whether to operate is the athlete who has a history of significant prior ankle sprains and then presents with a recurrent severe sprain. This is the situation that requires a thorough discussion with the patient to determine the best course of action, and extenuating circumstances often play a role. In other words, is this the correct time to repair what is clearly chronic lateral ankle instability?

TABLE 15.1 Sources of Chronic Pain or Instability After Ankle Sprain

Articular injury	Impingement
Chondral fractures	Anterior tibial osteophyte
Osteochondral fractures	Anterior inferior tibiofibular ligament
Nerve injury	Miscellaneous conditions
Superficial peroneal	Failure to regain normal motion (tight Achilles)
Posterior tibial	Proprioceptive deficits
Sural	Tarsal coalition
Tendon injury	Meniscoid lesions
Peroneal tendon (tear or dislocation)	Accessory soleus muscle
Posterior tibial tendon	Unrelated ongoing pathology masked by routine sprain
Other ligamentous injury	Unsuspected rheumatologic condition
Syndesmosis	Occult tumor
Subtalar	Chronic ligamentous laxity (collagen disease)
Bifurcate	Neuromuscular disease (Charcot-Marie-Tooth disease)
Calcaneocuboid	Neurologic disorders (L ₅ radiculopathy, poststroke)

Nonoperative Treatment (see Chapter 14)

When nonoperative treatment is the chosen course, a comprehensive plan needs to be drafted that includes evidence-based interventions. This includes functional rehabilitation with a supportive ankle brace in Grade I and II injuries, while Grade III injuries are more controversial regarding functional treatment versus a short period of immobilization in a walking boot or short-leg cast for 10–14 days.^{15–17} Additional treatment includes rest, ice, compression, and elevation method (RICE), antiinflammatory medication (if there are no contraindications), physical therapy with supervised early exercise when possible, and crutches, but only when weight bearing is not possible. In this latter situation, it is important to immobilize the ankle in neutral to slight dorsiflexion, or the ankle will naturally assume a plantarflexed position and heal with the ligaments in an elongated state.¹⁸ Improvement in ankle dorsiflexion, increased strength in the kinetic chain, manual therapy techniques, and balance training have all been shown to have evidence-based gains in ankle function and pain.^{16,19} Other treatments with less evidence include laser therapy, dry needling, vascular restriction, electrotherapy, vascular restriction, diathermy, and ultrasound.¹⁶ The majority of patients treated with evidence-supported methods improve relatively quickly. According to the classification of acute lateral ankle sprains as Grade I, II, IIIA, or IIIB, full recovery averages 8, 16, 25, and 39 days, respectively.²⁰ However, when this is not the case, further evaluation is warranted, particularly in the high-level athlete where time loss is critical.

Operative Treatment

In the situation where surgical repair of the torn ligaments is necessary, it is important to define the tissues and repair them anatomically whenever possible (Figs. 15.1 and 15.2).²¹ In the acute situation, the injured tissues typically become immediately evident on opening the skin incision. It is important to assess all local structures including the articular cartilage of the ankle joint, the anterior capsule, the deltoid ligament, the peroneal tendons, and superior peroneal retinaculum. At the same time, the surgeon should be protective of the superficial nerves.

In severe sprains, which are essentially dislocations of the ankle joint, one or more of these structures may be injured and need repair. Ligaments torn in midsubstance are repaired with sutures, while tears off bone or with a bony avulsion are repaired with suture anchors. Newer methods of augmentation of repairs have been introduced and proven to be effective.^{22,23} In cases of acute on chronic tears, tissue quality may be lacking and repair with augmentation may be more critical to obtain

TABLE 15.2 Classification System of Lateral Ankle Sprains

Grade	Clinical Tests	Decreased ROM	Edema	Stress Radiographs	Return to Sport (days)
I		<5°	<0.5 cm	Normal	7.24 ± 1.63
II	+ anterior drawer	5–10°	0.5–2 cm	Normal	14.95 ± 2.1
IIIA	+ anterior drawer & talar tilt	>10°	>2 cm	Normal	30.65 ± 3.07
IIIB	+ anterior drawer & talar tilt	>10°	>2 cm	Joint laxity >3 mm	55.41 ± 4.92

ROM, Range of motion.

Modified from Malliaropoulos N, Papacostas E, Papalada A, Maffulli N. Acute lateral ankle sprains in track and field athletes: an expanded classification. *Foot Ankle Clin.* 2006;11(3):497-507, Tables 3 and 4.

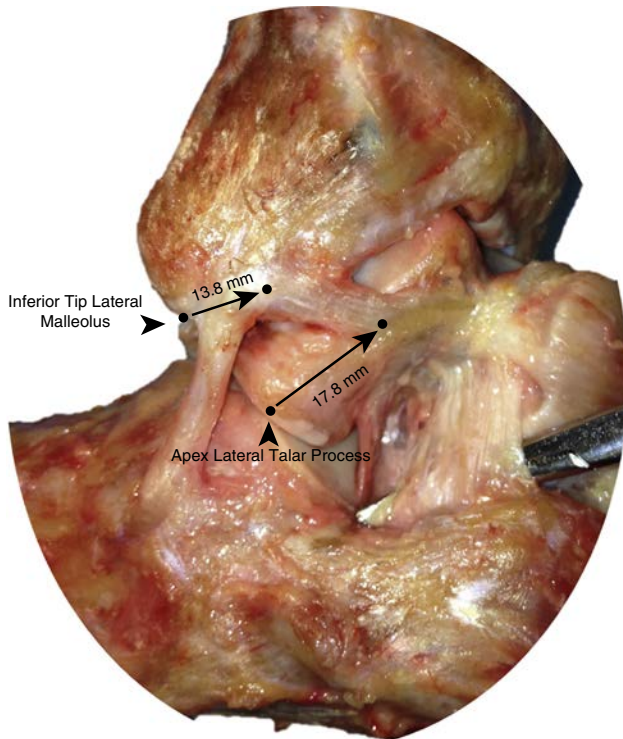


Fig. 15.1 Position of Single Band Anterior Talofibular Ligament. Origin located at average distance from inferior tip of the lateral malleolus of 13.8 mm (50% of the distance from the inferior tip to the fibular anterior tubercle [anterior-most point of the fibula near the tibial plafond]). Insertion is located at average distance of 17.8 mm from apex of the lateral talar process (61% of the distance from the apex of the lateral talar process to the anterolateral corner of the talar trochlea). (From Clanton TO, Campbell KJ, Wilson KJ, et al. Qualitative and quantitative anatomic investigation of the lateral ankle ligaments for surgical reconstruction procedures. *J Bone Joint Surg Am.* 2014 Jun 18;96(12):e98, with permission "Courtesy of Thomas O. Clanton, MD")

adequate stability versus reconstruction with a tendon autograft or allograft or a tendon transfer (see next section).^{24–28} The rehabilitation program in these cases is very important and is outlined in the section on rehabilitation (Boxes 15.1 to 15.3 and Tables 15.3 to 15.5).

Chronic Lateral Ankle Instability

Assessment

Most patients with chronic lateral ankle sprains and instability present with either recurrent ankle sprains or with the feeling of looseness in the ankle and the sensation of “giving way.” Patients may complain of ankle pain, but it is not a requirement for this diagnosis. The examination typically confirms the presence of a positive anterior drawer test and/or a positive inversion stress test. Tenderness may be present, but often is more indicative of associated pathology, such as synovitis, an osteochondral injury, or tendonitis. The examiner must be thorough enough to rule out other sources of symptoms (Table 15.1), because the clinical diagnosis of chronic lateral ankle instability has been associated with the intraoperative findings of peroneal tendon pathology (tenosynovitis, tears, dislocation), anterolateral impingement lesions, ankle synovitis, intra-articular loose bodies, talar osteochondral lesions, and medial ankle tenosynovitis.²⁹ A comprehensive physical therapy program should be initiated first. Symptoms often will resolve

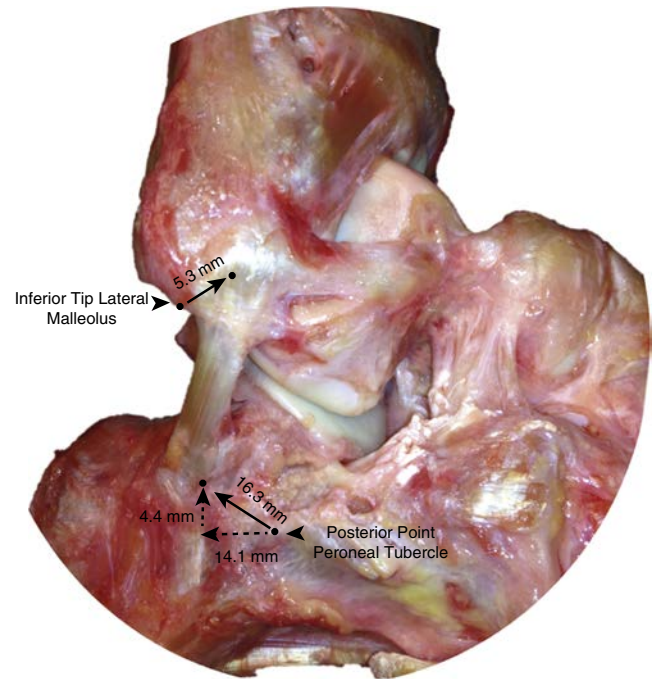


Fig. 15.2 Position of the Calcaneofibular Ligament. Origin located at an average of 5.3 mm from the inferior tip of the lateral malleolus and insertion located 14.1 mm posterior to the posterior point of the peroneal tubercle of the calcaneus and 4.4 mm superior. (From Clanton TO, Campbell KJ, Wilson KJ, et al. Qualitative and quantitative anatomic investigation of the lateral ankle ligaments for surgical reconstruction procedures. *J Bone Joint Surg Am.* 2014 Jun 18;96(12):e98, with permission "Courtesy of Thomas O. Clanton, MD")

BOX 15.1 Criteria for Progression—Phase I—Range of Motion

Clinical Finding or Test

1. Use objective outcome measures throughout recovery. Recommend FAAM, FAOS, SANE, VAS scores.
2. Little to no edema, recommend Figure of Eight method, ICC = 0.99 (Tatros-Adams)
3. Symmetrical AROM and PROM
4. Weight Bearing Lunge Test, achieve at least 50% as compared with uninvolved side. Measure distance of foot from wall in cm and tibial angle with inclinometer. ICC = 0.96 to 0.99 (Konor et al.).
5. Demonstrate proper open chain muscle recruitment for all lower extremity musculature through full available range of motion. Patient can resist increasingly difficult resistance bands × 15 reps through full ROM without pain.
6. Demonstrate ability to perform short arch exercise in seated position, and intrinsic flexion exercises (McKeon et al.)
7. Patient can ambulate with safe and appropriate gait pattern in boot without crutches.

AROM, active range of motion; FAAM, Foot and Ankle Ability Measure; FAOS, Foot and Ankle Outcome Score; ICC, intraclass correlation coefficient; PROM, patient reported outcome measure; SANE, single assessment numeric evaluation; VAS, visual analog scale (for pain). Tatros-Adams D, McGann SF, Carbone W. Reliability of the figure-of-8 method of ankle measurement. *J Orthop Sports Phys Ther.* 1995;22(4):161–163.
Konor MM, Morton S, Eckerson JM, Grindstaff TL. Reliability of three measures of ankle dorsiflexion range of motion. *Int J Sports Phys Ther.* 2012;7(3):279–287.
McKeon PO, Hertel J, Bramble D, Davis I. The foot core system: a new paradigm for understanding intrinsic foot. *Br J Sports Med.* 2015;49(5):290.

with correction of the deficits in proprioception, strength, and flexibility. This is specifically the case in patients with functional instability.^{30,31} Regardless, preoperative therapy can improve the

BOX 15.2 Criteria for Progression—Phase II—Endurance

Clinical Finding or Test

1. Objective outcome measures score reassessed.
2. Normal Gait pattern demonstrated at varied cadence on flat surface.
3. Foot Lift Test, demonstrate <5 errors during testing period (Linens et al.)
4. Weight-Bearing Lunge Test, within 75% of uninvolved leg.
5. Standing Double-Leg Heel Raise, can demonstrate equal heel height.
6. Double-Leg Squat, demonstrate proper technique.
7. Isotonic Single-Leg Leg Press: Achieve 50–60% of body weight × 15 reps
8. Demonstrate ability to perform seated towel curls with added weight, pulling weight equal to capability of uninvolved side

Linens S, Ross S, Arnold B, Gayle R, Pidcoe P. Postural-Stability tests that identify individuals with chronic ankle instability. *J Athl Train*. 2014;49(1):15–23. <https://doi.org/10.4085/1062-6050-48.6.09>.

BOX 15.3 Return-to-Sport Criteria

1. Criteria for progression met. If any goals are not achieved, this is reviewed and discussed with rehabilitation team and physician.
2. Subjective outcome measure reassessed; progress suggests return-to-sport readiness. SANE score >93%.
3. Athlete reports he/she has resumed preinjury training volume.

Return to practice noncontact × 1 week

Return to practice contact × 1 week

Return to game play, progressively working up to preinjury position time of play.

SANE, single assessment numeric evaluation.

results in patients who ultimately require surgery. Nonoperative treatment also includes activity and/or shoe modification (e.g., lateral heel wedge), an ankle-foot orthosis, and/or orthotic devices incorporating a lateral heel wedge. Broström found that symptoms of instability remained in 20% of his patients who were treated in a conservative fashion, and this has been a consistent finding.^{18,32,33} Athletes may use a nonoperative approach to get through a season but rarely consider this an acceptable long-term solution unless their symptoms are minimal.

Indications for surgical treatment include instability symptoms and signs found in young to middle-aged, active individuals who have not responded to a well-designed, nonoperative treatment program. Radiographic criteria for surgical consideration include an anterior drawer greater than 1 cm (or a side-to-side difference of >3 mm), and a talar tilt greater than 15 degrees (or a side-to-side difference of >10 degrees) as guidelines, but in general, the symptoms and signs are most critical.³⁴ An in-office mini C-arm is a convenient tool to confirm radiographic instability. Contraindications to surgery include other causes of instability (collagen diseases, tarsal coalitions, neuromuscular diseases, neurologic disorders, or functional instability), older patients with sedentary lifestyles, patients with serious medical conditions that would preclude anesthesia and major surgery, circulatory impairment, presence of ongoing infection, lateral ankle pain without documented lateral instability, history of complex regional pain syndrome, or degenerative arthritis. A relative contraindication is failure of the patient to participate in a preoperative rehabilitation program.

Ankle arthroscopy is warranted before ankle stabilization. Chondral injury is the most common problem discovered at arthroscopy, with almost 30% of acute ankle injuries and 95% of chronic ankles having this lesion in one study of an athletic

TABLE 15.3 Overview of Rehabilitation Orders: Ankle Stabilization Procedures

Procedure(s)	Broström Repair	Broström Repair + Internal Brace	ATFL and CFL Reconstruction With Allograft	Syndesmosis Repair of AITFL and Tight Rope	Syndesmosis Repair, Deltoid Repair and Fibular ORIF (no cartilage injury)
Range-of-Motion Precautions	No inversion or plantarflexion × 6 weeks	<ul style="list-style-type: none"> • Full active, pain free range of motion • No passive inversion × 6 weeks 	<ul style="list-style-type: none"> • Full active, pain-free range of motion • No passive inversion × 6 weeks 	None	No eversion × 6 weeks
Weight-Bearing Orders	<ul style="list-style-type: none"> • Splint × 7–10 days • When out of splint, PWB → FWB over 4-week period • Walk in boot without crutches, pain, or swelling for 5–7 days, then: • Wean out of boot over 2-week period 	<ul style="list-style-type: none"> • Splint × 7–10 days • When out of splint, PWB → FWB over 2-week period • Walk in boot without crutches, no pain or swelling for 5–7 days, then: • Wean out of boot as tolerated 			<ul style="list-style-type: none"> • Splint × 7–10 days • NWB × 4 weeks unless specified by surgeon • After 4 weeks, PWB to FWB over 2–4 week period as tolerated • Once FWB in boot × 5–7 days without crutches, no pain or swelling then: • Wean out of boot × 2 weeks
Return to Sport	<ul style="list-style-type: none"> • Must pass all criteria for progression (Boxes 15.1-15.3 and Tables 15.4-15.5) • Outcomes measures match clinical findings • MD clearance for noncontact practice granted (Table 15.3) 				

AITFL, Anterior inferior tibiofibular ligament; ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; FWB, full weight bearing; NWB, Non-weight bearing; PWB, partial weight bearing; ORIF, open reduction internal fixation.

TABLE 15.4 Criteria for Progression—Phase III—Strength

Clinical Finding or Test

Subjective outcome measure reassessed.

Patient can demonstrate ability to maintain proper arch posture in single leg tasks.

Test	Passing Score	Notes
Weight Bearing Lunge Test	Symmetry to uninvolved side, within 5* or 1.5 cm	ICC = 0.96 to 0.99 (Konor et al.)
Heel Raise Endurance Test	Within 90% of uninvolved side, recorded in successful repetitions	ICC = 0.97 (Sman et al.)
Single Leg Squat Endurance Test	Within 90% of uninvolved side, recorded in repetitions	ICC = 0.95–1.0 (Garrison et al.)
Y Balance Test	Composite score above 90% Anterior reach within 4 cm of uninvolved side	ICC = 0.85–0.93 (Shaffer et al.)
Isotonic Leg Press	Within 90% of uninvolved 3 × 5 reps	

ICC, intraclass correlation coefficient.

Konor MM, Morton S, Eckerson JM, Grindstaff TL. Reliability of three measures of ankle dorsiflexion range of motion. *Int J Sports Phys Ther*. 2012;7(3):279–287.Sman AD, Hiller CE, Imer A, Ocsing A, Burns J, Refshauge KM. Design and reliability of a novel heel rise test measuring device for plantarflexion endurance. *Biomed Res Int*. 2014;2014:391646. <https://doi.org/10.1155/2014/391646>.Garrison JC, Shanley E, Thigpen C, Geary R, Osler M, DelGiorno J. The reliability of the vail sport test™ as a measure of physical performance following anterior cruciate ligament reconstruction. *Int J Sports Phys Ther*. 2012;7(1):20–30.Shaffer SW, Teyhen DS, Lorenson CL, et al. Y-balance test: a reliability study involving multiple raters. *Mil Med*. 2013;178(11):1264–1270.

TABLE 15.5 Criteria for Progression—Phase IV—Power and Agility

Clinical Finding or Test

Pick Appropriate Test(s) from each category related to patient's sport. Not all tests need to be performed.

Subjective outcome measure reassessed. Progress suggests return to sport readiness. SANE score >93%.

Test	Passing Score	Notes
Single hop for distance	Within 90% of uninvolved side	ICC = 0.92–0.97 (Ross et al.)
Triple hop for distance		ICC = 0.84–0.98 (Kockum et al.)
Square Hop	Within 90% of uninvolved side, measured in seconds	Square Hop: ICC = 0.90
Figure of 8 Hop		SEM 1.40 sec MDC 3.88 sec
Side Hop		Side Hop: ICC = 0.84 SEM 2.10 sec MDC 5.82 sec
Cross-Over Hop		6 Meter Cross Over Hop: ICC = 0.6
		SEM 0.37 sec MDC 1.03 sec (Caffrey et al.)
MAT Test	Within 90% of uninvolved side, measured in seconds	ICC = 0.825 (Hickey et al.)
Modified T Test		

ICC, intraclass correlation coefficient; SANE, single assessment numeric evaluation; SEM, standard error of the mean; MDC, minimal detectable change; MAT, modified agility T Test.

Ross MD, Langford B, Whelan PJ. Test-retest reliability of 4 single-leg horizontal hop tests. *J Strength Cond Res*. 2002;16(4):617–622.Sman AD, Hiller CE, Imer A, Ocsing A, Burns J, Refshauge KM. Design and reliability of a novel heel rise test measuring device for plantarflexion endurance. "https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4022004/". *Biomed Res Int*. 2014;2014:391646. <https://doi.org/10.1155/2014/391646>.Caffrey E, Docherty CL, Schrader J, Klossner J. The ability of 4 single-limb hopping tests to detect functional performance deficits in individuals with functional ankle instability. *J Orthop Sports Phys Ther*. 2009;11:799–806.Hickey KC, Quatman CE, Myer GD, Ford KE, Brosky JA, Hewett TE. Methodological report: dynamic field tests in an NFL combine setting to identify lower extremity functional asymmetries. *J Strength Cond Res*. 2009;23(9):2500–2506.

population.³⁵ The value of ankle arthroscopy in discovering and treating chondral injury, loose bodies, osteophytes, and soft-tissue impingement has been confirmed in several studies.^{36–39} Not only is this important for recognizing and treating additional pathology, but it is also helpful in discussing prognosis with the patient following surgery.

Operative Treatment

Similar to the surgical history of shoulder and knee instability, more anatomic reconstructions have gained popularity for ankle instability. This began with secondary repair of the previously injured anterior talofibular ligament by Lennart Broström

in 1966.⁴⁰ It has taken almost five decades for the accumulation of scientific evidence to cast doubt on the tenodesis procedures described by Evans, Watson-Jones, Larsen, and Chrisman and Snook.^{41–45} The following discussion focuses on the anatomic procedures, whether by direct repair in the tradition of Broström or by using tissue transfer or tissue grafts placed anatomically.

Broström described his anatomic repair as a delayed procedure for chronic lateral ankle instability. The procedure is a straightforward division and imbrication of the anterior talofibular ligament. The calcaneofibular ligament was not addressed in his original description, and others have since indicated that ATFL secondary repair alone is adequate.^{46,47} Various modifications have been



Fig. 15.3 Stretched anterior lateral ligaments found in typical chronic ankle sprains. (Courtesy Matthew Morrey, MD)



Fig. 15.4 Imbrication of stretched ligaments in anatomical reconstructive procedure. (Courtesy Matthew Morrey, MD)

described, the most popular being a reinsertion into a bony trough, imbrication of the calcaneofibular ligament, reinforcement with the inferior extensor retinaculum, and, more recently, the use of suture tape augmentation.^{25,48,49} Other authors have described the use of different graft sources to rebuild the lateral ankle ligaments while emphasizing the anatomic placement of bone tunnels. Graft sources have included the plantaris tendon, the split peroneus brevis tendon, hamstring tendons, and allograft tendons.

Our preferred method when adequate tissue is available is the Broström-Gould procedure repairing the ATFL with sutures or suture anchors (Figs. 15.3 and 15.4) followed by placement of an Internal Brace that acts as a checkrein to protect the healing



Fig. 15.5 Drawing depicting the position of an InternalBrace over the top of an anterior talofibular ligament secondary repair with suture anchors in the fibula. (From Arthrex Inc. with permission)

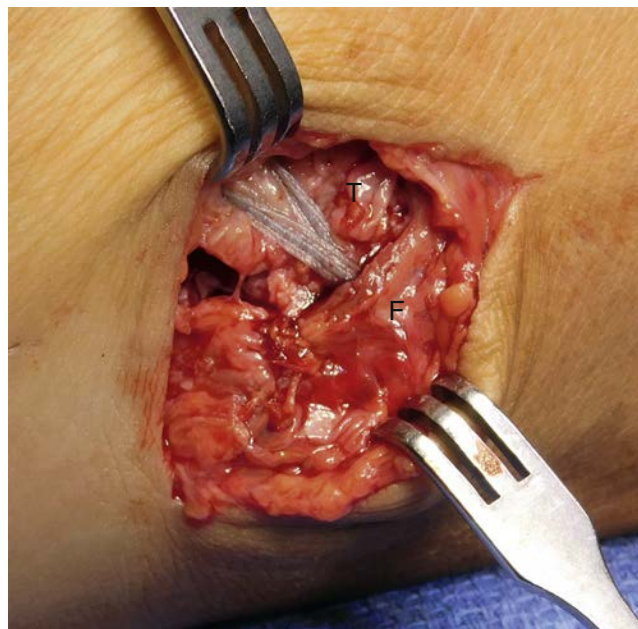


Fig. 15.6 Intraoperative picture of an InternalBrace (IB) anchored into talus (T) and fibula (F) over the top of a repaired anterior talofibular ligament. The IB acts as a checkrein over the repaired ligament and not as an artificial ligament. (Courtesy Thomas O. Clanton, MD)

tissue (Figs. 15.5 and 15.6) The calcaneofibular ligament (CFL) is also imbricated when it is loose. This is performed most frequently by reattaching the CFL to the distal fibula 5 mm anterior to the inferior tip of the fibula with a suture anchor. The CFL can often be intact visually as well as on magnetic resonance imaging (MRI) but on direct inspection can be confirmed to be stretched or attached to periosteum of the distal fibula that has become disengaged from the distal fibula. The authors feel that including secondary repair of the CFL ensures that talar tilt ankle stability will be restored along with subtalar stability when that pattern of instability is present. Tightening the inferior extensor retinaculum adds an extra degree of stability to the repair (Fig. 15.7).^{50,51}

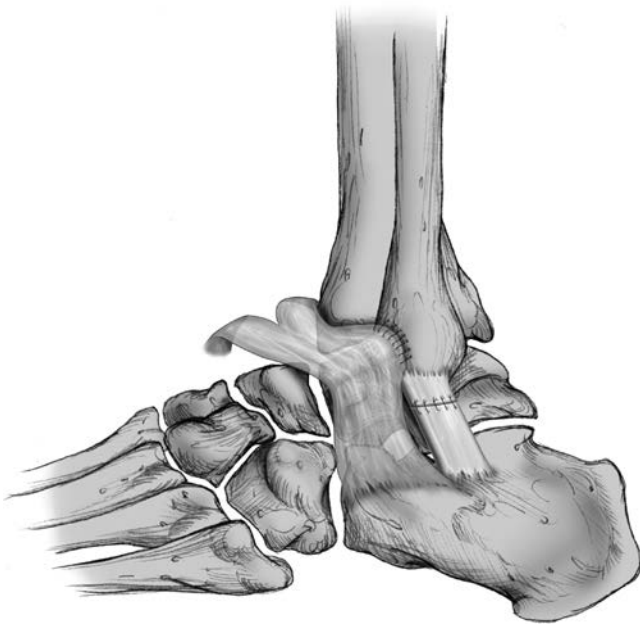


Fig. 15.7 Imbrication of inferior extensor retinaculum to periosteum of distal fibula. (Courtesy Matthew Morrey, MD)



Fig. 15.8 Drawing depicting the position of the tendon allograft passing through tunnels in the talus, fibula (2 fibular bone tunnels denoted by dashed lines), and calcaneus with fixation using bioabsorbable interference fit screws. (From Arthrex Inc. with permission)

In the circumstance where no adequate tissue is available for repair, a tendon graft (autograft or allograft) or tendon transfer can be effective (Figs. 15.8 and 15.9).^{24,26,28}

Failed Prior Surgery

The results of surgical treatment of chronic lateral ankle instability are generally above 90% regardless of the method. However, there are isolated cases where the surgery fails either due to reinjury or mechanical breakdown of the previous fixation. Nonanatomic reconstructions will typically stretch out over time or will over-constrain the joint, leading to stiffness, which may be unacceptable to the patient. These are the situations most suited to reconstruction with a tendon graft or a tendon transfer, and the authors prefer the former. The graft is placed into an anatomically oriented tunnel(s) in the fibula and fixed with an interference fit screw(s) or suture button (Figs. 15.10 and 15.11). Since previous hardware or bone tunnels may

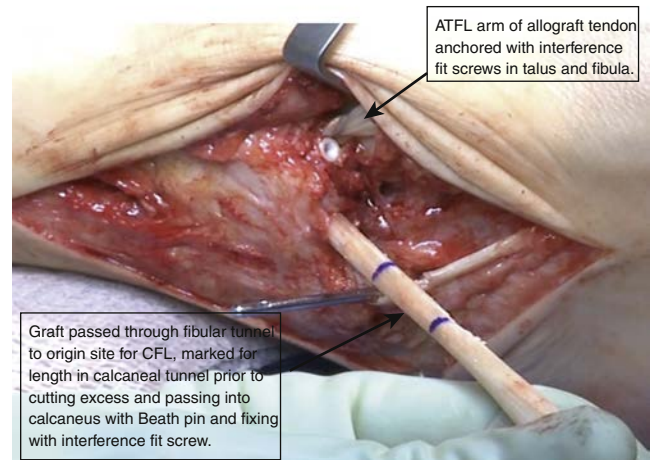


Fig. 15.9 Intraoperative picture showing the graft in the talar and fibular tunnels prior to suturing with Krakow stitch, cutting excess tendon length, and passing graft into calcaneal tunnel and fixing with interference screw. (Courtesy Thomas O. Clanton, MD)



Fig. 15.10 Anterior-posterior x-ray of suture-button used to fix revision allograft into fibula of small patient with prior secondary repair with bone anchors. (Courtesy Thomas O. Clanton, MD)

have been used, it is critical to evaluate their location and size to determine the best method to address placement and fixation of the graft or tendon transfer. Understanding anatomical landmarks from a radiographic standpoint can be helpful, along with computed tomography (CT) imaging.⁵²

MEDIAL ANKLE SPRAINS

Acute Medial Ankle Sprain

The deltoid ligament is injured in up to 15% of all ankle sprains.^{53,54,55} Injuries to the medial ankle ligamentous complex rarely occur in isolation. Broström's original study reported on 105 ankle sprains and showed only three cases of isolated medial ankle sprains.⁵⁶ The majority of deltoid ligament injuries occur in



Fig. 15.11 Lateral x-ray of endobutton used to fix revision allograft into fibula of small patient with prior secondary repair with bone anchors. (Courtesy Thomas O. Clanton, MD)

association with lateral malleolus fractures, syndesmosis injuries, injuries to the lateral ligament complex, and/or damage to the articular cartilage surfaces of the tibiotalar joint.⁵⁷ In a study of 1206 West Point cadets, isolated medial ankle sprains accounted for 5.1% of injuries. Males had a three times greater risk of medial ankle sprains than females.⁵⁵ Diagnosis of deltoid ligament tears in complex ankle injuries have increased due to use of arthroscopy and advanced imaging studies. These injuries can be missed with history and physical examination alone.^{55,58,59} A study of NCAA collegiate athletes reported 380 deltoid ligament injuries over a 6-year period.⁶⁰ In their study, men's football and men's and women's soccer had the highest rates of deltoid ligament sprains. Player contact was the most common mechanism of injury, occurring in nearly 50% of athletes.

Assessment

As stated previously, concurrent injuries are common with injuries to the deltoid ligament. The history and mechanism of injury can lead the clinician to an accurate differential diagnosis, but an examination of the entire leg is essential to rule out associated injuries. The entire length of the fibula should be palpated to rule out a high fibula fracture or proximal tibiofibular injury as seen in a Maisonneuve-type injury. A squeeze or external rotation test can be performed to help rule out an acute syndesmosis injury.

The lateral ligaments should each be examined carefully with an anterior drawer and talar tilt test, keeping in mind that anterior instability relies on the secondary restraints of the deltoid ligament. A thorough understanding of the anatomical location of the medial ankle structures is necessary to locate the point of maximum tenderness (Figs. 15.12 to 15.15).⁶¹ Individual palpation, range of motion, and excursion of the posterior tibial, flexor digitorum longus, and flexor hallucis longus tendons should be performed along with assessment of the spring ligament. A standing examination with assessment of the longitudinal arch

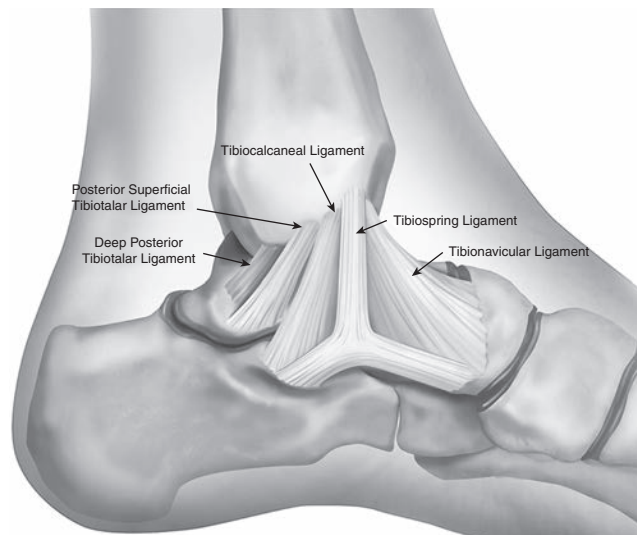


Fig. 15.12 Drawing of medial left ankle depicting the superficial components of the deltoid ligament. (From Campbell KJ, Michalski MP, Wilson KJ, et al. The ligament anatomy of the deltoid complex of the ankle: a qualitative and quantitative anatomical study. *J Bone Jt Surg.* 2014;96(8):e62-e62.)



Fig. 15.13 Photograph of cadaveric dissection depicting the superficial components of the deltoid ligament. (From Campbell KJ, Michalski MP, Wilson KJ, et al. The ligament anatomy of the deltoid complex of the ankle: a qualitative and quantitative anatomical study. *J Bone Jt Surg.* 2014;96(8):e62-e62.)

can reveal an increasingly flat foot or pronated foot deformity that is actively correctable with contraction of the posterior tibial muscle. A sensory examination, with a Tinel's test, can identify traction injuries to the saphenous or posterior tibial nerve.

Radiographs are important in the evaluation of an injury to the deltoid ligament, especially in the setting of a fibular fracture and/or a syndesmotic injury. A small avulsion fracture of the tip of the medial malleolus can be seen on standard anterior-posterior (AP), oblique, and/or lateral radiographs (Figs. 15.16 and 15.17). If the history and physical examination are suggestive of a more proximal injury to the lower leg, supplemental radiographic views of the leg should be obtained to rule out any pathology proximal to the ankle.

While a weight-bearing AP ankle radiograph can reveal valgus tilt of the talus in a complete deltoid rupture, manual



Fig. 15.14 Drawing of medial left ankle depicting the deep components of the deltoid ligament. (From Campbell KJ, Michalski MP, Wilson KJ, et al. The ligament anatomy of the deltoid complex of the ankle: a qualitative and quantitative anatomical study. *J Bone Jt Surg.* 2014;96(8):e62-e62.)

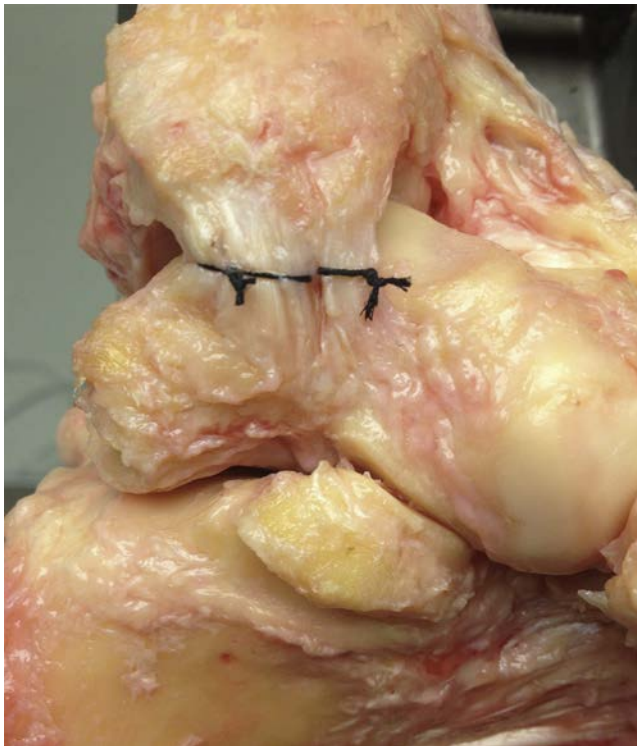


Fig. 15.15 Photograph of cadaveric dissection depicting the deep components of the deltoid ligament. (From Campbell KJ, Michalski MP, Wilson KJ, et al. The ligament anatomy of the deltoid complex of the ankle: a qualitative and quantitative anatomical study. *J Bone Jt Surg.* 2014;96(8):e62)

stress radiographs remain the gold standard for evaluating medial ankle instability especially if an incomplete injury is present. The stress can be applied as manual external rotation stress or as a gravity stress. If the athlete has significant pain and guarding with external rotation stress, the gravity stress is useful, though the validity of the test has not been confirmed

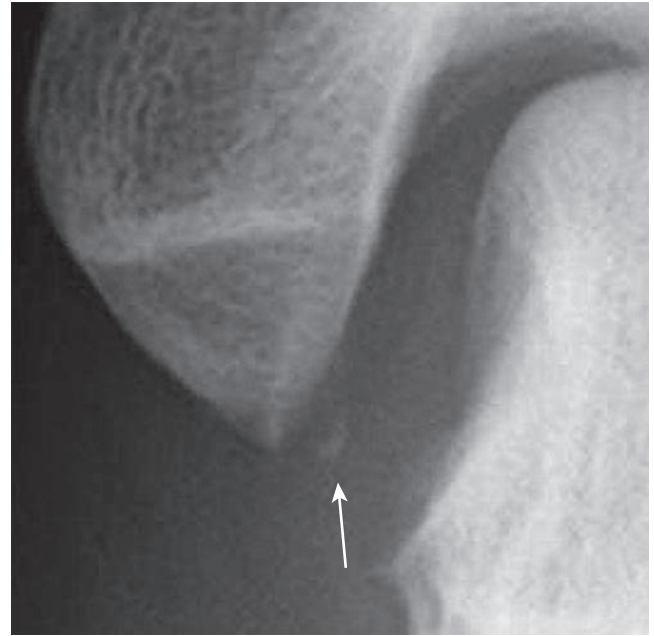


Fig. 15.16 Radiographic example of small avulsion of deltoid ligament with widened medial clear space. Arrow denotes the small bony avulsion. (Courtesy Thomas O. Clanton, MD)



Fig. 15.17 Radiograph of larger medial malleolar avulsion injury in elite snowboarder. (Courtesy Thomas O. Clanton, MD)

in patients without an associated fibular fracture.⁶² The exact amount of medial joint space widening on stress radiographs is a controversial topic, but most authors accept that greater than 4 mm (or 1 mm greater than the superior tibiotalar joint space) indicates an injury to the deltoid ligament.^{63,64}

MRI has become more widely used for diagnosis of deltoid ligament and associated soft-tissue injury. In the athlete with signs and symptoms of a deltoid ligament sprain but with equivalent stress radiographs, an MRI can be useful to obtain an objective confirmation of the diagnosis. However, MRI findings must be used cautiously, as MRI is highly sensitive and can demonstrate

clinically insignificant changes in the medial ligamentous complex.⁶⁵ In a series of 36 patients with acute deltoid ligament injuries evaluated with MRI, Jeong et al. showed that 58% of patients had complex tears of the superficial and deep components of the deltoid ligament after rotational ankle fractures. The tibionavicular ligament was the most commonly disrupted, occurring most often at the anterior colliculus of the medial malleolus.⁶⁶

Ankle arthroscopy is another alternative for diagnosis when nonoperative treatment fails in the setting of possible deltoid ligament injuries.⁵⁹ Hintermann et al. found that at least 40% of 288 ankle fractures had deltoid ligament damage upon arthroscopic assessment.⁶⁷

Nonoperative Treatment

In general, isolated acute deltoid ligament ruptures are treated nonoperatively. Treatment of athletes with isolated grade 1 and most grade 2 deltoid sprains is similar to the nonoperative treatment of acute lateral ankle sprains, but return to play can be more prolonged.^{55,68} Cold therapy and the use of a pneumatic brace, walking boot, or rarely a walking cast is started as soon as possible. The athlete can be weight bearing as tolerated with the use of crutches, advancing weight as pain allows. Return to play can be expected in 3 to 6 weeks depending on functional progression.

More severe grade 2 and grade 3 deltoid sprains can be treated nonoperatively in a walking boot or cast if the mortise reduction is maintained on radiographs. Similar weight-bearing progression can be used, but immobilization may be necessary for 6 to 8 weeks to prevent external rotation or medial collapse of the foot and allow healing of the deltoid complex, which can include injury to the spring ligament. The foot and ankle are supported with a custom orthotic device upon return to sport.

Operative Treatment

Proponents of surgical repair of the deltoid ligament in the athlete believe that nonanatomic healing may lead to persistent medial gutter pain, instability, and functional loss over the long term, with the potential for early arthritis.⁶⁹ In a prospective, randomized controlled study of 41 patients with supination-external rotation type 4 equivalent injuries, Rungprai showed that while there were no differences in functional outcome between casting and repair of deltoid ligament injuries, deltoid repair resulted in less medial-sided ankle pain and decreased medial clear space widening.⁷⁰ Woo et al. showed in their retrospective, comparative case series that patients undergoing syndesmotic fixation with deltoid repair had a significantly smaller medial clear space and better outcome scores as compared with conservative treatment of the deltoid ligament at an average of 17 months postoperatively.⁷¹ While there is controversy regarding treatment of the deltoid ligament injury in well-reduced mortises, an entrapped deltoid ligament or posterior tibial tendon (Fig. 15.18) preventing reduction of the ankle mortise requires an open medial ankle arthrotomy and repair of the deltoid ligament and/or the posterior tibial tendon.

Numerous descriptions of deltoid ligament repair and reconstruction techniques can be found in the literature. These vary based on the severity of the injury and surgical indications. Hintermann et al. described approximation of the avulsed tibionavicular and tibiospring portions of the deltoid ligament with

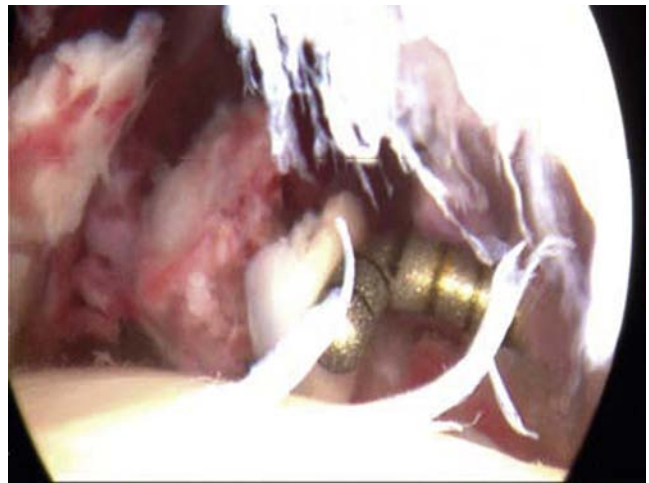


Fig. 15.18 Clinical photograph of a torn and entrapped posterior tibial tendon in a patient with a Weber C ankle fracture. (Courtesy Thomas O. Clanton, MD)

a suture anchor 6 mm proximal to the inferior tip of the medial malleolus.⁷² Jackson et al. used a Kessler-type suture tied over drill holes in the medial malleolus to repair an isolated, complete rupture of the anterior deltoid in a football player.⁷³

Before an open deltoid repair, ankle arthroscopy is performed to diagnose any associated injuries and evaluate the articular surface. Once this is completed, the surgical repair of the deltoid ligament begins with an incision made from 3–4 cm above the posterior edge of the medial malleolus paralleling the posterior tibial tendon and extending to the talonavicular joint. The superficial and deep bands of the deltoid are inspected as well as the posterior tibial tendon. The deltoid is repaired anatomically using nonabsorbable or slowly absorbable sutures. The deep deltoid is approximated before repairing the superficial structures. In the setting of an avulsion of the ligament, or a short stump at the distal or proximal end, reattachment with a suture anchor is preferable. The neurovascular bundle should be protected throughout the procedure.

Postoperative care may be dictated by the associated injuries (fibular fracture, syndesmosis tear, etc.) rather than the deltoid repair. For the deltoid, the ankle is splinted for 7 to 10 days and the athlete is kept nonweight bearing. The athlete is then transitioned to a boot or cast and remains nonweight bearing for another 3 weeks. At 4 to 6 weeks from surgery, the athlete starts a weight-bearing progression over 2 to 4 weeks based on radiographic evidence of healing of any associated fracture. An ankle brace with a medial arch support is advised for the first 6 months after surgery.

Limited research is available regarding treatment of deltoid ligament injury in the athlete. Such injury is usually reported in studies of other injuries to the ankle. In a level 4 case series, Hsu et al. reported on 14 NFL players treated with ankle arthroscopy with debridement, followed by fibular fracture fixation with plate and screws, syndesmotic fixation with suture-button devices, and open deltoid complex repair with suture anchors. Offensive lineman was the most common position sustaining this injury (9/14). All players returned to running and cutting maneuvers by 6 months after surgery. Return to play was 86% for all players.⁶⁹ In another series of 61 deltoid ligament reconstructions performed in the acute setting, Aljabi et al. reported that 100% of patients returned to sports within 3.5 months postoperatively.^{70a} In

general, athletes are unique in their ability to return to play, and that may not always be the case among the general population, as shown in the study by Hong et al., in which only 27% of patients from the general population with rotational ankle fractures were able to return to their preinjury athletic activity.⁷⁴

Currently, it is unclear whether surgical repair of a complete deltoid rupture offers the athlete a quicker return to sport when compared with conservative treatment. Further research is needed to compare the long-term outcomes of nonoperative treatment versus operative repair of deltoid ligament injuries, particularly in the absence of fibular fracture and/or syndesmosis injury. With the small numbers of isolated deltoid ligament injuries and the confounding effects of associated injuries, it is difficult to predict whether repair of the deltoid ligament leads to decreased rates of chronic medial instability and posttraumatic arthritis in the athlete. Nevertheless, it seems intuitive that if there is clear injury to the deltoid ligament and instability is present, repairing the deltoid and restoring stability offers the athlete a better chance of returning to their preinjury level of performance.

Chronic Medial Instability

Very few series of chronic deltoid injuries have been reported, and none have specifically addressed the athletic situation.^{75,76} Hintermann et al. arthroscopied 148 ankles for chronic instability and found a 40% incidence of deltoid lesions.⁷⁶ All were associated with a lateral ligament injury. The addition of arthroscopy contributed to a 10% higher diagnosis of medial instability than that determined by clinical diagnosis alone. Almost 100% of ankles with deltoid ligament injuries had evidence of cartilage damage in contrast with 66% of the ankles with lateral ligament injury alone.

Assessment

Patients with medial instability usually have pain and tenderness at the medial ankle. They generally exhibit increased heel valgus when examined during weight bearing. Their ability to actively correct their heel valgus by contracting the tibialis posterior muscle is indicative of a deltoid injury.⁷⁷ Valgus stress radiographs usually show valgus instability of the ankle or medial clear space widening, but the AP and oblique weight-bearing views of the ankle may show a decreased medial or lateral joint space. MRI is helpful in identifying deltoid pathology and visualizes the separate components of the superficial and deep deltoid. An arthroscopic method for grading medial ankle joint stability using probes or shavers of varying size has been proposed by Hintermann et al.⁷⁵ The joint is considered stable if only a 2-mm probe or shaver can be passed into the medial gutter, moderately unstable with a 5-mm arthroscope or probe, and severely unstable with opening greater than 5 mm and visualization of the posterior ankle joint.

Nonoperative Treatment

Conservative treatment of chronic deltoid instability includes various methods for specific goals: (1) reduction of inflammation through oral or topical medications, physical therapy modalities, and immobilization; (2) stabilization of the ankle with taping or bracing; (3) improving the foot and ankle position through use of inserts or orthoses; and (4) strengthening the surrounding musculature to help stabilize and protect the ankle joint. Normally, a combination of these methods is utilized with



Fig. 15.19 Drawing depicting allograft tendon reconstruction of the deltoid ligament using a TightRope through a tibial tunnel with suture-button and/or interference screw fixation through talar and calcaneal tunnels. (From Arthrex Inc. with permission)

varying degrees of success. If these measures fail to achieve satisfactory results, surgical reconstruction is the next step.

Operative Treatment

Two approaches may be used depending on the availability and quality of tissue apparent at surgery. If the tissues are of good quality, direct imbrication may be performed.⁷⁷⁻⁷⁹ A suture anchor can be used to tighten the tibionavicular component of the superficial deltoid and/or the deep deltoid ligament. In contrast, if the tissues are inadequate, an autograft or allograft tendon can be used to reconstruct the deficient deltoid (Fig. 15.19).⁸⁰⁻⁸³

Hindfoot valgus is an indication to perform a medial displacement calcaneal osteotomy combined with the use of an allograft reconstruction technique to protect the reconstructed ligament and other medial structures. According to a finite element analysis of deltoid reconstructive techniques, no method can fully restore the kinematics of the normal ankle with regard to flexibility and stresses on the ligament components.⁸⁴ Evaluating the procedures used to date for chronic deltoid instability reconstruction reveals no satisfactory long-term results, indicating a need for more work in this area.

Two series of surgically treated chronic medial ankle instability include younger, more athletic patient populations. Hintermann et al. reported on 51 patients with 52 ankles treated for medial ankle instability. Patients had an average age of 36 years (range, 16–60) and their average follow-up was 4.43 years (range 2–6.5 years).⁷⁷ In 49 of the 52 cases the deltoid was reattached and imbricated utilizing sutures and suture anchors. Three patients were treated with reconstruction using a plantaris tendon autograft. A calcaneal osteotomy was performed in the 14 patients with more severe deformity. Results were good to excellent in 46 cases (90%). In the only other series of younger individuals with chronic deltoid instability, Buckhorn et al. reported a series of combined medial and lateral ankle ligament reconstructions in 81 patients. There was no indication of the exact number and level of athletes, but the mean age was

32 years (range, 14–44) with 1 year follow-up.⁷⁹ The authors commented that they felt that professional athletes, in particular, needed medial side stabilization along with lateral ligament reconstruction in cases of rotational instability.

Not only is there a deficiency in level 1 and 2 studies on chronic deltoid instability; there are no long-term studies of this condition and its surgical or nonsurgical treatment in an athletic population. What is known is that it can occur in association with lateral ankle instability and that medial imbrication can be effective when there is good tissue and a well-aligned foot. In other cases, there is no evidence-based answer. Considering that, the authors recommend reconstruction with an autograft or allograft tendon and a realigning calcaneal osteotomy. Potentially, there will be a role for augmentation using an InternalBrace. When reconstruction is performed, restoration of the anatomic footprint of the deltoid ligament complex is essential. A clear understanding of the anatomic locations of the components of the superficial and deep components of the deltoid, as well as their radiographic locations, can be valuable.^{61,85} If the deltoid heals in a nonanatomic fashion, this may lead to worse clinical and functional outcomes in the short and long term.^{86–89} Regardless, it should be recognized that this can be a career-ending injury in a high-level athlete.

SYNDESMOSIS SPRAINS

Acute Syndesmosis Sprain

Acute injuries to the syndesmosis continue to be challenging both for diagnosis and treatment. Unlike a lateral ankle sprain, distal tibiofibular joint injury, or a “high ankle sprain,” results in significantly increased disability and often requires surgical management.^{90–94} Further confounding the subject is the frequent presence of concurrent injuries such as fractures and deltoid injury. This section will specifically deal with syndesmosis injury alone, although some of the principles of diagnosis, treatment, and return to sport can apply to the other situations. Injury can occur both without a fracture and without obvious diastasis.^{90,95} Due to the configuration of the joint and elasticity of the tissues, spontaneous reduction is common in less severe cases, and diastasis will not be apparent initially. Advanced imaging technology and increased focus on this topic in research journals and medical conferences has led to an increased ability to diagnose this injury. However, treatment is controversial and evolving in terms of when to treat, how to treat, and when to allow weight bearing and return to play.

Assessment

An understanding of the mechanism of injury in syndesmosis sprains has become clearer due to high-grade videos of the injury in professional athletes, with most injuries demonstrating external rotation of the body and leg over the foot that is fixed on a surface. Varying degrees of ankle dorsiflexion and load are contributing factors.^{90,96,97} If the athlete presents shortly after the injury, localized tenderness and swelling at the syndesmosis can be clearly defined. The physical examination is notable for being difficult without using multiple parameters. These include an inability to hop, anterior inferior tibiofibular ligament (AITFL)-specific tenderness, a positive dorsiflexion–external rotation stress test, along with pain out of proportion to the injury and a positive squeeze test.

BOX 15.4 Classification of Traumatic Syndesmosis Disorders

Acquired: Traumatic

A. Acute

1. Sprain without diastasis
2. Latent diastasis
 - a. Stable, no deltoid injury, negative squeeze test
 - b. Unstable, deltoid injury and/or positive squeeze test, ± PITFL injury
3. Frank diastasis (per Edwards and DeLee)
 - Type I. Lateral subluxation without fracture
 - Type II. Lateral subluxation with plastic deformation of fibula
 - Type III. Posterior subluxation/dislocation of fibula
 - Type IV. Superior subluxation/dislocation of talus into mortise

B. Subacute (3 weeks to 3 months)

C. Chronic (longer than 3 months)

1. Without tibiotalar arthritis
 - a. Without synostosis
 - b. With synostosis
2. With tibiotalar arthritis

Modified from Edwards GS Jr, DeLee JC. Ankle diastasis without fracture. *Foot Ankle* 1984;4:305-312; Clanton TO, Schon LC. Athletic injuries to the soft tissues of the foot and ankle. In: Mann RA, Coughlin MJ, eds. *Surgery of the Foot and Ankle*, 6th ed, vol 2, St. Louis, Mosby; 1993, p. 1149, Table 27.6; Calder JD, Bamford R, Petrie A, McCollum GA. Stable versus unstable grade II high ankle sprains: a prospective study predicting the need for surgical stabilization and time to return to sports. *Arthrosc* 2016;32(4):634-642.

It can be useful when the radiographic signs from a weight-bearing set of ankle radiographs are positive. Unfortunately, the tendency toward self-reduction makes reliance on radiographic views and measurements unreliable for diagnosis unless they are very clearly positive.⁹⁸ Even stress radiography and intraoperative stress fluoroscopic imaging are often inaccurate. Diagnostic ultrasound is helpful but very dependent on the experience of the examiner.⁹⁹ CT and MRI have emerged as the most accurate imaging methods, with MRI having 97% accuracy and the ability to visualize individual ligaments.^{100,101} In the care of high-level athletes, examination under anesthesia and ankle arthroscopy have become more commonplace for diagnosis and treatment.^{102–104}

Nonoperative Treatment

In the acute injury, initial management includes rest, ice, compression, and elevation (RICE). The affected extremity should be immobilized in a splint or CAM boot, the patient should remain nonweight bearing, and the appropriate diagnostic tests should be ordered. Treatment is based on a modified classification system of syndesmosis injuries (Box 15.4).^{34,105,106} Within the acute traumatic injuries are type 1—stable, which can be treated nonsurgically. Patients are made weight bearing as tolerated in a CAM boot or brace. Crutches are used if pain prevents weight bearing. Physical therapy can start when pain subsides and weight bearing becomes easier. Expected time to recovery for these patients in one of the West Point studies was 43 days, barring reinjury. Type 2—latent diastasis injuries have normal routine weight-bearing x-rays but are diagnosed by positive stress x-rays. These are typically the most troubling to diagnose and treat, leading to a subdivision of type 2a which are stable, have a normal deltoid, and a negative squeeze test. If confirmed by imaging to have an anatomically reduced syndesmosis, this

group of patients can be treated nonoperatively with cast or CAM boot immobilization for 2 to 4 weeks, careful weight-bearing progression, and gradual rehabilitation. Average return to play (RTP) in this group is 6 weeks. Subtype 2b patients are unstable, with a deltoid injury and/or a positive squeeze test, and may have injury to the posterior inferior tibiofibular ligament (PITFL).¹⁰⁶ This group typically takes longer to recover (average RTP is 9 weeks). The authors have a low threshold for performing an examination under anesthesia and arthroscopy in athletes in this subtype, since we believe that there is more assurance that they will heal with stability and earlier return to play, with less chance of time loss from a reinjury. Type 3 frank diastasis patients were subcategorized by Edwards and DeLee into four different subtypes of frank diastasis injuries: type I, lateral subluxation without fracture; type II, lateral subluxation with plastic deformation of the fibula; type III, posterior subluxation/dislocation of the fibula; and type IV, transsyndesmotom dislocation of the talus—also called the “Logsplinter” injury.^{105,107}

Operative Treatment

Patients with latent (type 2b) or frank diastasis (type 3) are considered unstable and are treated surgically if their medical comorbidities do not preclude surgery. The patient is taken to the operating room as soon as the soft-tissue envelope allows. We prefer to operate as soon as possible following the injury before the ankle becomes massively swollen. If significant swelling is already present when the patient is evaluated, it is best to wait until the swelling resolves (5 to 10 days) before operating. In the surgical care of acute syndesmosis injuries, we always utilize ankle arthroscopy to assist in the diagnosis and treatment. Using both anteromedial and anterolateral portals, the articular surfaces are inspected, and a 3-mm probe or 2.9-shaver is used to confirm injury to the syndesmosis. Additionally, one can use a Freer elevator to actively stress the distal tibiofibular joint. Injury to the deltoid ligament and the AITFL can be visualized and probed to confirm a tear. We place the lateral portal so that it can be incorporated into the subsequent exposure of the anterior syndesmosis. This can then be used to place sutures or suture anchors for the torn AITFL and confirm an anatomical reduction of the syndesmosis. Since the lateral branch of the superficial peroneal nerve is within millimeters of this incision, careful dissection and gentle retraction are essential to avoid nerve injury. The distal tibiofibular joint is inspected after exposing the tear in the AITFL. Any ligamentous tissue or debris in the tibiofibular space is removed. If there is an avulsed bone fragment from the anterior tubercle, it is evaluated to determine whether it is large enough to take a screw without fragmenting. If it is, a small screw, with or without a soft-tissue washer, is used to reattach the fragment. If it is too small, the fragment is excised, and the ligament is repaired with a suture anchor.

An additional incision is made at the posterior edge of the fibula just above the plafond to accommodate fixation and aid in reduction. The authors now perform the reduction manually, since this has been shown to be more likely to produce anatomic reduction than with clamp fixation.^{108,109} A syndesmosis clamp is applied to hold that reduction while fixation is secured; however, only one or two clicks are used with the clamp, because multiple studies suggest that a syndesmosis clamp overcompresses the tibiofibular joint, although the

BOX 15.5 Research Supporting Use of Suture-Button Fixation of the Syndesmosis

1. Suture-buttons allow triplanar motion.¹⁵⁵
2. Suture-button system promotes self-reduction of the joint.¹⁵⁶
3. Suture-buttons have faster rehabilitation and quicker return to work than screw fixation.^{157–162}
4. Biomechanics studies and clinical studies show that suture-buttons provide the same stability as syndesmotom screws.^{163–166}
5. Cost-effective solution because these implants rarely require removal.^{167–168}
6. Syndesmotom screws experience improvement in symptoms and function following removal.¹⁶⁹

clinical significance of this finding is still unknown.^{109–113} It is critical to correct any external rotation deformity and confirm an anatomic reduction visually and radiographically, since this correlates directly with improved outcomes.^{114,115} The only other reliable method to confirm reduction is the use of CT in the operating room, with an O-ring system.¹¹⁶ We have used this technology, and believe it is accurate, but widespread utilization is doubtful because of cost and radiation exposure to the patient and operating room personnel.

If anatomical reduction cannot be obtained, a medial incision is made over the deltoid ligament to remove any interposed tissue blocking reduction. In some cases, this tissue can be identified arthroscopically and removed. Sutures or suture anchors are placed in the ruptured deltoid ligament but not tied. A second attempt at reduction is performed. If the fibula still cannot be reduced in the incisura, the preoperative x-ray films and fluoroscopic views are reevaluated in younger patients to see whether plastic deformation of the fibula might have been missed. When present, this deformation requires that an osteotomy of the fibula be performed before stabilizing the distal tibiofibular joint with a syndesmotom screw. Edwards and DeLee recommend that the osteotomy of the fibula be performed proximally because of the instability of a distal osteotomy with damage to the interosseous membrane.¹⁰⁵

Once anatomic reduction is achieved and held, a drill hole is made in the anatomic plane 1.5 to 2.5 cm proximal to the tibiotalar joint.¹¹⁷ Biomechanically, a screw or suture-button placed 1.5 to 2.5 cm above the joint line results in less widening (compared with fixation placed more proximally) and will not violate the synovial capsule or articular cartilage of the distal tibiofibular joint.^{118,119} It is our preference to use endobuttons for fixation in lieu of screws for several reasons (Box 15.5).

An additional suture-button is often placed 1 cm or one plate hole superior to the first in a divergent fashion aiming 15 degrees posterior to the first implant when it is considered to be necessary for additional stability. Once the screws or suture buttons are in place, any previously placed deltoid sutures are tied (if a medial incision was necessary), and absorbable sutures are placed and tied in the AITFL, as it contributes at least 24% of syndesmotom stability.¹²⁰ When the tissue for the AITFL repair is of poor quality and will not hold sutures, an Internal Brace is placed over the anatomical location of the AITFL (Fig. 15.20).^{121,122}

If one chooses to use a screw, a fully threaded cortical screw that crosses four cortices is our recommendation, since this will allow easier removal of a broken screw. Otherwise, based on

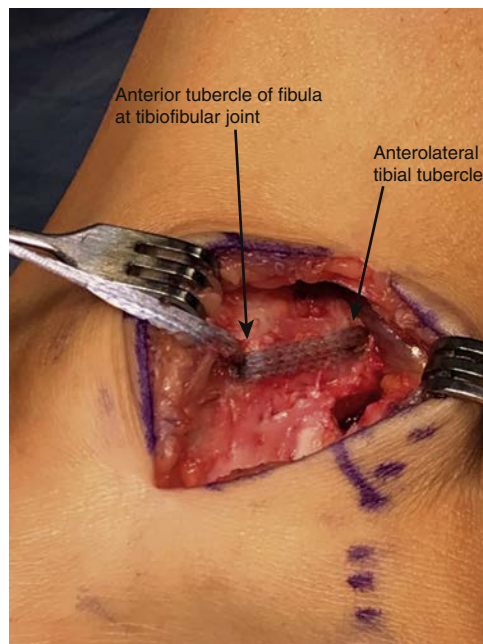


Fig. 15.20 Intraoperative image of anterior inferior tibiofibular ligament (AITFL) Internal Brace placed over repair of torn AITFL. (Courtesy Thomas O. Clanton, MD)

available literature, there is no major difference in functional outcomes between tricortical and quadricortical screws at 1 year, nor is there a difference between titanium and stainless steel, one or two screws, metal or bioabsorbable screws, or transsyndesmotomic and suprasyndesmotomic fixation.^{123,124} The screw(s) may be placed through a 3- or 4-hole plate with unicortical screws at the top and bottom, thus allowing removal of the syndesmosis screw(s) while keeping the plate to protect the screw hole(s) during return to athletics.¹²⁵ Alternatively, an endobutton can be placed through one of the screw holes and can be left in place when the syndesmosis screw is removed. Fixation with the ankle in dorsiflexion has been advocated to prevent overtightening of the syndesmosis, but this does not appear necessary with screws or suture-buttons.^{110,126,127}

Rehabilitation is covered in a later section for this condition, but in general, patient care is individualized based on the severity of the injury, the stability of the fixation, and the progress of the patient in reaching specific goals with therapy. Most patients with syndesmosis injuries alone can be well stabilized, which can allow splinting until wound healing (typically 7 to 14 days). Once the splint is removed, the patient can be placed in a CAM boot and removed intermittently for range of motion. Progressive weight bearing can begin over a 2- to 6-week period, guided by pain and function. A more aggressive alternative in a well-stabilized syndesmosis can be early use of a cold compression system (e.g., Game Ready) along with weight bearing as tolerated in a CAM boot with earlier return to sport using taping, bracing, and potentially sparring the shoe.

Chronic Syndesmosis Sprain and Instability

Chronic syndesmosis injuries are classified as persistent widening of the distal tibiofibular joint greater than 3 months old. These chronic injuries can be sources of chronic pain and dysfunction. Such injuries can lead to poor outcomes and significant disability for the patient. The classification of chronic syndesmotomic injuries

is based on the presence or absence of tibiotalar arthritis (Box 15.4). In many cases, these injuries have been treated conservatively and the athlete is now complaining of residual pain from the prior injury. Sometimes this is from malreduction, while in others the injury was missed altogether. Treatment depends on the findings and degree of pain and disability, but typically requires surgical intervention.

Assessment

In patients who complain of symptoms from a syndesmosis sprain for more than 3 months, it is important for the physician to have a high index of suspicion. Advanced imaging, in addition to standard radiographs, is warranted. Both CT scan and MRI have a role in the diagnosis and treatment of these patients. CT scan allows better visualization of the position of the fibula within the distal tibiofibular joint. It also allows the physician to better visualize the presence of synostosis and the amount of heterotopic bone present. CT scan has been shown to be more effective at finding patients who have subtle diastasis, in particular when the widening was 3 mm or less above the normal upper limit of the tibiofibular clear space of 6 mm.^{128,129} These cases of subtle diastasis are often missed on plain radiographs.¹²⁹ Both angular and area measurements taken with CT scan are reliable measurements and make chronic malreduction of the fibula easier to identify.¹³⁰ MRI also aids diagnosis and assessment of intraarticular pathology and has been proven to be a sensitive, specific, and accurate tool for the diagnosis of chronic syndesmotomic injury.¹³¹ One of the MRI findings that can be useful in a chronic situation where the patient has clinical findings indicative of injury to the syndesmosis is the “Lambda sign” described by Ryan et al.¹³² Their study found this to be both sensitive (75%) and specific (85%) when judged by 2 mm diastasis seen on subsequent arthroscopy (Fig. 15.21). MRI is also effective at determining the amount of inflammation present and determining the amount of cartilage damage.

Operative Treatment

Ankle arthroscopy is the standard for visualizing the condition of the entire ankle joint. Significant synovitis is expected in the chronic setting and a “meniscoid-type” lesion is often seen with hypertrophic scar tissue filling the lateral gutter and syndesmosis from the initial injury. A probe or a shaver can be used to determine if there is diastasis present. In many cases where the joint is salvageable, debridement and reconstruction of the syndesmosis can be performed. In cases of chronic injuries with associated cartilage pathology, the extent of damage becomes crucial in determining whether microfracture (with or without use of biologics), autologous osteochondral transplantation, or fresh allograft transplantation may be needed. In more severe cases, arthroplasty or arthrodesis may be necessary to relieve symptoms.

Arthroscopic debridement of the joint has been used previously for treatment of chronic syndesmosis injuries. In one of the original studies of this method, Ogilvie-Harris and Reed reported significant improvement in patients’ symptoms with debridement of the hypertrophied synovitic tissues within the joint.¹³³ In a more recent prospective randomized trial of 20 patients, Han et al. showed no difference in outcome scores in patients with chronic syndesmosis injuries with arthroscopic debridement alone versus those with debridement and screw fixation.¹³¹

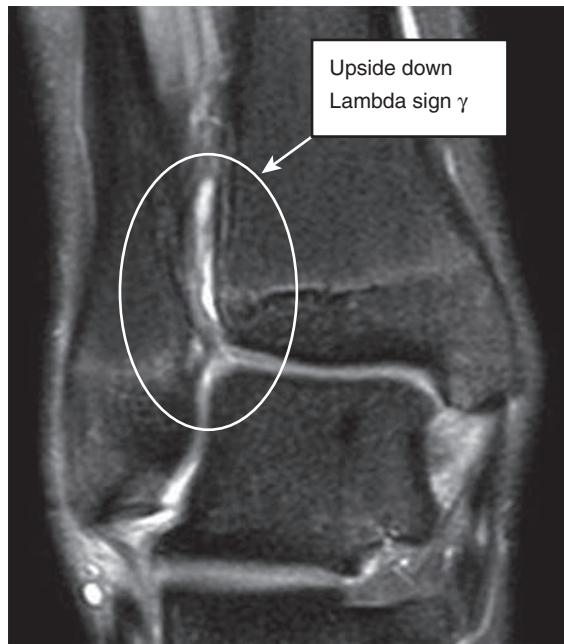


Fig. 15.21 Example of a magnetic resonance image with lambda sign indicative of latent syndesmotic injury. (Courtesy Thomas O. Clanton, MD)

In many cases, reconstruction of the joint is necessary. The goal remains restoration of the normal relationship between the distal tibia and the distal fibula while stabilizing the ankle mortise. Various techniques have been described. In most cases, simple stabilization of the mortise after debridement of the medial gutter and the syndesmosis with either a suture-button construct or screws is enough. In some cases, however, more extensive reconstructive efforts are needed.

In cases where the previously ruptured AITFL is not useful, autograft or allograft reconstruction is the best alternative. Multiple techniques have been described. Grass and colleagues described a peroneus longus ligamentoplasty reconstructing the entire ligamentous complex with excellent outcomes in 16 patients who had resolution of symptoms 1 year out from surgery.¹³⁴ The technique was complicated, and as a result, simpler techniques were devised. Morris et al. described reconstruction with a hamstring autograft using a two-tunnel technique with interference screw fixation. Excellent outcomes were achieved in this small cohort of patients.¹³⁵ Lui described a minimally invasive technique utilizing three tunnels and the peroneus longus tendon. There were no long-term outcomes reported for this technique.¹³⁶ Finally, Moravek and Kadakia used a double-limbed hamstring allograft reconstruction of the syndesmosis in six patients. In contrast to the previously described methods, this technique primarily reconstructs the interosseous ligament (IOL) and is augmented with suture-button fixation. No long-term follow-up was available.¹³⁷

A final option for patients with chronic syndesmosis malunion or instability is arthrodesis of the distal tibiofibular joint. While arthrodesis of this joint can be challenging to obtain, the stability of the joint long term has been shown to be effective. Olson and colleagues described debriding the distal tibiofibular joint and fixating the arthrodesis with two 3.5-mm cortical screws placed in a lag fashion through four cortices. At an

average follow-up of 41 months, mean American Orthopaedic Foot and Ankle Society (AOFAS) scores increased from 37 ± 15 to 87 ± 11 . All associated deformities, such as fibular malunions and equinus contractures, were corrected at the time of the index procedure.¹³⁸ This study supported earlier findings by Pena and Coetzee who also recommended arthrodesis for patients with an injury older than 6 months, severe incongruity, or a recurrence of diastasis after removal of fixation.¹³⁹ The authors thought this procedure should be reserved for low-demand patients. Overcompression of the syndesmosis should be avoided, as this creates a nonanatomic mortise, increasing the risk of tibiotalar arthritis.

Chronic syndesmosis injuries are challenging injuries, and a high index of suspicion is often necessary to discover the correct diagnosis; advanced imaging is necessary for the correct diagnosis. Surgical options range from simple arthroscopy and debridement to ligamentous reconstruction of the entire complex. The degree of instability, the amount of displacement, and the extent of the arthritis all play a role in the decision-making process. Ultimately, restoration of normal anatomy with a stable, congruent joint is the goal. However, in some scenarios, this is unattainable, and viable salvage options through arthrodesis or arthroplasty must be considered.

REHABILITATION

Rehabilitation after surgical treatment of acute or chronic ankle instability related to ligamentous injury has evolved due to changing surgical techniques described earlier in this chapter. Physical therapy can be ordered earlier (as soon as the post-operative dressing is removed), with a shorter time of limited weight bearing and less restriction on allowable range of motion (ROM) (Table 15.3).^{140–142} The benefits of early rehabilitation are largely due to the process of mechanotransduction where cellular stress stimulates healing in bone, tendon, muscle, and cartilage, whereas longer periods of immobilization have been shown to be detrimental to tendon-to-bone healing.^{143,144}

Communication between the therapist and surgeon should address any ROM restrictions to protect the repair, as well as any other notable findings, such as quality of cartilage or concurrent injuries noted during the arthroscopy. Manual techniques are recommended to improve joint mobility and reduce swelling and pain. Compression stockings are useful at least until the patient is fully weight bearing, if not through the endurance phase.

Rehabilitation after surgery has shifted to a criteria-based progression (Boxes 15.1 and 15.2, and Tables 15.4 and 15.5) in lieu of earlier protocols driven by tissue-healing timelines. This gives the therapist the opportunity to treat the patient based on their own rate of recovery. The criteria have been generated from the available body of evidence related to lower extremity injury risk factors,^{145–148} tests that are effective in identifying deficits in those with foot/ankle dysfunction,^{149–154} and tests that are clinically relevant, reliable, valid, and simple to perform.

The therapist is instrumental in recommending and managing load progression from initial weight-bearing phase through to their preinjury training schedule. In the authors' experience, loading the patient too quickly can increase joint signs such as pain and swelling, but in the later phases are

often reflected only in functional ROM loss (functional plantarflexion, dorsiflexion for example) for which the loading progression should be adjusted. Objective outcome measures help define confidence in one's own recovery, which is paramount to success on the competitive stage. In contrast, poor perception of recovery and/or pain with movement are risks factors for reinjury and should trigger a follow-up physician visit.¹⁴⁵ Once the patient demonstrates symmetry or competency in the tests reflected in [Boxes 15.1 and 15.2](#), and [Tables 15.4 and 15.5](#), a conversation takes place between the athlete, surgeon, and rehabilitation team and recommendations for return to practice are established ([Box 15.3](#)).

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Osteochondral Lesions of the Ankle and Occult Fractures of the Foot and Ankle

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INTRODUCTION

Fractures of the foot and ankle are a common occurrence in the athletic population. Sometimes fractures may not be obvious on x-rays, and one must be on the lookout for occult fractures. Ankle fractures are approximately 7% of all sport-related fractures,¹ and as high as 11% in National Football League (NFL) players evaluated at the combine.² Disability due to foot and ankle stress fractures is a major source of injuries in athletes, and is seen to be as high as 15% in the running population.¹⁻³

OCCULT FRACTURES OF THE HINDFOOT

Occult fractures of the hindfoot are a significant and common cause of injury and can often be misdiagnosed as a soft-tissue injury without having a high level of suspicion. In-depth knowledge of the anatomy, coupled with a thorough history and physical examination, and a keen differential diagnosis will assist

the clinician in an accurate diagnosis. Often, further diagnostic testing, such magnetic resonance imaging (MRI) or computed tomography (CT) scans, can confirm a diagnosis.

Occult fractures of the hindfoot include anterior process of the calcaneus, talonavicular avulsion injuries, cuboid fractures, navicular stress fractures, distal tibia or fibular stress fracture, and calcaneal or talar stress fractures.

Clinical Anatomy

The foot and ankle are comprised of 28 bones, multiple joints, and connecting ligaments. The foot and ankle are vulnerable to compression and avulsion injuries with many complex and diverse movements during competitive sports. The hindfoot is made up of the tibia, fibula, talus, calcaneus, navicular, and cuboid. Often, the talus is prone to injury during athletics as it moves through both dorsiflexion/plantarflexion and inversion/eversion motions. The talus is connected at the ankle joint to

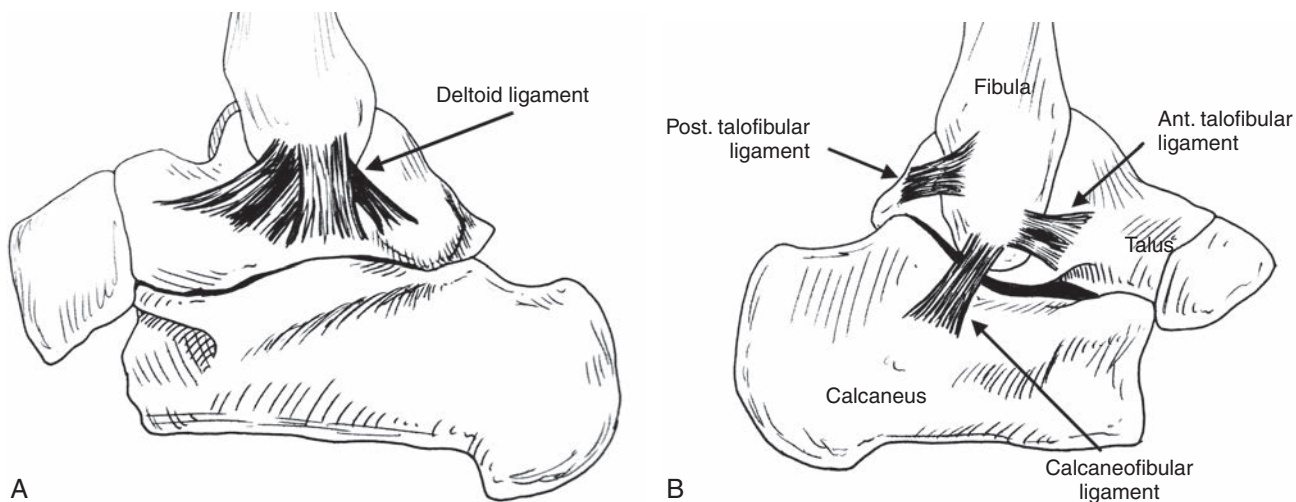


Fig. 16.1 Hindfoot anatomy of the ankle. Note the (A) medial attachment of the talus the tibia with the deltoid and (B) laterally to the fibula with the anterior and posterior talofibular ligaments.

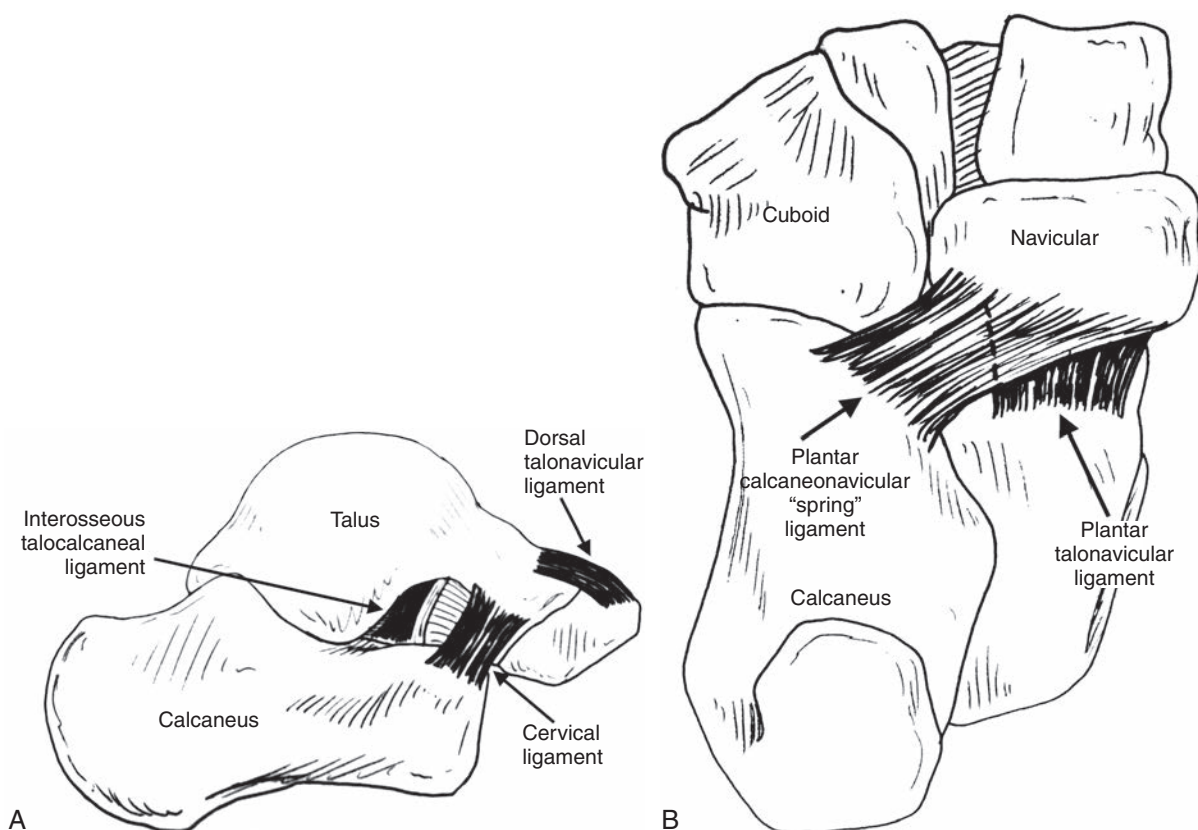


Fig. 16.2 Hindfoot anatomy of the subtalar joint. Note the attachment of the talus to the calcaneus via the (A) talocalcaneal and cervical ligaments and the talus to the navicular via the (A) dorsal and (B) plantar talonavicular ligaments.

the tibia medially through the deltoid ligament (Fig. 16.1A) and to the fibula laterally through the anterior talofibular ligament (ATFL) and posterior talofibular ligament (Fig. 16.1B). The posterior talofibular ligament attaches at the posterior talus, on the Stieda process (posterolateral) or os trigonum.

The talus is connected to the calcaneus by the talocalcaneal interosseous ligament and the cervical ligament (Fig. 16.2A). The dorsal (see Fig. 16.2A) and plantar (Fig. 16.2B) talonavicular

ligaments connect the talus and navicular. Approximately 60% to 70% of the talar surface is articular, with the ankle joint superiorly, the talonavicular joint anteriorly, and the subtalar joint inferiorly.^{4,5} Blood supply to the talus therefore is limited, coming from its ligamentous attachments and a leash of vessels surrounding the talar neck that receive contributions from the artery to the tarsal canal medially, the dorsalis pedis artery anteriorly, and the artery to the sinus tarsi laterally (Fig. 16.3A-C).^{4,6} The

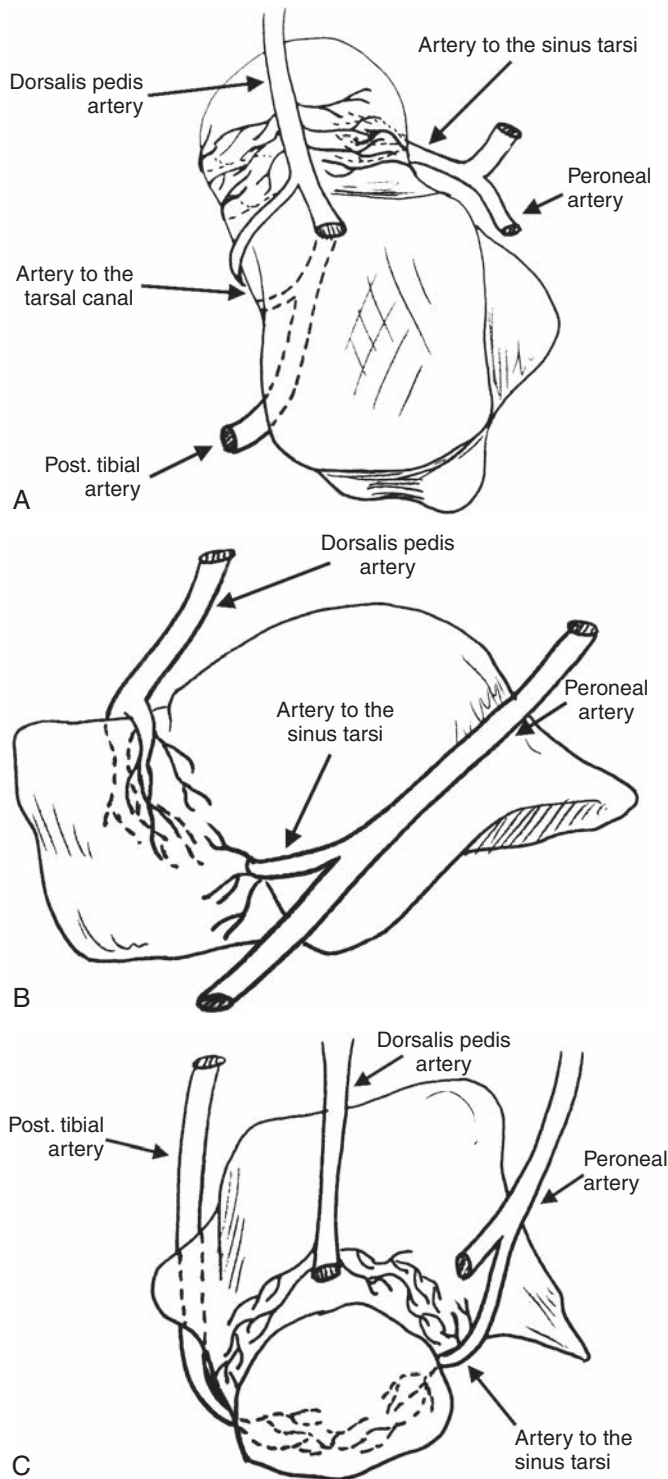


Fig. 16.3 Vasculature supply anatomy for the talus. Note contributions from the (A-C) dorsal pedis artery, (A-C) peroneal (artery to the sinus tarsi), (A) artery to the tarsal canal, and (A and C) posterior tibial artery.

internal vasculature of the talus varies considerably (Fig. 16.4).⁷ External athletic injuries to the talus that involve disruption of the vascular leash or the ligamentous attachments often produce vascular insult to the talar body or neck and may produce talar fractures or compression injuries that have delayed healing. The talus is unique in that it has no direct muscular attachments. It has seven articular surfaces along with the head, neck, body, and

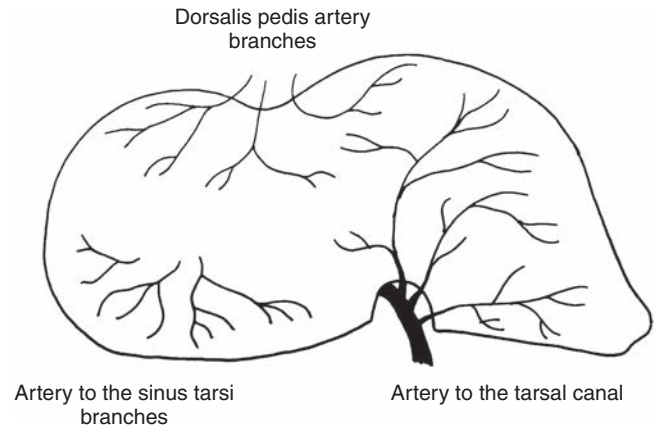


Fig. 16.4 Internal vasculature anatomy of the talus.

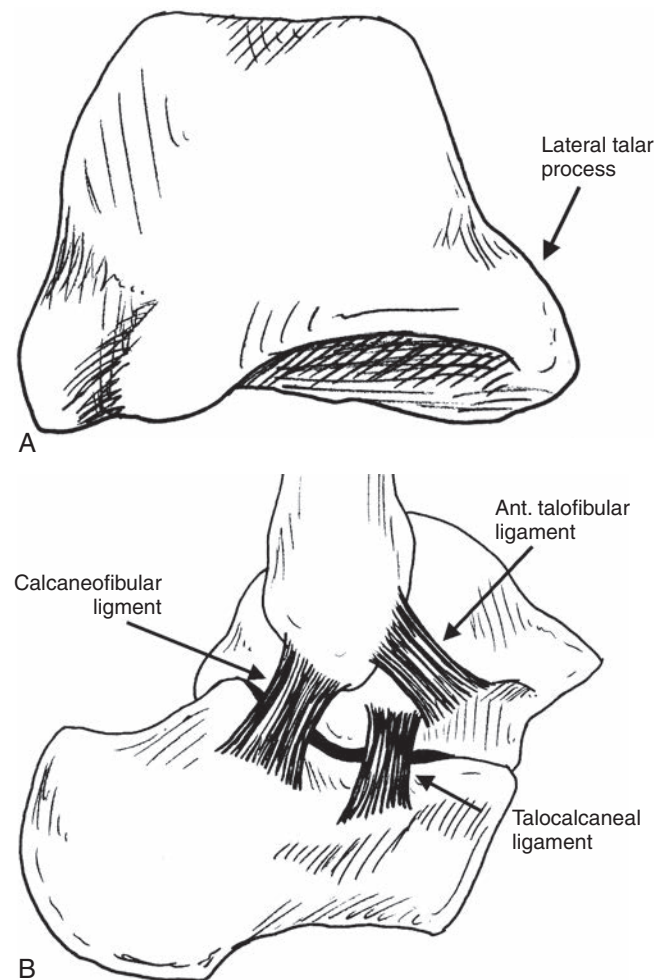


Fig. 16.5 Anatomy of the talus. Note the (A) predominance of articular surface and (B) laterally the attachment of the talocalcaneal ligament to the lateral process.

two processes: posterior and lateral. The lateral process of the talus is a wide, triangular-shaped process that slopes down to meet the lateral calcaneus (see Fig. 16.5A). On the lateral view it is wedge-shaped and articulates superiorly with the fibular surface and inferiorly with the calcaneus, forming the lateral portion of the subtalar joint (see Fig. 16.5A). The lateral talocalcaneal ligament attaches to the lateral process (Fig. 16.5B).

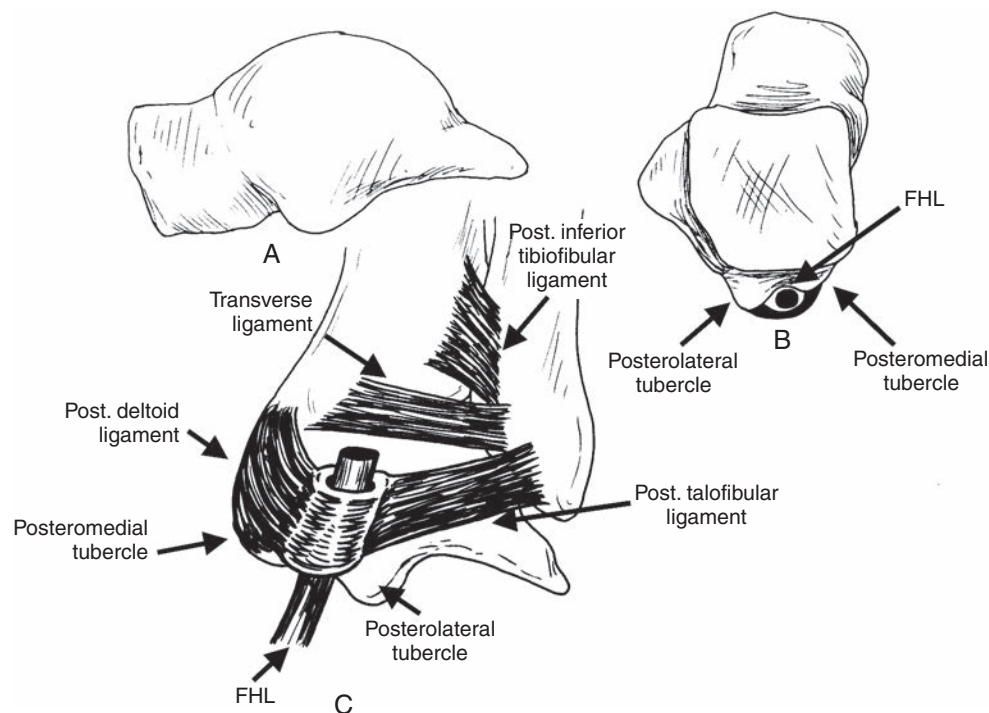


Fig. 16.6 Posterior anatomy of the talus. Note the (A) posterior process of the talus, the (B) flexor hallucis longus between the two tubercles of the posterior talus, and (C) the posterior ligamentous anatomy.

The posterior process of the talus originates from the convex-curved posterior half of the talar dome and slopes down and back to form the posterior talar “beak” or the Stieda process. Saraffian calls it the trigonal process.⁸ Inferiorly, it is concave and articulates with the posterior subtalar facet of the calcaneus. The posterior process has both a posteromedial tubercle and posterolateral tubercle. In between lies the flexor hallucis longus, which is commonly involved in posterior talar injuries (Fig. 16.6). This posterior process is widely variable in shape, from a short, rounded end to a long “beak” that is prone to injury.

The posterolateral tubercle (Stieda’s process) is larger than the posteromedial tubercle. In approximately 7% to 10% of humans a separate os trigonum may exist, connected to the posterolateral tubercle by a fibrous cartilaginous synchondrosis (Fig. 16.7A and B).⁹ The posterior talofibular ligament attaches the fibula, to the posterolateral tubercle, or the os trigonum (see Fig. 16.7B). The posterior third of the deltoid or posterior talotibial ligament attaches the posterior tibia to the posteromedial tubercle of the talus. The Y-shaped transverse or bifurcate talocalcaneal ligament is a thickening in the posterior ankle capsule that holds the two tubercles together and restrains the flexor hallucis longus (Fig. 16.8). Hallux saltans can develop at this site due to a stenosing tenosynovitis of the flexor hallucis longus that creates pain and triggering.

The calcaneus is a complex, bony structure, the largest in the foot, providing attachment for the Achilles posteriorly and the plantar fascia and plantar intrinsic muscles of the foot inferiorly. It articulates with the talus superiorly, as well as with the cuboid and navicular anteriorly. The peroneal tubercle is prominent laterally and serves as a point at which the peroneal longus and brevis tendon sheath separate. The anterolateral process of the calcaneus extends forward to form the calcaneocuboid joint.

The saddle-shaped anterior surface articulates with the cuboid anteriorly, and the superior tip articulates to a varying degree with the lateral navicular. The extensor digitorum brevis also originates from this calcaneal process. The blood supply to the calcaneus is quite robust, and fractures of the calcaneus tend to heal more easily than other fractures. The ligamentous attachments at the calcaneus are the talocalcaneal interosseous ligament, lateral talocalcaneal ligament, and cervical ligament to the talus and the calcaneofibular ligament laterally (Fig. 16.9). The posterior, lateral, and anterior calcaneocuboid ligaments and the plantar calcaneonavicular (spring ligament) and lateral calcaneonavicular ligaments connect the calcaneus anteriorly to the cuboid and navicular, respectively. The strong plantar calcaneonavicular or “spring” ligament acts as a “sling” to hold the talar head in place. The bifurcate ligament (Y-ligament) is composed of the anterior and lateral calcaneocuboid ligament (Fig. 16.10A and B) and is commonly injured during “sprain-type” inversion injuries, producing an avulsion fracture at the anterolateral process of the calcaneus. Inversion/adduction injuries of the midfoot also may produce avulsion fractures at the base of the cuboid.

The saddle-shaped cuboid forms the base of the lateral column and articulates with the anterior process of the calcaneus and may be involved in either compression or avulsion tension-type injuries. The peroneus longus tendon courses along the lateral border of the cuboid.

The tarsal navicular is a “C” or saucer-shaped bone articulating with the talus posteriorly and the cuboid laterally. The dorsal talonavicular ligament and capsule can be injured in avulsion-type injuries of the navicular from plantarflexion-type injuries. Compression-type injuries also may be produced by the impact of the talar head on the navicular. The blood supply

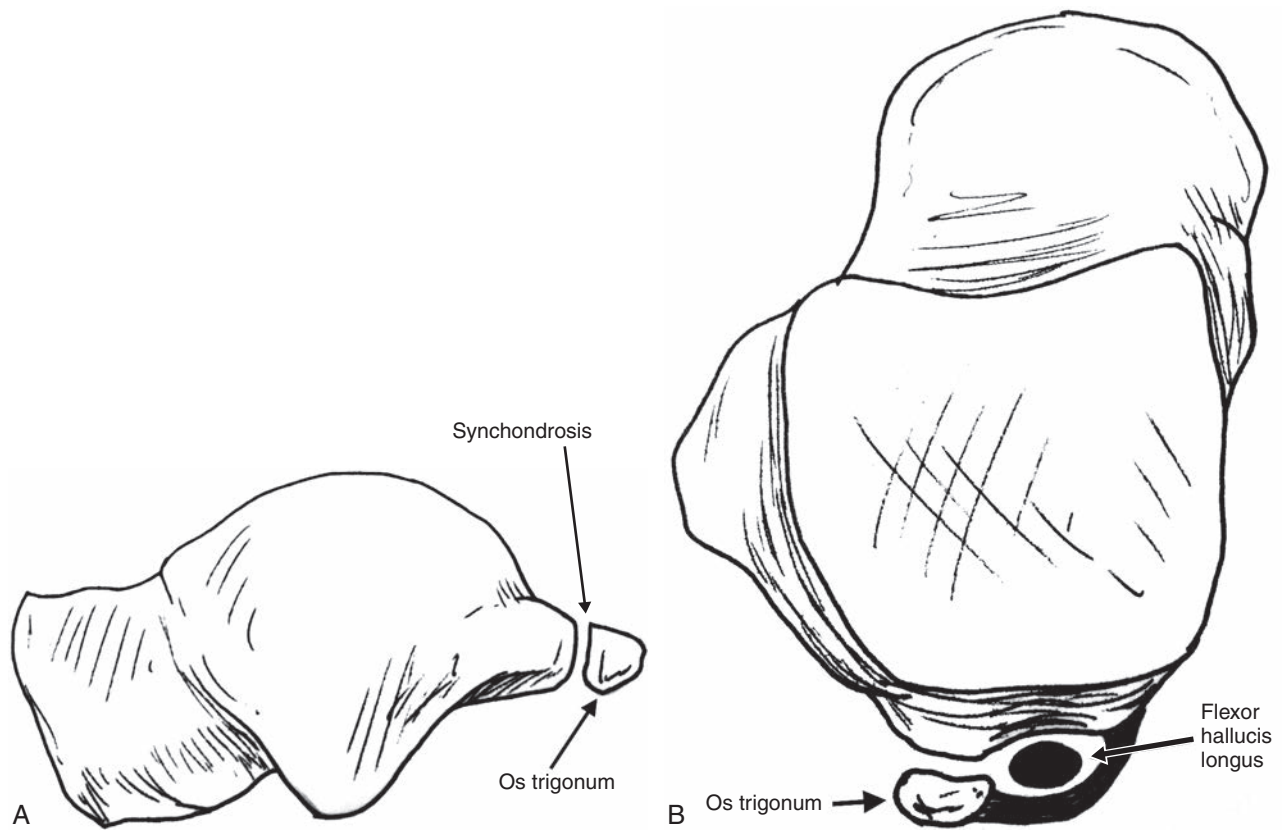


Fig. 16.7 (A) Lateral view. Anatomy of the os trigonum. Note that the os trigonum is the posterior process that is attached to the talus via a synchondrosis and (B) is attached to the posterior talofibular ligament (axial view).

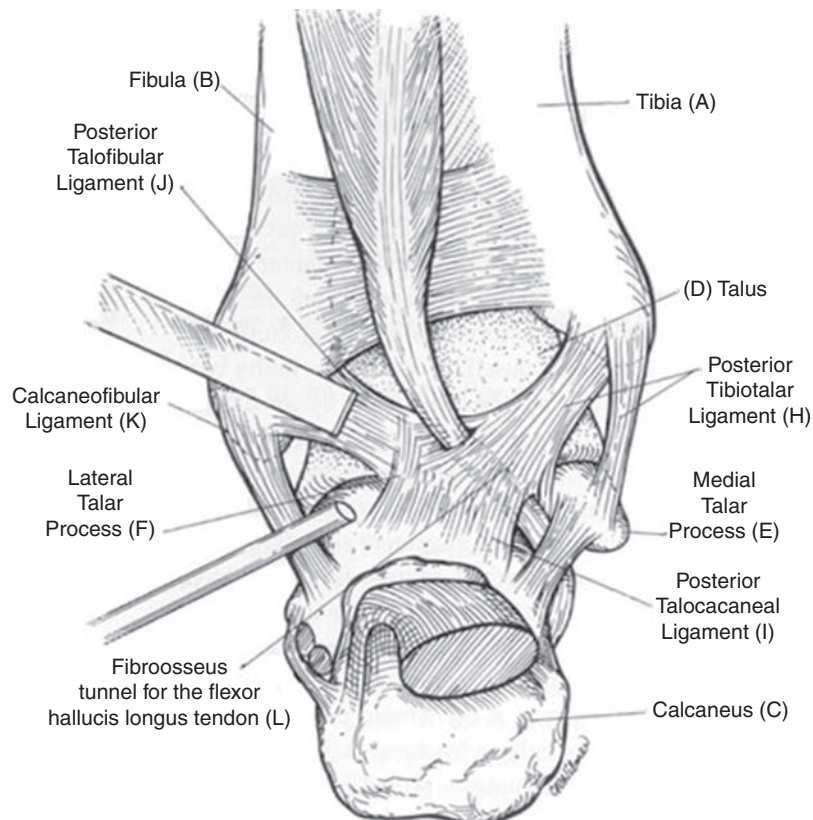


Fig. 16.8 Anatomy of the posterior hindfoot, including the ankle and subtalar joint. a = tibia; b = fibula; C = calcaneus; d = talus; e = medial talar process; f = lateral talar process; g = posterior tibiotalar ligament; h = posterior talofibular ligament; i = calcaneofibular ligament; j = fibrous tunnel for passage of the flexor hallucis longus tendon. (From *Foot and Ankle Arthroscopy*, 3e, Guhl, Boyden & Parissien, eds; Springer, 2004.)

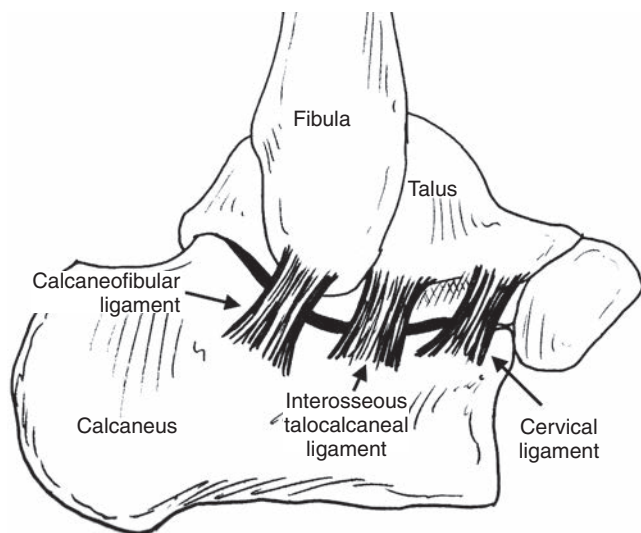


Fig. 16.9 Calcaneal ligaments. Note laterally the calcaneofibular, cervical, and lateral talocalcaneal ligaments.

to the midportion of the navicular is poor (Fig. 16.11) and may contribute to delayed healing or nonunion of such fractures.¹⁰ The articulation between the cuboid and the navicular varies from a true articulating joint to a fibrous connection to a bony bridge (tarsal coalition). Various important and powerful tendons attach to the hindfoot; these produce considerable forces during athletic activities and can create injuries. The posterior tibial tendon attaches to the navicular (Fig. 16.12A and B), producing inversion/supination and adduction while elevating the arch. It fires twice during each gait cycle or step—both eccentrically as a shock absorber and concentrically during push-off. The anterior tibial tendon, with attachments to the cuneiform and first metatarsal, is the primary dorsiflexor for the ankle and also inverts the foot. It also fires eccentrically during heel strike to decelerate and cushion the landing foot. The peroneus brevis and longus tendons (Fig. 16.13) both evert the foot and ankle and resist inversion injuries. The peroneus brevis attaches to the base of the fifth metatarsal. The peroneus longus wraps around the cuboid at the trochlea to insert broadly underneath the foot near the base of the first metatarsal, which allows the longus also to help plantarflex and stabilize the medial foot.

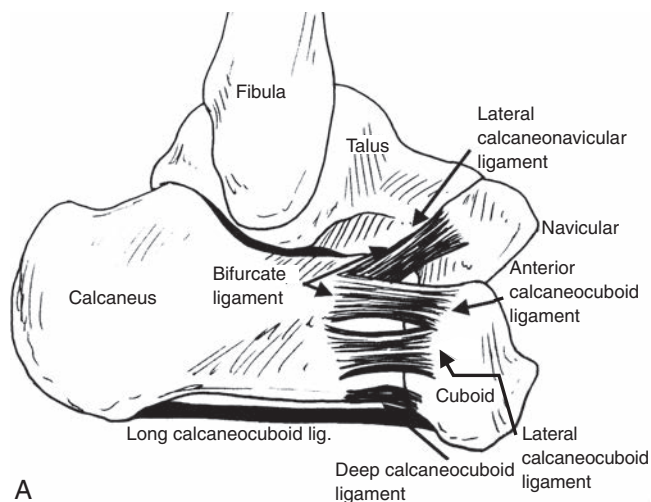
OCCULT FRACTURES OF THE CALCANEUS

The majority of fractures of the calcaneus occur from high-energy trauma, such as a motor vehicle accident or a fall from height, but there are many commonly missed calcaneal fractures and related injuries seen in the sporting population.

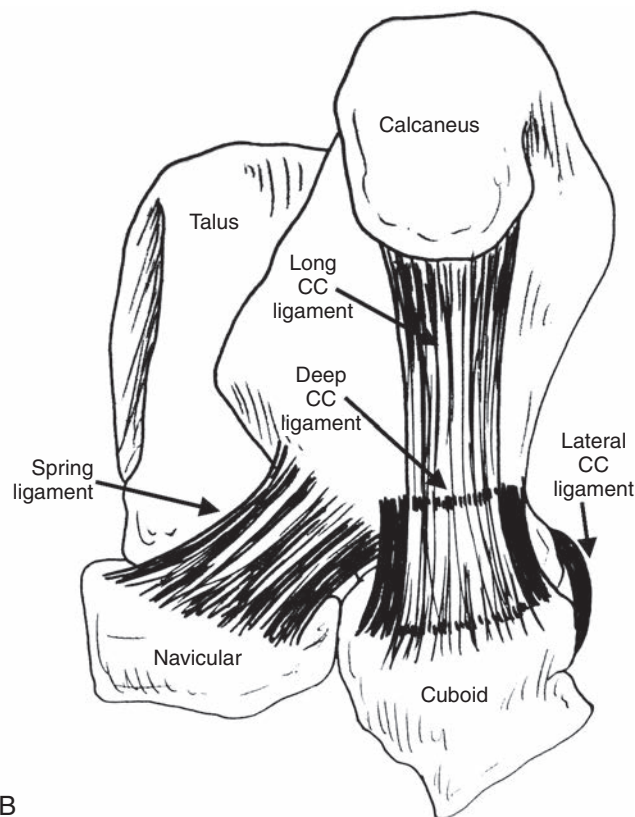
Sustantaculum Tali Fractures

Mechanism of Injury

Sustantaculum tali fractures are rare, extra-articular fractures of the calcaneus. The sustentaculum tali is a medial projection of the calcaneus that serves as an attachment of the plantar calcaneonavicular ligament (spring ligament) and for the deltoid ligament. It is also important in that the flexor hallucis longus tendon runs in a plantar groove of the sustentaculum tali. Fractures of this can lead to subtalar joint discordance that can



A



B

Fig. 16.10 Lateral plantar transverse tarsal ligaments.

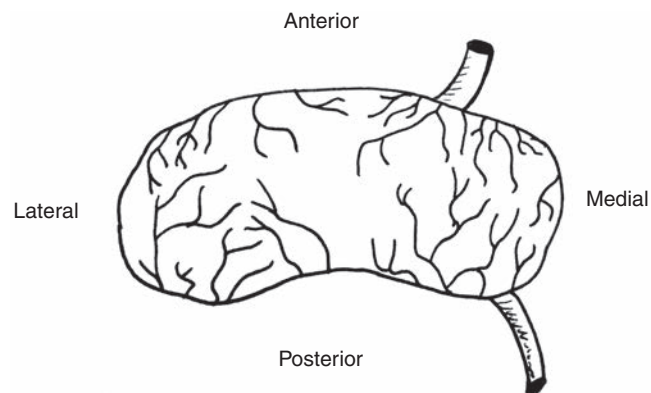


Fig. 16.11 Vasculature anatomy of the tarsal navicular. Note the central area of decreased blood supply corresponding to areas of navicular stress fractures.

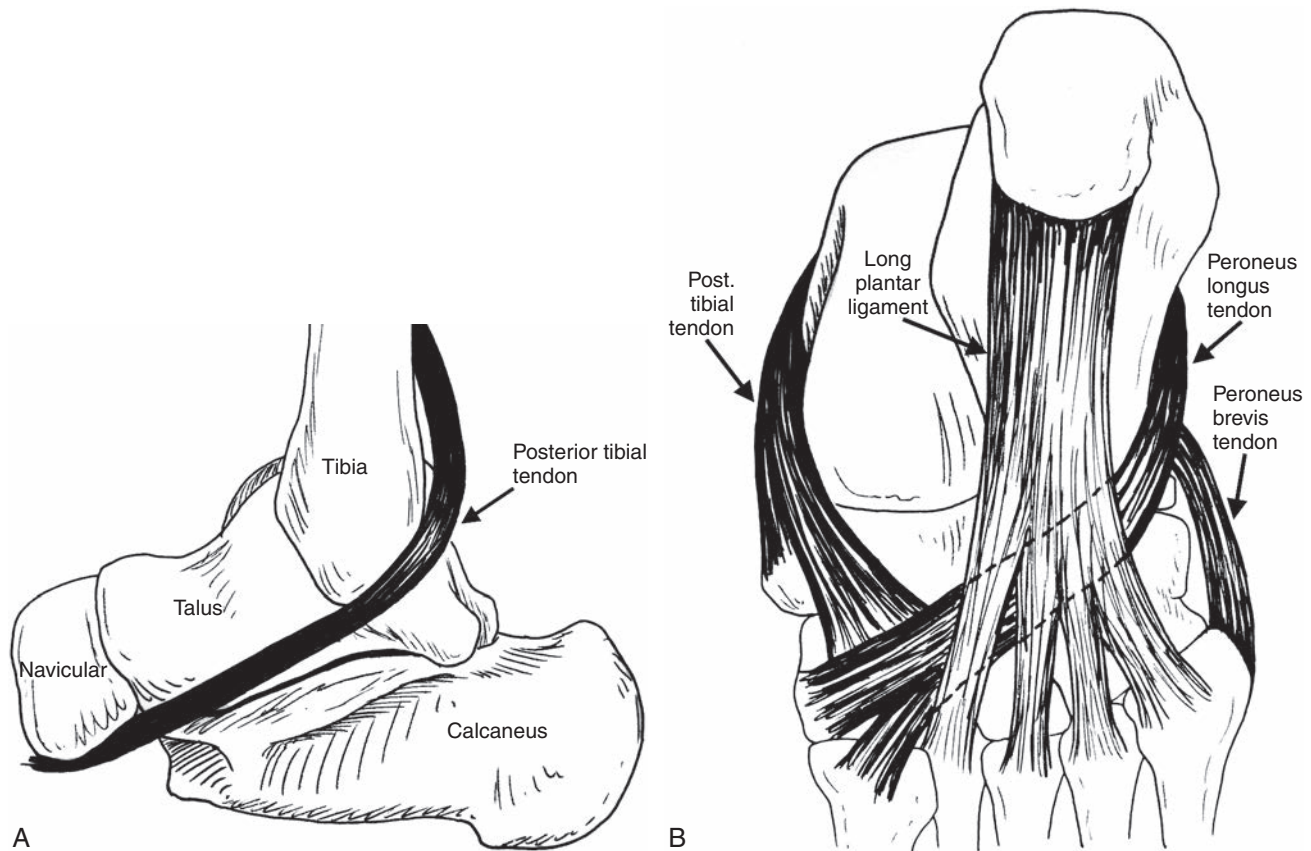


Fig. 16.12 Posterior tibial tendon anatomy. Note the attachment to the medial navicular, medial cuneiform, and lateral cuneiform that produces inversion, supination, and adduction.

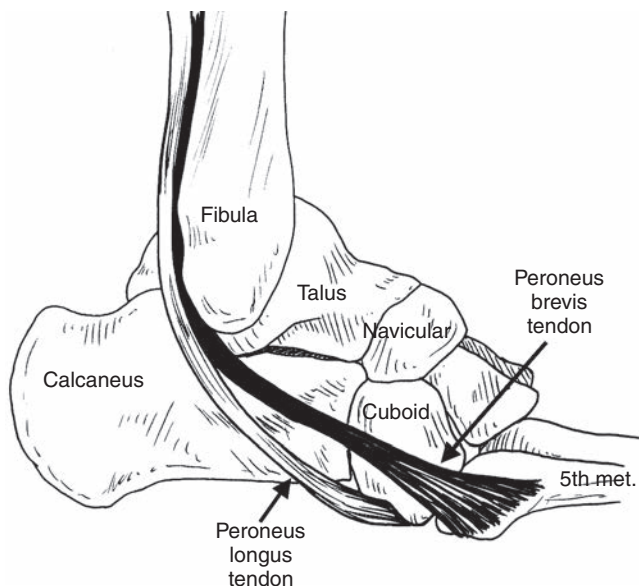


Fig. 16.13 Anatomy lateral ankle depicting peroneus longus and brevis tendons.

possibly lead to subtalar joint and hindfoot stiffness, flexor hallucis longus (FHL) entrapment, and medial sided ankle pain. Isolated fractures are rare but can occur with a direct trauma or a fall onto an axial loaded, rotated foot and can be associated with a talus fracture.¹¹ Misdiagnosis of this fracture can lead to a nonunion, tarsal tunnel syndrome, chronic FHL impingement, and progressive pes planovaglus deformity.¹²

Presentation and Physical Exam

The patient will often present with medial sided ankle pain, swelling, and ecchymosis. One should assess the flexor hallucis longus for direct trauma to the tendon or entrapment.

Imaging

Plain foot and ankle radiographs may show a normal Bohler's angle; however, a Harris axial x-ray can often assist with the diagnosis and show the fracture. CT is most useful to identify the fracture displacement and determine the need for surgical management.

Treatment

Operative management of these fractures should be considered in cases of any displacement.¹² Open reduction internal fixation of these fractures is best done with a direct medial approach and lag screw construct with a cannulated partially threaded screw fixation while protecting the FHL and neurovascular bundle.

Rehabilitation and Return to Sports

Rehabilitation will usually consist of nonweight bearing in a splint or cast for the first month, followed by a boot and nonweight bearing until 6 weeks postop. Thorough follow-up with axial Harris x-rays will allow one to determine union (or a CT scan if the plain imaging is inconclusive) and the beginning of weight bearing. The athlete should focus on joint mobilization and specifically focus on the FHL tendon to prevent adhesions.

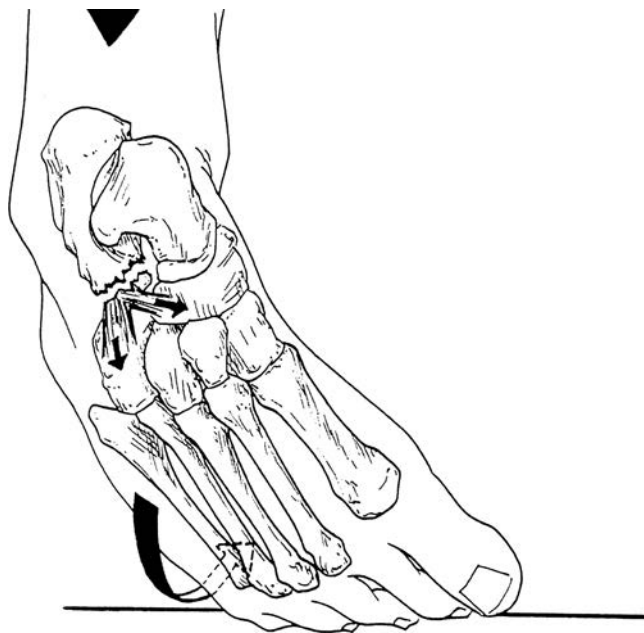


Fig. 16.14 Diagram of right foot demonstrating supination and inversion of hindfoot causing avulsion of anterior process of calcaneus with tension on bifurcate ligament.

Anterolateral Process of the Calcaneus Fractures

Mechanism of Injury

The anterior process of the calcaneus makes up 23% of fractures of the calcaneus.¹³ The injury occurs via an inversion injury mechanism with the ankle in plantarflexion, which can lead to an avulsion injury at the tip of the anterolateral process through tension of the bifurcate ligament that connects the anterior process to the cuboid and navicular (Fig. 16.14).

A second injury mechanism is forced dorsiflexion, eversion, abduction injury leading to a compression fracture of the anterior process between the cuboid and talus.¹⁴

Presentation and Physical Exam

When an athlete presents with pain over the hindfoot following a mechanism of injury as stated above, it is crucial to have a high level of suspicion to avoid delayed diagnosis of the fracture. Typically, the patient will present with ecchymosis and swelling with tenderness 2 cm anterior and 1 cm inferior to the ATFL (Fig. 16.15).¹⁴ Often pain may be produced by inversion stress through the subtalar joint (distracting the fragment). There may be instability of the transverse tarsal joint, which is tested by holding the heel stable with one hand and pronating and supinating the midfoot with the other hand (Fig. 16.16A and B).

Imaging

X-rays may present as negative if the fracture is nondisplaced. If displaced, the fracture through the tip of the anterolateral process of the calcaneus is best seen with the beam directed 20 degrees superior and posterior to the midportion of the foot (Fig. 16.17).¹⁵



Fig. 16.15 Clinical photograph of right foot demonstrating area of hindfoot that is tender with underlying anterior process fracture of calcaneus.

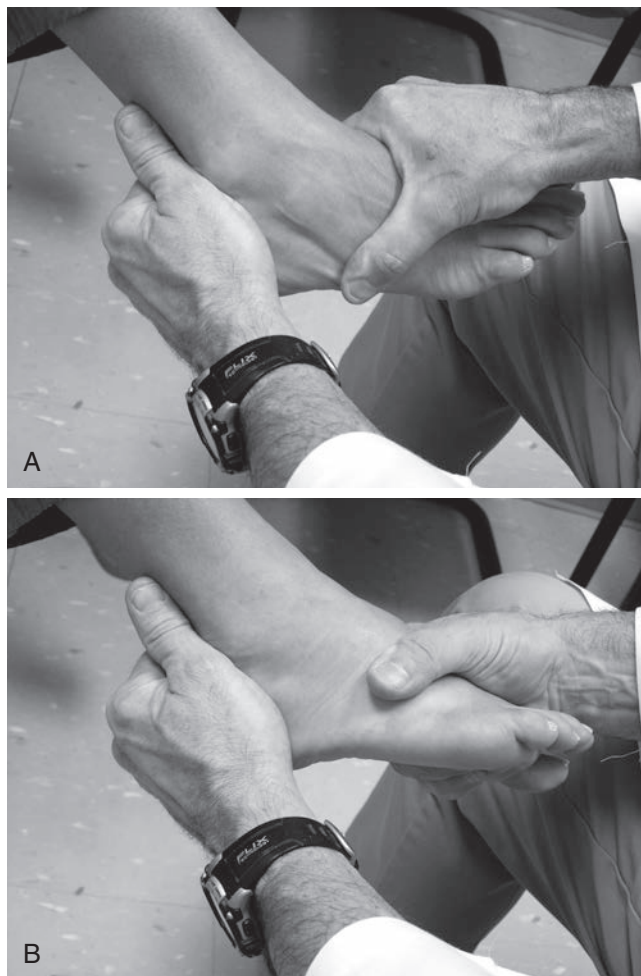


Fig. 16.16 Clinical photograph of right foot demonstrating assessment of transverse tarsal instability by stressing the hindfoot in (A) supination and (B) pronation.

A CT scan is useful to evaluate displacement of the fracture, and in severe cases, the advanced imaging is necessary to plan for surgical management. (Fig. 16.18A-C)



Fig. 16.17 Lateral radiograph of hindfoot demonstrating small anterior process fracture (arrows) of calcaneus.

Classification

Degan et al. proposed the following classification for fractures of the anterior lateral process of the calcaneus, which is useful in the diagnosis and treatment (Table 16.1).¹⁴

Treatment

Small nondisplaced fractures and those less than 2 mm can be treated in a cast and boot for 6 weeks of nonweight-bearing management until the fracture has fully healed.

In cases of chronic nonunion of the anterolateral process of the calcaneus, asymptomatic athletes are treated with observation only. For large fragments, greater than 1 cm or involving a significant portion of the articular surface, the fracture site is debrided and internal fixation is applied. For smaller fragments, the fragment is excised. The calcaneocuboid joint is inspected and debrided if necessary. The bifurcate ligament may be repaired back to the calcaneal process if any instability of the transverse tarsal joint exists (though this is not common).

For cases of malunited fractures, arthritic changes in the superior portion of the calcaneocuboid joint and/or the junction between the process of the calcaneus and navicular may exist. In these cases, a trial injection of cortisone in the calcaneocuboid joint and calcaneonavicular space may provide relief or help to establish the diagnosis of arthritic changes. Surgical treatment involves open resection of a portion of the anterolateral process of the calcaneus, trimming it back to a point at which a healthy calcaneocuboid joint is present. Recently, arthroscopic resection through a subtalar approach has been described.¹⁶ We prefer a limited debridement of the area and arthroscopic excision of the nonunion. If the fracture is quite large, a small open incision is made rather than arthroscopically removing a large amount of soft tissue to gain access to the anterior subtalar joint.

Rehabilitation and Return to Sports

In cases in which excision is required, boot immobilization and nonweight bearing are used for 2 weeks, followed by gentle active

range of motion (AROM) of the foot and protected weight bearing in the boot for an additional 4 weeks. General ankle rehabilitation then is begun, followed by sports-specific exercises.

Athletes with anterolateral process fractures treated by open reduction internal fixation (ORIF) or excision and ligament repair are placed in a nonweight-bearing boot for 6 weeks until healed. They then begin general ankle rehabilitation followed by sports-specific exercises. Return to sports usually occurs within 8 to 12 weeks.

OS PERONEUM AVULSION FRACTURES (SEE ALSO CHAPTER 8)

Mechanism of Injury

Os perineum fractures are a rare but often overlooked diagnosis in athletes that can be associated with complete rupture of the peroneus longus tendon and requires a high index of suspicion. The os peroneum is a sesamoid bone that can be found in the peroneus longus tendon, often found adjacent to the plantar-lateral aspect of the cuboid. It has been reported in 5% to 26% of the population.¹⁷ It is important to recognize this injury early to plan for management of an associated tendon injury.

Presentation and Physical Exam

Patients will often present after an inversion ankle injury with a swollen, painful, and occasionally ecchymotic foot and ankle. Clinicians will observe point tenderness plantar-laterally along the inferior lateral cuboid area and pain with inversion and eversion of the hindfoot.

Imaging

Plain x-rays will reveal a proximally migrated bony fragment that may be misread as a “benign” avulsion, or different accessory ossicles. Radiographic features of an acute fracture of the os peroneum will demonstrate the presence of “cortical discontinuity with nonsclerotic margins” and a “pieces of a puzzle” appearance.¹⁸ A normal-appearing os perineum will appear as an oval, well-corticated ossicle near the calcanealcuboid joint.

MRI imaging is essential to evaluate the appearance and possible retraction of the peroneus longus tendon and rule out any other surrounding soft tissue injury. A retracted os peroneum is highly suggestive of a complete peroneus longus rupture.

Treatment

Treatment for an os peroneum fracture can include nonoperative management when the os perineum fracture is minimally displaced.¹⁷ Excision of the ossicle with primary repair of the peroneus longus or tenodesis of the peroneus longus to the brevis is most often the preferred method for managing this injury in the athlete. Repair or tenodeses helps the athlete to avoid loss of eversion strength, and first metatarsal plantarflexion strength. Postoperatively, the ankle is immobilized in a cast nonweight bearing for 3 weeks in slight eversion, followed by a boot or cast for another 3 weeks. Therapy will begin at 6 weeks with the focus on ankle and subtalar mobilization.

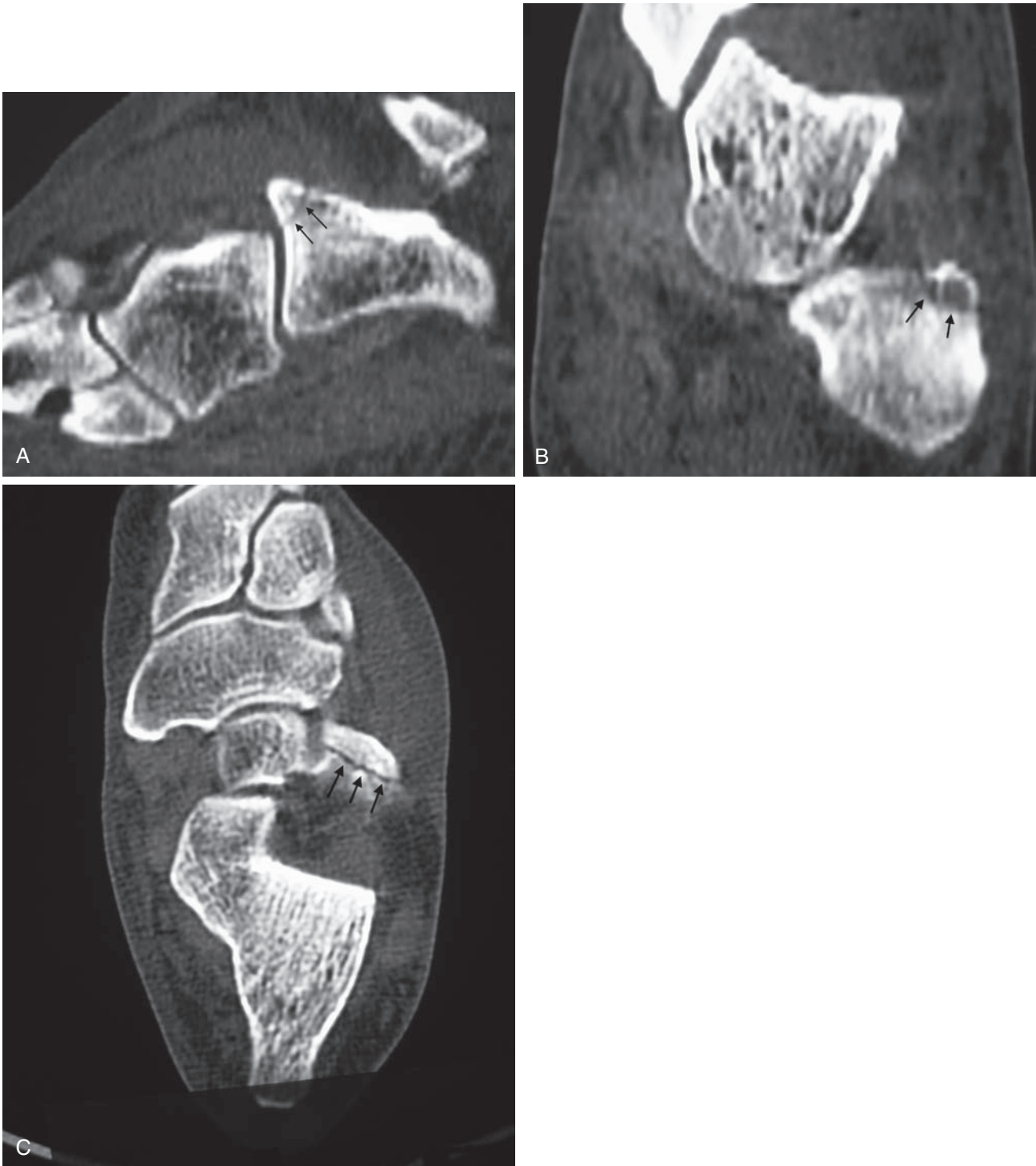


Fig. 16.18 (A) Sagittal reconstruction, (B) coronal, and (C) axial computed tomography view of occult anterior process fracture (*arrows*) of calcaneus. Plain radiographs did not reveal fracture, but athlete had tenderness over anterior process.

TABLE 16.1 Classification of Fractures of the Anterior Process of the Calcaneus ¹⁴	
Type I	Nondisplaced tip avulsion
Type II	Displaced avulsion fracture not involving the calcaneocuboid articulation
Type III	Displaced, larger fragments involving the calcaneocuboid joint

OCCULT FRACTURES OF THE CUBOID

Mechanism of Injury

Midfoot injuries, specifically of the cuboid, are rare in the athlete, but can often be overlooked as a midfoot sprain. They can be observed in two different variants in athletes: as either a capsular avulsion injury or a compression cuboid injury. Capsular

avulsion injuries are seen during an adduction/inversion injury when the patient lands in plantarflexion. This is common in sports like basketball, volleyball, and dancing.¹⁹ In an avulsion cuboid injury, the calcaneocuboid capsule and plantar calcaneocuboid ligament are torn producing a small avulsion fragment off the plantar posterior cuboid. Alternatively, a compression injury can occur, creating a nutcracker mechanism of acute compression of the cuboid between the anterior process of the calcaneus and the base of the fourth and fifth metatarsal, accompanied by medial column distraction injury of the mid-foot. It is important to rule out injuries to the Chopart joint in this situation, which can be seen by widening of the talonavicular joint.^{19,20}

Presentation and Physical Exam

The patient will typically present with lateral foot pain (just proximal to the insertion of the peroneus brevis), ecchymosis, and swelling with difficulty walking, especially with plantarflexion.

Imaging

X-rays can show a small lateral cuboid avulsion injury or will reveal compression fractures (Fig. 16.19). CT scan imaging is best used to more clearly visualize the fracture, its pattern and its extent. CT imaging can help establish the extent of fracture and the amount of joint depression that can be involved with the impaction injury.

Treatment

Surgery will be required when there is significant shortening of the cuboid in an impaction injury. Surgery is also required if displacement results in destabilization of the lateral column. This can be performed with mini-fragment screws with or without bone graft and plating to restore the lateral column.²¹

Conservative management is typically used to treat most types of painful cuboid injuries. Cast and boot treatment are preferred with pain and a return to sports is advised when the patient is pain free.

OCCULT FRACTURES OF THE TALUS

Frequently missed or misdiagnosed fractures after an ankle sprain comprise a large number of occult talus fractures. These can be classified into dorsal talar injuries, lateral process fractures, posterior process fractures, and compression injuries of the talus and osteochondral fractures of the talus.

Talonavicular Avulsion Injuries

Mechanism of Injury

Talonavicular avulsion injuries are a commonly missed diagnosis in patients presenting with an acute ankle sprain. The dorsal talonavicular capsule ligament will pull off with a forced plantarflexion moment (Fig. 16.20). The dorsal force can be created



Fig. 16.19 Oblique radiograph of foot demonstrating small fracture of cuboid.

by the opposing force of the bifurcate ligament attachment on the lateral navicular and the posterior tibial tendon attachment to the medial navicular. This injury occurs commonly in sports in which a sudden change of direction is followed by the athlete planting his foot, decelerating, and twisting a plantarflexed foot to reaccelerate and push off. The force of the posterior tibial tendon on the navicular may produce an avulsion at its insertion (Fig. 16.21).

Presentation and Physical Exam

The patient will often complain of pain dorsally at the navicular just lateral to the tibialis anterior insertion. Dorsal mid-foot swelling and ecchymosis can be seen along with pain with resisted dorsiflexion and active inversion/passive eversion (Fig. 16.22).^{22–24} Frequently a bony firmness can be noted over the dorsal navicular or talar head.

Imaging

Plain radiographs will show an avulsion fracture of the dorsum of the navicular or talar head on the lateral view (Fig. 16.23). CT and MRI can be useful in chronic cases of nonunion or to rule out a concomitant navicular stress fracture (Figs. 16.24 and 16.25).

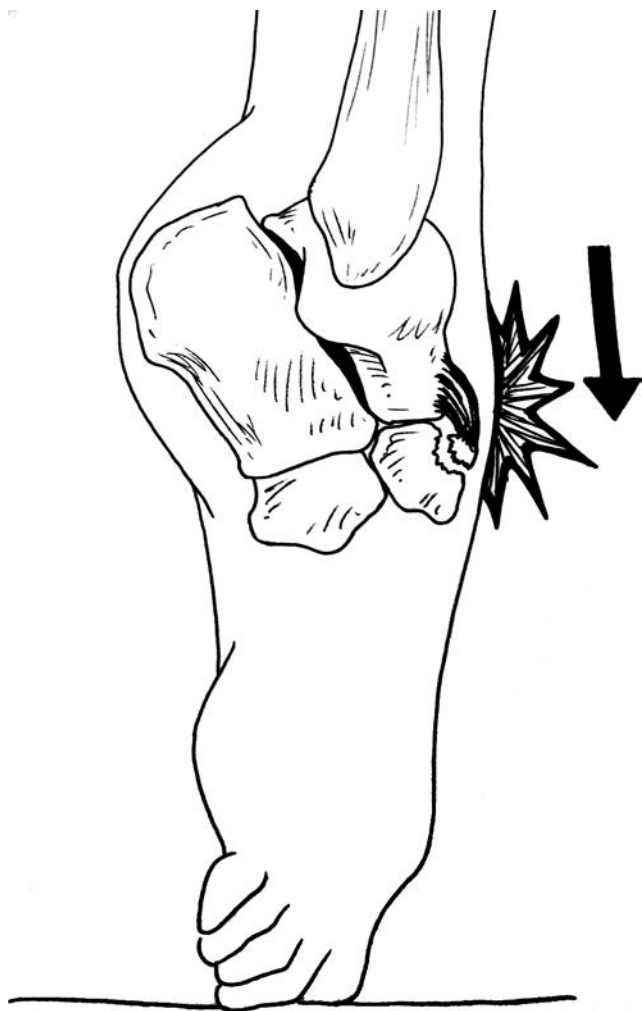


Fig. 16.20 Fracture of navicular caused by plantarflexion of foot and ankle with avulsion of dorsal fragment.

It is important to differentiate this injury from os supranavicular, which represents a normal variant.

Treatment

Management of a talonavicular avulsion fracture is typically treated conservatively in a short-leg cast for 3-4 weeks non-weight bearing, followed by a boot. If there is a painful nonunion, excision of the fragment can be considered or ORIF if the fragment is greater than 5 mm.

Rehabilitation and Return to Sports

Rehabilitation will usually begin at 4-6 weeks after the injury with a focus on edema control, range of motion (ROM), proprioception, and progressive resisted exercises (PREs) (especially the posterior tibial tendon). Running is done first, initially on an underwater treadmill or AlterG, and jumping activities are added next, followed by sports-specific exercises. The athlete may return to practice/play on successful completion of the program (6 to 10 weeks post injury).

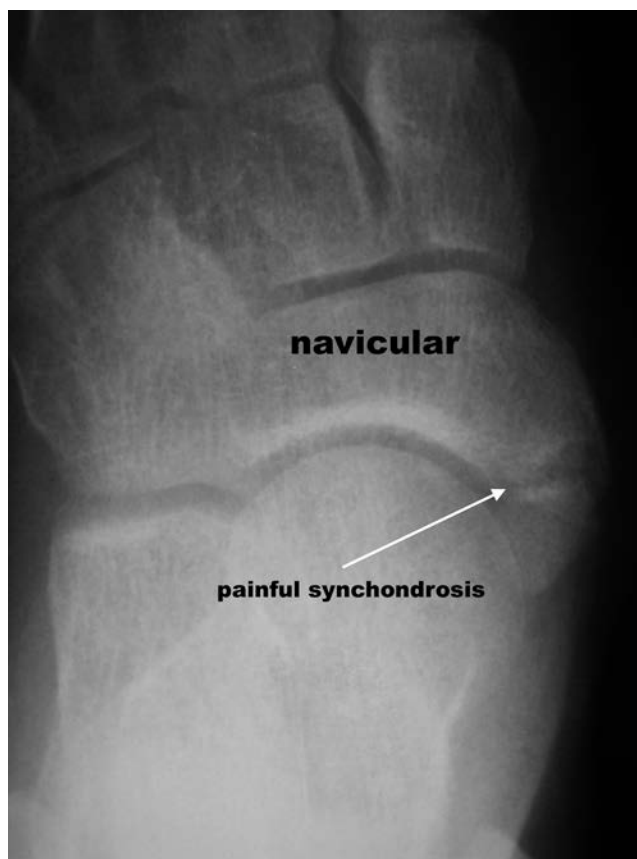


Fig. 16.21 Anterior-posterior radiograph of athlete's foot depicting painful medial accessory navicular attached by synchondrosis.



Fig. 16.22 Clinical examination of athlete's foot depicting area of pain noted on foot with dorsal avulsion fracture of navicular.

Posterior Talus Fractures/Posterior Impingement Syndrome (see also Chapters 2 and 24)

Mechanism of Injury

Posterior impingement syndrome can result from trauma, overuse or abnormal anatomy and is seen with chronic forceful plantarflexion leading to pain at the os trigonum, especially when the os trigonum is large. However, other causes of



Fig. 16.23 Radiographic findings of dorsal, wafer-like fracture with acute injury.

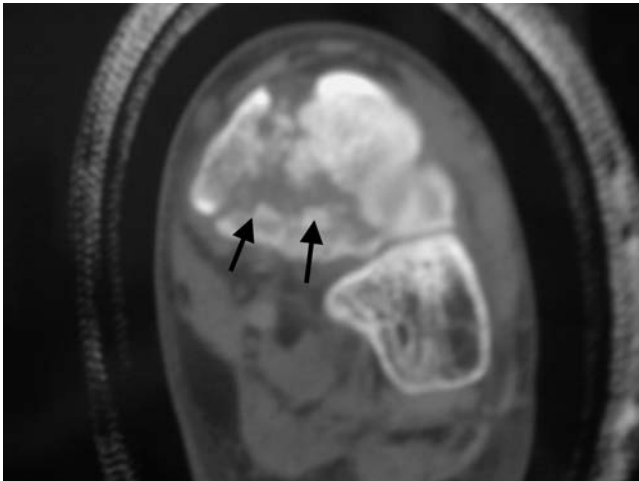


Fig. 16.24 Computed tomography (coronal view) demonstrating more involved navicular body fracture with comminution.

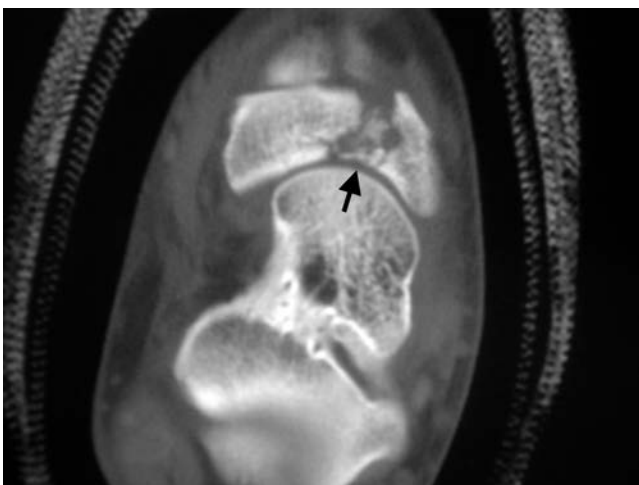


Fig. 16.25 Computed tomography (axial view) demonstrating navicular body fracture with displacement.

posterior impingement can be caused by prominent Stieda process, laxity in the ATFL after an ankle sprain (due to excessive anterior sliding of the talus when performing a toe rise), poor-fitting dance shoes or bad technique in the *demi-pointe* or *en pointe* positions. These injuries are most often seen in sports involving repetitive plantarflexion such as ballet, soccer, or jumping and landing sports like gymnastics, volleyball, and basketball. Occasionally a large Stieda process can be fractured during a plantarflexion inversion injury (sometimes referred to as a Shepherd's fracture) (Fig. 16.26).

A forced ankle dorsiflexion and pronation injury such as forced planting of the foot backward with force applied (Fig. 16.27, A) or weight applied to the back of a dorsiflexed ankle at the bottom of a scrum or pileup (Fig. 16.28) may produce an avulsion fracture of the posteromedial tubercle by traction on the posterior deltoid ligament (see Fig. 16.27B).^{9,25}

Presentation and Physical Exam

Athletes will complain of posterior ankle pain that is often provoked by forced plantarflexion or pain with push-off, jumping, and landing. Dancers will often note that going into the *demi-pointe* or *en pointe* position is very painful and limiting. It is important to differentiate this pain from tenosynovitis of the flexor hallucis longus, although they can exist in concert with each other.

The posterior talar process can be best palpated between the Achilles and peroneal tendon. The “pinch test” posteromedially or posterolaterally just posterior to the ankle (Fig. 16.29) will produce pain. The posterior impingement test (Fig. 16.30A and B) will produce pain and possibly clicking. Manipulation of the great toe, producing stretch on the flexor hallucis longus, also may produce posterior ankle pain. A diagnostic injection into the posterior capsule or posterior process can be helpful in differentiating symptoms.

Imaging

Radiographs will often be normal except to demonstrate a large os trigonum. CT scans can be helpful to demonstrate the bony fragments or multiple os trigonum fragments. MRI is the best imaging to evaluate the soft tissue and show edema at the synchondrosis and possible disruption. It is also helpful to assess any FHL pathology as well as concomitant ankle or subtalar joint pathology that may need to be addressed at the time of surgery.

Treatment

The authors feel an arthroscopic approach is the best way to treat posterior impingement. The arthroscopic technique can be performed supine or prone. The supine position technique was first described by R. Ferkel and Marumoto, with excision of the os trigonum through subtalar arthroscopy. The AOFAS scores improved from 45 to 86.²⁶ Since that time, several hundred os trigona have been removed with similar results at Southern California Orthopedic Institute. van Dijk has shown the success of the prone arthroscopic excision as well with AOFAS score improvement from 75 to 90 at 36 months.^{27–29}

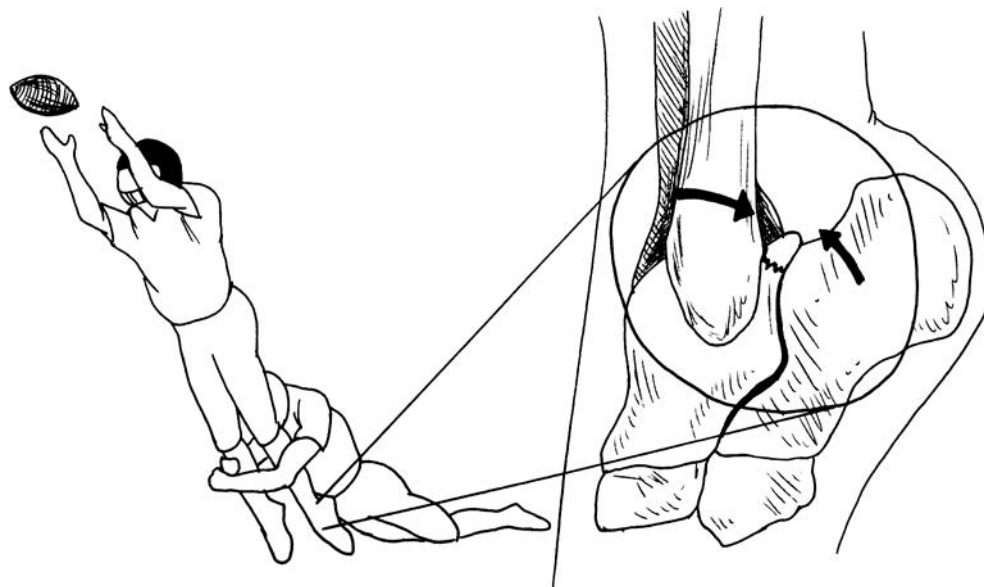


Fig. 16.26 Common mechanism for posterior process fracture with compression of posterior process between calcaneus and posterior tibia in severe plantarflexion of ankle.

For acute nondisplaced fractures of the Stieda process or os trigonum, immobilization in a boot/cast and limited weight bearing may lead to healing in 4 to 6 weeks.

For large displaced fractures that extend into the talar body region, internal fixation through a posterolateral or posteromedial approach with cannulated 4.5 screws or headless screws is indicated. An unusual cause of posterior impingement is a bipartite talus.^{30,31}

Rehabilitation and Return to Sports

After internal fixation of talus fracture, the patient should be in cast followed by a boot and remain nonweight bearing for 6 weeks. When the fracture is healed, progressive weight bearing and ankle rehabilitation is begun, followed by sports-specific exercises. Range of motion and strengthening of the FHL is emphasized.

After os trigonum excision, the authors will typically keep the patient in a splint the first week followed by boot or cast with the progression to weight bearing by weeks 2 to 3. The with gentle ROM of the ankle and subtalar joint is allowed out of the boot and physical therapy is commenced. A typical time to return to sports after excision is 6–8 weeks.

Lateral Process of the Talus Fracture (see also Chapter 6)

Lateral processes of the talus fractures are commonly misdiagnosed and often mimic the clinical symptoms of ankle sprains with up to 59% missed initially.^{32,33} It is estimated that they are present in 0.86% of all lateral ankle sprains.³⁴

Mechanism of Injury

Lateral processes of the talus fractures are most commonly seen in the snowboarder, accounting for 2.5%–6.3% of all snowboarding injuries.³⁵ Typically “snowboarder’s fracture” will occur with a dorsiflexed and inverted force on the ankle and talus. Recent studies suggest that external rotation

applied to a dorsiflexed inverted foot (Fig. 16.31) may produce a force to the lateral process and result in a fracture.^{36,37} Both the body and the snowboard act as a lever arm on the ankle and talus, with the skier’s leading foot injured most frequently.³⁸

Presentation and Physical Exam

The athlete will typically present with history of a rotational injury to the ankle with the complaint of lateral ankle pain, most often localized 1 cm distal to the tip of the lateral malleolus that is increased with weight bearing.³⁹ Often swelling and ecchymosis will be exhibited.

Imaging

Obtaining anterior-posterior (AP), lateral, and oblique ankle x-rays may show an avulsion-type fragment laterally (Fig. 16.32A) or be negative if the fracture is nondisplaced (Fig. 16.32B and C). The mortise or Broden’s view are felt to be best to visualize these fractures.

The authors believe that CT scan imaging should be ordered if there is a suspicion of a lateral process fracture. This study can aid in identification of lateral talar process fractures, aiding in sizing and surgical planning (Fig. 16.33). MRIs can also be ordered to rule out chondral pathology in the talus.

Classification

Two commonly used classifications for lateral talus process fractures exist: the Hawkins classification:¹⁶ type 1—simple two-part fracture, type 2—comminuted fracture, and type 3—avulsion fracture of the anterior inferior process; and the McCrory and Bladin classification, recently modified by Tinner and Sommer (Fig. 16.34):^{40,41} Type I—small, minimally displaced, extra-articular chip fracture, type II—simple large fragment fractures, and type III—comminuted fractures. It is also important to assess the fracture displacement: minimal displacement, <1 mm

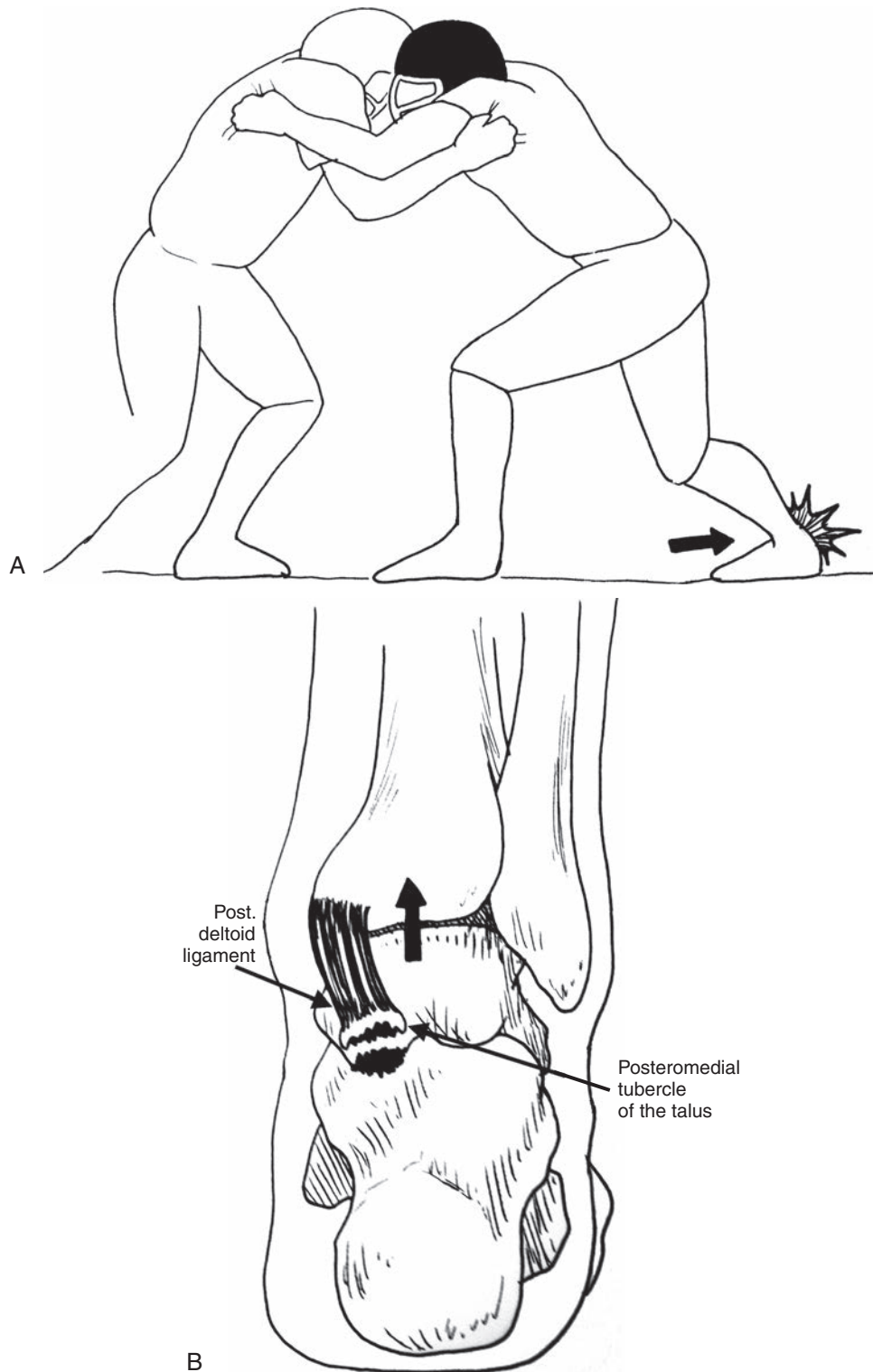


Fig. 16.27 (A) Lateral view of posterior talus process fracture caused by forced dorsiflexion of the ankle against a planted foot. (B) Posterior view, showing avulsion forces produced by the posterior deltoid ligament on the posterior medial tubercle of the talus.

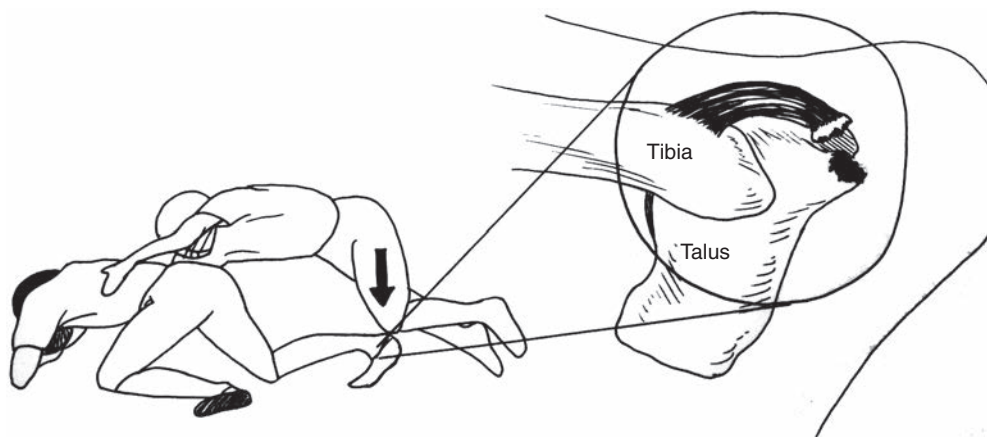


Fig. 16.28 Posterior talus process fracture caused by force on the back of the ankle, causing avulsion of the posterior talar process through tension on the posterior deltoid ligament.



Fig. 16.29 Clinical demonstration of “pinch test.” Compression of posterior process fracture of talus (os trigonum) in athlete just behind ankle from medial and lateral sides cause pain.

articular step off and <2 mm gap and distinguish it from displaced fractures, >1 mm articular step off and 2 mm or greater gap.

Treatment

Acute, nondisplaced talar lateral process fractures can be treated with immobilization in a splint followed by a boot, nonweight bearing for 4 to 6 weeks.

For smaller displaced fractures conservative treatment with boot or cast immobilization and nonweight bearing also is indicated. Early excision of displaced small fragments and progressive weight bearing can be considered.

For larger displaced fractures (> 2 mm) and/or with joint surface irregularity greater than 1 mm, open reduction internal fixation with headless screws through a curvilinear subfibular approach is preferred. Ankle arthroscopy should be considered prior to the open approach to evaluate the cartilage of the talus and address any chondral pathology. Some clinicians have proposed performing an arthroscopic reduction and internal fixation for this type of fracture using cannulated screws.⁴² Postoperative involves nonweight-bearing boot

immobilization with immediate, gentle AROM until healing is accomplished.

For chronic cases (previously undetected) or cases of nonunion after immobilization, treatment of large fragments (greater than 1 cm) or fragments involving the articular surfaces require debridement and/or internal fixation and can be addressed either open or through a combination of ankle and subtalar arthroscopy.

Rehabilitation and Return to Sports

Athletes with intra-articular lateral talus fractures requiring internal fixation should start gentle AROM exercises of the ankle and subtalar joints during this healing and nonweight-bearing phase to maintain joint mobility (typically around 3 weeks post-op). Full weight bearing can begin around 6 weeks post op with progression out of the boot by 8 weeks. When all fractures are healed, more aggressive ankle rehabilitation begins, followed by sports-specific exercises and return to sports. Initially, pool therapy can be very helpful as well.

OSTEOCHONDRAL LESIONS OF THE ANKLE

INTRODUCTION

Chondral and osteochondral lesions of the ankle continue to pose a challenge to orthopedic surgeons due to the anatomically limited joint surface, high degree of joint contact forces, difficulties of easily accessing the tibiotalar joint, and problems with cartilage healing. Osteochondral lesions of the talus (OLT) remain one of the most common lesions of the ankle and are often sequelae of ankle sprains and fractures. Despite significant advances in cartilage restoration therapy over the last two decades, the heterogeneity of injury patterns and location, depth, and acuity of treatment make development of a rational and evidence-based treatment algorithm difficult. This review will attempt to briefly summarize the historical treatment options for sport-related OLT and osteochondral lesions of the tibial plafond (OLTTP), and outline newer options for cartilage restoration.

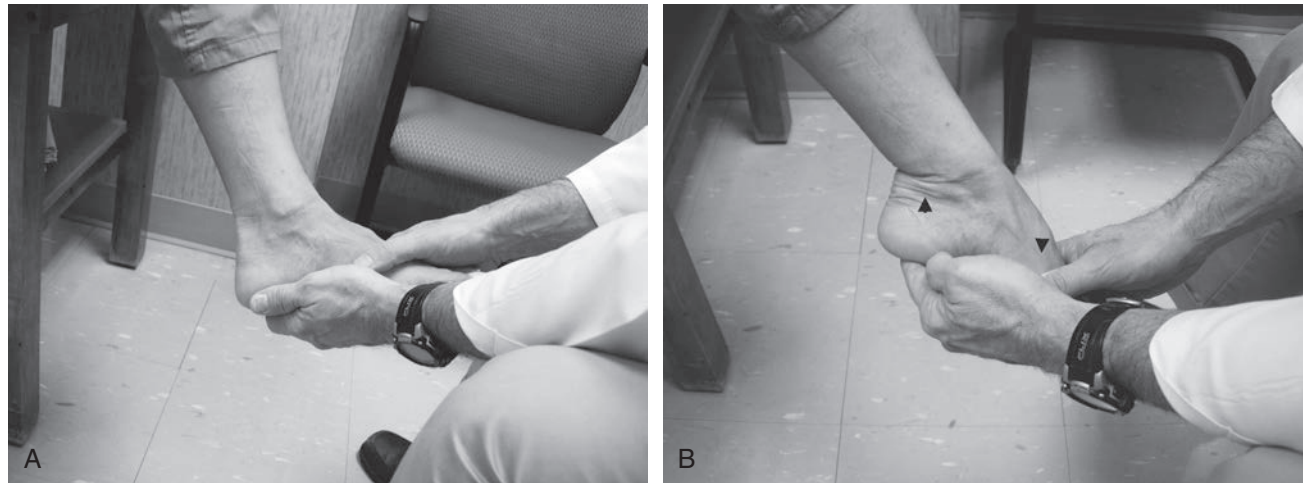


Fig. 16.30 Clinical demonstration of “posterior compression test.” Forced maximal plantarflexion (A and B) of ankle produces pain in athlete with posterior process fracture (os trigonum).

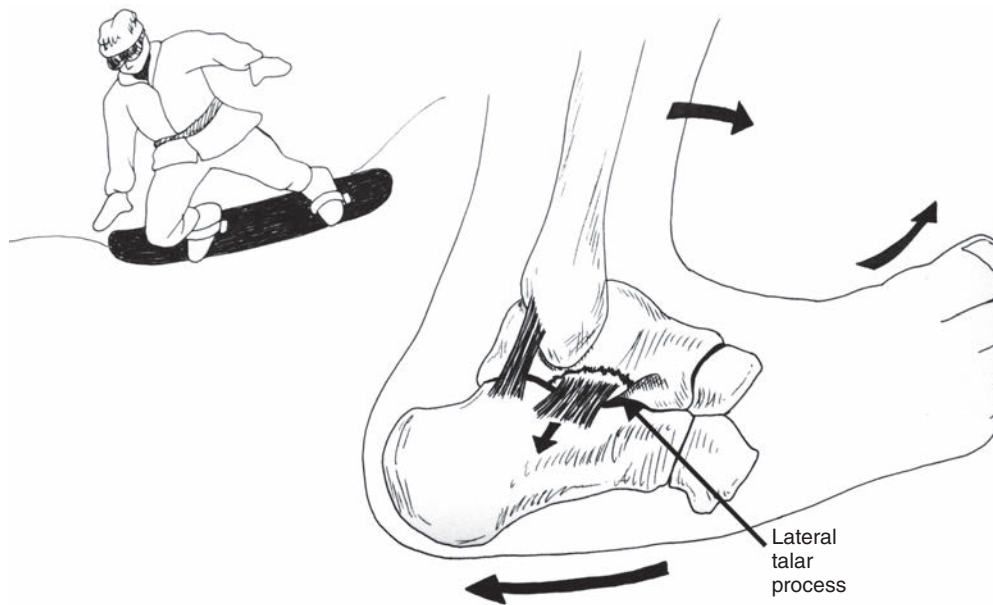


Fig. 16.31 Diagram noting mechanism for lateral process fractures of talus. Forced external rotation with the ankle in dorsiflexion and inversion results in a lateral process fracture.

HISTORY AND INCIDENCE

The description of OLT originated in a discussion of trauma-related loose bodies in the ankle by Alexander Monro in 1856.⁴³ The term *osteocondritis desiccans* was first termed by König when describing osteochondral lesions in the knee.⁴⁴ At that time, König proposed a theory of spontaneous necrosis as the cause of these lesions. In an effort to describe the etiology of the lesion, Barth proposed that the lesion was secondary to a fracture of the intra-articular surface in 1898.⁴⁵ Shortly thereafter, Kappis first applied the term *osteocondritis dissecans* to the ankle joint.⁴⁶ In 1959, Berndt and Harty suggested that the lesion had a traumatic etiology and coined the term *transchondral fracture of the talus*.⁴⁷ Since that time, nomenclature and discourse have centered on the role of trauma, subchondral bony vascular insult, and cartilage genetics in the development of OLT.⁴⁸

Given the tenuous blood supply of the talus, the genetic predisposition for OLTs, the 10% incidence of nontraumatic bilateral OLTs, and the association of OLTs with systemic inflammatory conditions, thrombotic conditions, and chronic steroid use, the pathogenesis of OLTs is clearly multifactorial.⁴⁹ It is estimated that there are over 1 million sports-related ankle sprains annually in the United States, and the estimated incidence of articular cartilage injury and OLT associated with ankle sprains are found to be between 6.5% and 50%.^{48,50,51} In an active military population, incidence was estimated to be 27 per 100,000 person-years.⁵² In a separate study, the mean age of patients with OLT was between 20 and 30 years old, with a slight predilection for men.⁵³

Medial OLTs are seen most frequently and are associated with trauma in 61% to 70% of cases. These lesions are also observed to have greater depth in comparison to lateral lesions. Lateral



Fig. 16.32 Lateral process fracture of talus. (A) Anterior-posterior (AP) radiograph of ankle demonstrating lateral process fracture (*arrows*) noted just inferior to the tip of the fibula. (B) Lateral and AP radiographs of athlete with lateral process fracture that was not able to be visualized on x-rays.

lesions are associated with trauma in up to 98% of cases.⁵⁰ In contrast to the relatively high incidence of OLT, OLTP are rare. In one cohort of more than 880 consecutive ankle arthroscopies, OLTP represented less than 3% of articular cartilage injuries, with less than 1% having a bipolar lesion of both the tibia and talus.⁵⁴

MECHANISM

The talus is anatomically remarkable in that it demonstrates a high percentage of articular cartilage surface area, a high-level

of congruence, and a thinner cartilage relative to that of the knee.⁵⁰ While these characteristics lend a low risk (2%) of primary osteoarthritis when compared to the hip or knee, they do predispose the talus to significant acute chondral injuries and progression to posttraumatic arthritis.⁵⁵

Multiple studies have examined the application of both compressive and shear force to the joint and its relation to osteochondral lesion location. Berndt and Harty reproduced lateral talar dome lesions via an inversion force applied to a dorsiflexed foot with the tibia internally rotated. Similarly, medial dome lesions were reproduced by applying an inversion force to a

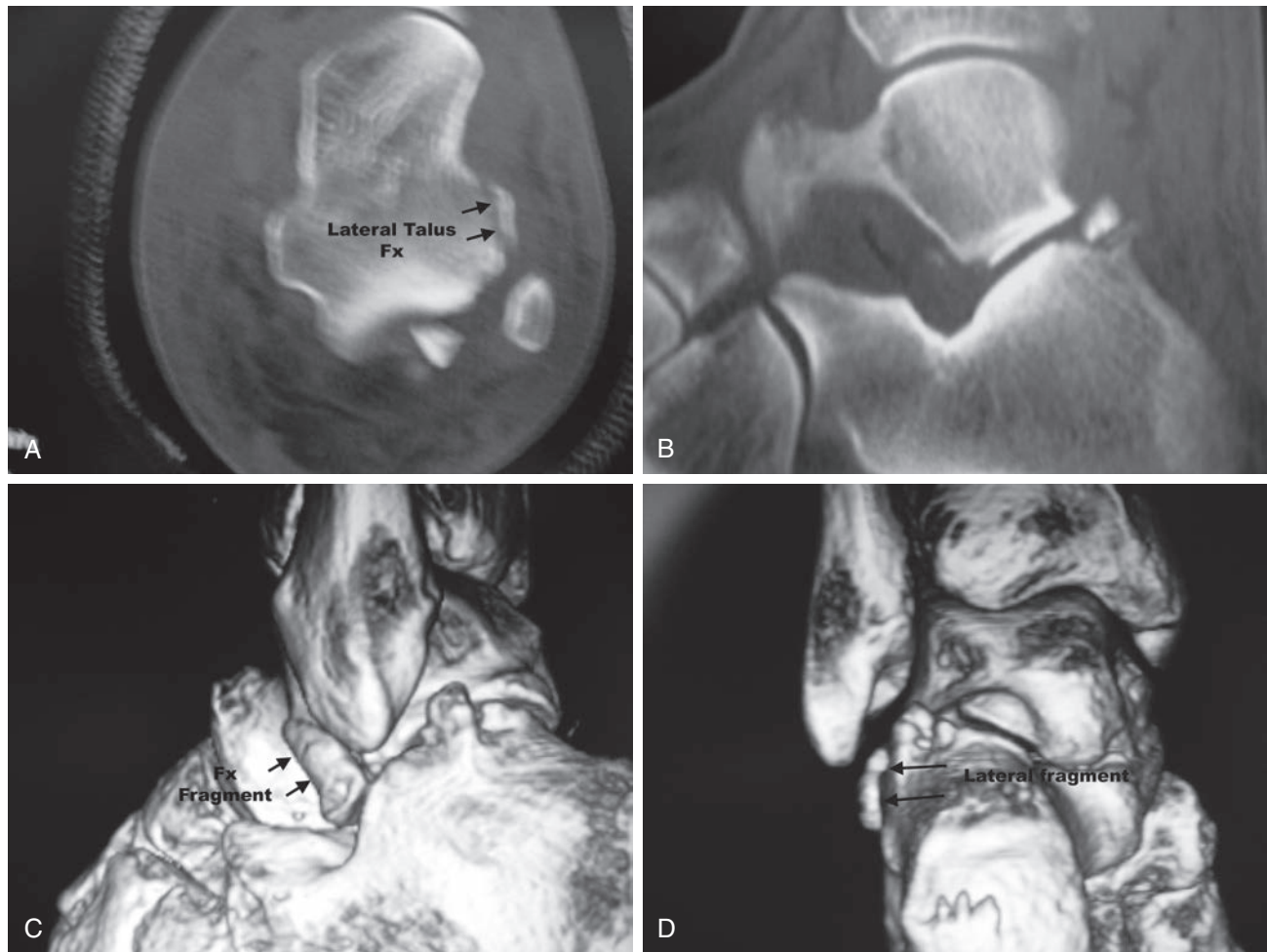


Fig. 16.33 Axial computed tomography scan of talus demonstrating lateral process fracture that was not identified on ankle radiographs in athlete.

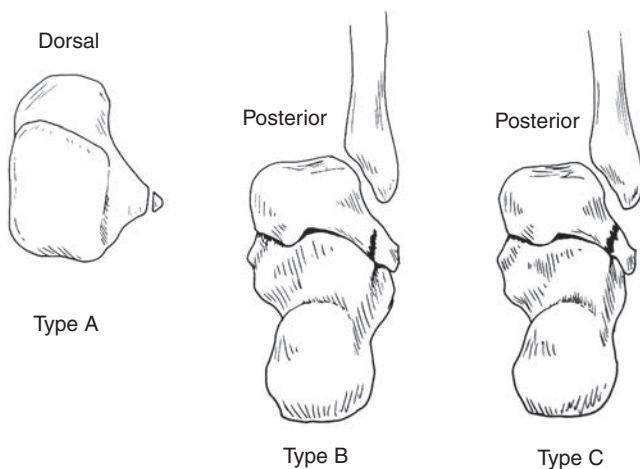


Fig. 16.34 Funk classification for lateral process fracture talus. Type A involves only a small avulsion fragment, type B involves only the talocalcaneal joint, and type C involves both the talocalcaneal and talofibular articulations.

plantarflexed foot with tibial external rotation. Other investigators have postulated that significant inversion of a plantarflexed foot would result in the application of distracting forces to the lateral talus, as well as compressive forces to the medial talus.⁵⁶

In contrast, posteromedial lesions are seen in the setting of ankle plantarflexion, while anterolateral lesions occur with inversion and ankle dorsiflexion. Yao and Weiss deduced that lateral OLTs were secondary to eversion dorsiflexion ankle injuries with the tibia in an internally rotated position.⁵⁷

Advanced imaging modalities may demonstrate subchondral cyst formation, a known sequela of OLT. Subchondral cysts are believed to develop as a result of a crack in the articular cartilage that allows synovial fluid to leak into the underlying bone. The increased hydrostatic pressure with continued walking forces synovial fluid into the potential space of the talus fissure and creates a cystic cavity. Egress of fluid from the joint space and enlargement of the developing cyst may be aided by damaged cartilage functioning as a one-way valve. The cartilaginous “valve” allows the flow of fluid from the joint space into the subchondral bone but not in the opposite direction (Fig. 16.35).⁵⁸

DIAGNOSIS AND EVALUATION

Examination

Distinguishing patients with osteochondral lesions from those with uncomplicated ankle sprains can be challenging, and the diagnosis is often delayed by the tendency to treat routine ankle

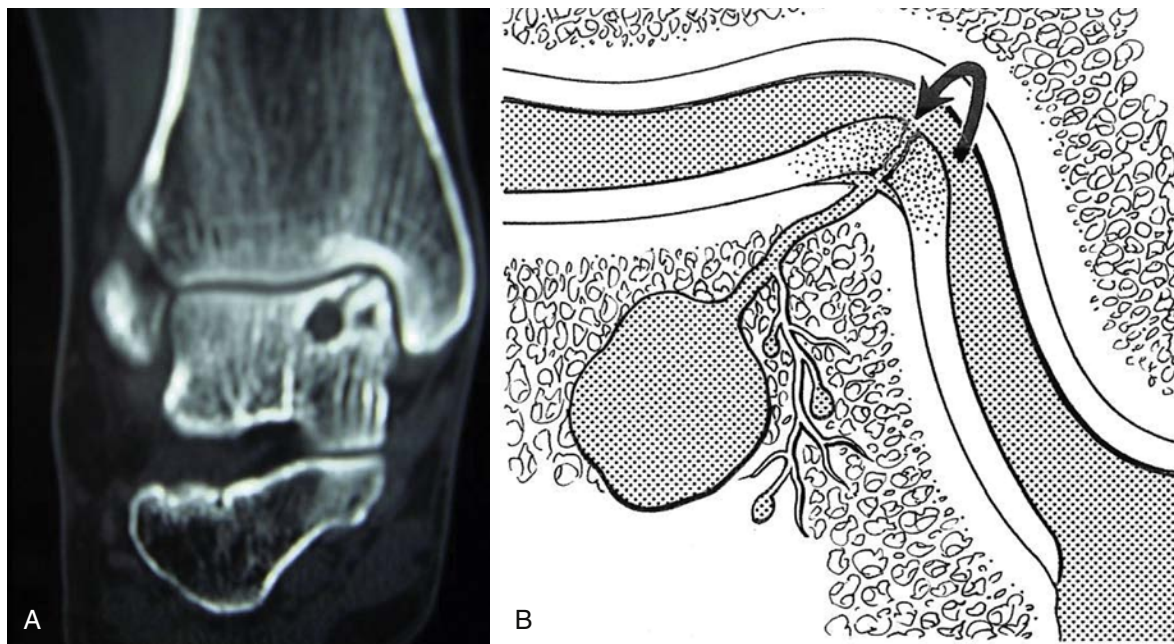


Fig. 16.35 Cystic osteochondral lesion of the right talus. (A) Coronal CT scan showing an oval shaped subchondral cystic lesion in the talar body. Note the line of communication from the cyst within the joint. (B) Accompanying diagram showing presumed mechanism of cyst formation that occurs with the synovial fluid leaking through the crack in the articular cartilage and forming a cyst in the bone. (Reprinted with permission from Ferkel RD: *Foot and Ankle Arthroscopy*, 2nd edition, Wolters Kluwer, 2017. Illustration by Susan Brust, MS.)

sprains conservatively. Many patients will describe a history of a sports-related injury with failure to improve after conservative management. Furthermore, patients are often unable to return to sports-related activities, and may report a history of an aching pain, ongoing edema, and prolonged stiffness after an otherwise uncomplicated injury. Given the variable presentation of ankle sprains, identifying OLTs requires a high index of suspicion in patients who fail to improve with standard conservative treatment. While physical exam findings are generally nonspecific, the most common exam finding may be anterior joint line tenderness with the foot plantarflexed. Mechanical symptoms, such as locking or catching, can occur in the setting of a loose or displaced osteochondral fragment. Given the association of OLTs with ankle sprains, ankle instability should be routinely evaluated with anterior drawer and inversion and eversion testing. In addition, subtalar motion should be evaluated to assess for bony coalition as a possible cause of recurrent instability. Moreover, the patient's alignment needs to be carefully assessed.

Imaging

Despite the routine necessity of advanced imaging for diagnosis, all patients should always undergo standard weight-bearing AP, mortise, and lateral plain radiographs with optional stress radiographs for those with possible ankle instability. Weight-bearing views can be helpful in evaluating ankle alignment and joint space collapse. Roughly 50% of lesions will be noted on radiographic examination.⁵⁹ Most patients with an OLT will require advanced imaging, either an MRI and/or CT scan. CT and/or MRI should be considered for patients with continued pain after ankle trauma or any mechanical symptoms. Zinman et al. reported on 32 patients with osteochondritis dissecans of the talar dome and

found CT scans to be superior to x-rays for both diagnosis and follow-up.⁶⁰ Although there remains debate regarding selection of follow-up imaging, either CT or MRI should be considered for diagnosis of OLT. In cases of known OLT, we favor a thin-slice CT scan with low-dose radiation protocol, as this modality allows for improved definition of the lesion and has been shown to correspond more accurately to the size of the lesion at the time of arthroscopy.⁶¹ In cases of ongoing pain without a clear diagnosis, MRI should be considered in order to evaluate bone marrow edema and to more accurately characterize overlying cartilage. MRI also reveals other soft-tissue abnormalities that can be associated with an OLT. However, it should be noted that MRI can overestimate the size of the OLT in a significant number of cases.⁴⁸ Novel imaging techniques, including T2rho cartilage mapping, show promise but are currently used experimentally and are not considered standard of care.

Classification

The diagnosis and evaluation of OLT have improved dramatically with advances in diagnostic imaging and operative arthroscopy. To further characterize and specify imaging findings, several classification and staging systems have been devised. In 1959, Berndt and Harty used x-ray analysis and specimen dissection to help develop a radiographic-based classification, which has been appended to include six stages of lesions visualized on x-ray imaging.⁴⁷ Stage 0 describes a lesion that is not seen on x-ray imaging but is visualized on MRI. Stage I denotes a nondisplaced compression fracture. Stage II is a partially detached osteochondral fracture. Stage III is a completely detached but nondisplaced fragment. Stage IV is a completely detached and displaced fragment. Lastly, stage V describes lesions with deep cystic change.⁶²

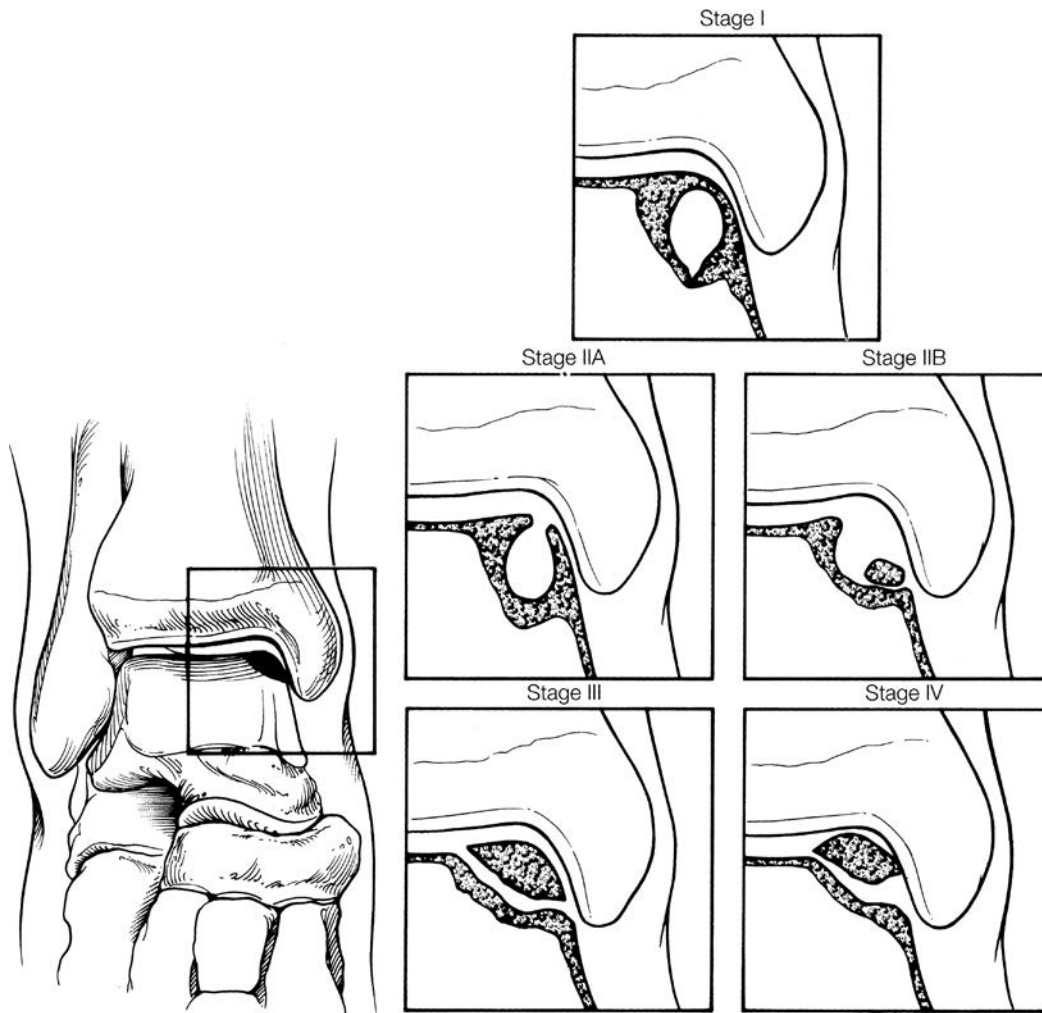


Fig. 16.36 Ferkel-Sgaglione CT classification for osteochondral lesions of the talus. (Reprinted with permission from Ferkel RD: *Foot and Ankle Arthroscopy*, 2nd edition, Wolters Kluwer, 2017. Illustration by Susan Brust, MS.)

MRI staging has been described by several different authors. Beginning in 1999, Hepple developed an MRI-based classification system wherein stage I described articular cartilage damage alone, stage IIA described cartilage injury with underlying fracture and surrounding bony edema, stage IIB described cartilage injury with underlying fracture without surrounding bony edema, stage III described detached and nondisplaced osteochondral fragment, stage IV described a detached and displaced osteochondral fragment, and stage V described an osteochondral lesion with subchondral cyst formation.⁶³ Anderson and colleagues applied MRI to modify the radiographic classification established by Berndt and Harty, allowing for further characterization of fragment separation, underlying bone marrow edema, and subchondral cyst formation.⁶⁴

Ferkel and Sgaglione developed a four-stage classification system based on two-plane CT imaging.⁶⁵ Stage I describes a cystic lesion within the intact dome of the talus seen in all views, stage IIA describes a cystic lesion communicating with the talar dome surface, stage IIB describes an open articular surface lesion with an overlying nondisplaced fragment, stage III describes a nondisplaced lesion with lucency, and stage IV describes a displaced fragment. This classification appeared to

correlate more accurately with arthroscopic findings and treatment outcome (Fig. 16.36).

Drawing on arthroscopic findings, Pritsch described a classification system based on the graded appearance of the overlying cartilage.⁶⁶ In this system, arthroscopic findings were stratified into three surgical grades: I) intact, firm, and shiny articular cartilage; II) intact but soft cartilage; and III) frayed cartilage. Citing poor correlation between x-ray imaging and arthroscopic findings, investigators asserted that arthroscopic evaluation and the intraoperative appearance of lesions in cases of OLT were the most valuable determinants of treatment.

Using data derived from 80 subjects treated for OLT at the Southern California Orthopedic Institute between 1985 and 1994, Cheng, Ferkel and Applegate developed a more detailed classification system based on the arthroscopic appearance of articular cartilage. In this study, investigators compared preoperative x-ray imaging and CT or MRI with intraoperative findings.⁶⁷ Using this classification, grade A describes articular cartilage that is smooth, intact, but soft or ballotable; grade B describes a rough surface; grade C describes fibrillations or fissures; grade D describes the presence of a flap or exposed bone; grade E describes a loose, nondisplaced fragment; and grade F describes a displaced fragment (Fig. 16.37).

All of the above-mentioned staging systems are helpful in the diagnosis and treatment of OLT (Table 16.2). However, each of these staging systems must be used together with the patient's symptoms and accurate sizing measurements to determine appropriate treatment.

TREATMENT

Treatment of an OLT must be customized to each unique patient. Treatment options are determined based on the patient's symptoms and activity level, stage and size of the lesion, and whether it is an acute or chronic cartilage injury. A summary of

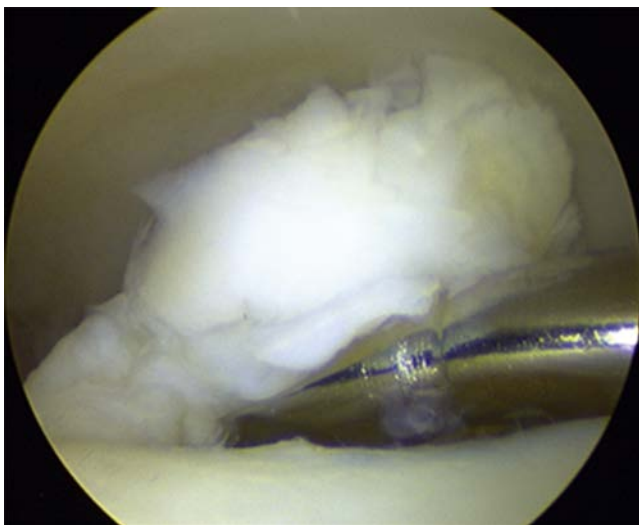


Fig. 16.37 Palpation of an arthroscopic grade D osteochondral lesion of the talus in a right ankle. The probe is brought in from the anteromedial portal while visualizing through the anterolateral portal.

specific evidence-based recommendations has been detailed by Kraeutler et al.⁵⁹ In addition, there are a number of risk factors for failed cartilage repair that are listed in Table 16.3.

Acute Injury Treatment

In the setting of acute OLT, identification of the lesion with x-ray imaging may be possible. CT or MRI may be used, if needed, to further characterize the lesion and determine radiologic staging. Although there is a paucity of evidence-based data regarding the role of weight-bearing or nonweight-bearing ambulation in the treatment of OLT, nonoperative treatment with immobilization and nonweight bearing is commonly employed in cases of acute nondisplaced OLTs.^{6,68–71} To assess evolution of the acute lesion, serial imaging, including x-ray, CT, or MRI, is routinely performed. If the acute lesion is displaced and purely chondral in nature, the lesion should be treated by arthroscopy and excision with subsequent debridement, drilling, and possible microfracture to stimulate angiogenesis and formation of fibrocartilage.⁶⁹ If the displaced chondral piece has adequate underlying bone, the fragment should be reattached with bioabsorbable pins, K-wires, or headless screws. Cases that do not heal should be treated as chronic cases.

Chronic Injury Treatment

Numerous operative techniques have been described to treat OLT. These treatment strategies can be classified as cartilage repair, cartilage regeneration, cartilage replacement, or prosthetic replacement (Table 16.4).⁷² Full-thickness loss of articular cartilage presents a significant therapeutic challenge, as only 50% of patients report “good” or “excellent” results with conservative treatment.^{4,6,73} A combination of x-ray, CT scan, and/or MRI evaluation along with arthroscopic assessment

TABLE 16.2 Classification of Osteochondral Lesions Based on Radiographs, MRI, CT, and Arthroscopy

Radiographic Classification Berndt and Harty ⁴⁷	MRI Revised Classification Anderson ⁶⁴	CT Classification Ferkel & Sgaglione ⁶⁵	Arthroscopic Grade Based on Articular Cartilage Ferkel et al. ⁶⁷
Stage 0 Not visible on radiographs	Stage I Subchondral trabecular compression—plain radiograph normal, positive bone scan; marrow edema on MRI	Stage I Cystic lesion within dome of talus, intact roof on all views	Grade A Smooth, intact cartilage, but soft and ballotable
Stage 1 Trabecular compression of subchondral bone	Stage IIA Formation of subchondral cyst	Stage IIA Cystic lesion with communication to talar dome surface	Grade B Rough articular cartilage surface
Stage 2 Partially detached osteochondral fragment	Stage II Incomplete separation of fragment	Stage IIB Open articular surface lesion with overlying nondisplaced fragment	Grade C Articular cartilage has fibrillations and fissures
Stage 3 Detached and nondisplaced osteochondral fragment	Stage III Unattached, nondisplaced fragment with presence of synovial fluid around fragment	Stage III Nondisplaced lesion with lucency	Grade D Articular cartilage flap present or bone exposed
Stage 4 Detached and displaced osteochondral fragment	Stage IV Displaced fragment	Stage IV Displaced fragment	Grade E Loose, un-displaced fragment
			Grade F Loose, displaced fragment

of the cartilage surface allows accurate staging. Furthermore, arthroscopic evaluation may aid in identifying appropriate surgical management. While excision alone has demonstrated only moderate patient satisfaction outcomes (40% good to excellent result), excision and curettage yield 78% good to excellent results.⁶ Satisfaction outcomes were further improved with the addition of drilling or microfracture to excision and curettage, yielding 86% good to excellent results.^{4,73–76}

Several techniques have been used for penetrating subchondral bone, including resecting sclerotic subchondral bone, drilling through the subchondral bone, abrading the articular surface, and creating small-diameter defects with microfracture pics. These techniques are called *bone marrow stimulation* (BMS) and are reparative procedures that are the most common operative treatment choice for an OLT. The procedure is done arthroscopically in the following steps: (1) debridement of the unstable cartilage and necrotic bone; (2) debridement of the calcified layer; and (3) penetration of the subchondral bone plate (SBP) using a thin microfracture pick or small diameter K-wire. Penetration of the subchondral bone disrupts subchondral blood vessels, leading to the formation of a fibrin clot, and ultimately to fibrocartilage tissue forming on the surface. Studies have demonstrated that undifferentiated mesenchymal cells are released from the underlying marrow. These cells then differentiate into chondrocytes and chondroblasts that ultimately form the fibrocartilage cells that infiltrate the articular surface.⁷⁷

In the chronic setting, the lesion should be carefully probed. If the lesion is intact, antegrade or retrograde drilling through the talus can be done. Fixation techniques with absorbable pins, K-wires, or screws can be used in loose fragments with healthy-appearing articular cartilage. Superficial displaced fragments with unhealthy articular cartilage are typically excised.

Once excised, curettage, antegrade or retrograde drilling, and/or microfracture are performed. In large lesions, the entire cystic region, including the deformed loose cartilage, should be curetted out and the area debrided, drilled, and microfractured. While some asymptomatic lesions can be followed over time without intervention, bone graft with autograft or demineralized bone matrix (DBM) allograft should be used on cysts >5 mm.^{78,79}

AUTHORS PREFERRED TREATMENT

Acute

Principles of treating acute osteochondral lesions are different than those for chronic lesions. In an acute OLT, the first priority is to characterize the lesion as soon as possible. X-ray, MRI, and/or CT are helpful to further visualize the exact size and location and amount of bone that may or may not be associated with the chondral fragment. Overall steps for treatment of an acute OLT are summarized in [Box 16.1](#).

Acute anterolateral osteochondral lesions are usually displaced and oftentimes upside-down in the joint. We have coined the term for these lesions: “Lateral Inverted Osteochondral Fractures of the Talus (LIFT).”⁸⁰ These lesions must be debrided as soon as possible and are relative surgical emergencies. Often, these patients are very swollen after a severe ankle sprain, and clinical exam and MRI will reveal a complete tear of the anterior talofibular and calcaneofibular ligaments. Initially these patients are treated in a bulky dressing and posterior splint, and elevated for 1–2 weeks until the swelling is diminished enough to proceed with surgery.

LIFT lesions are best approached arthroscopically in the supine position, with the arthroscope in the anteromedial portal, inflow posterolaterally, and instrumentation anterolaterally. Loose lesions need to be flipped back to the proper position and then reduced and fixated if enough bone is attached to the cartilage. If there is minimal or no bone, the lesions need to be excised. With maximum plantarflexion, sometimes these lesions can be fixated with absorbable pins or headless screws arthroscopically, if the abnormality is anterior enough to clear the tibial plafond. An absorbable pin made of polyglycolic acid can be inserted in a triangular fashion, so that three pins are usually used to stabilize the displaced fragment.

More often, acute anterolateral osteochondral lesions are not amenable to arthroscopic fixation alone. In this situation,

TABLE 16.3 Risk Factors for Failed Cartilage Repair

Age	Chondrocyte function decreases as patients get older
Obesity	Inferior functional outcomes associated with elevated BMI
Smoking	Overall negative influence on cartilage repair
Activity level	After a certain threshold, an increase can have negative effect on articular cartilage
Inflammatory disease	Current treatments could affect cartilage health

TABLE 16.4 Treatment Strategies for Osteochondral Lesions of the Talus: Repair, Replace or Regenerate

Repair	Regeneration	Replacement
Marrow stimulation (microfracture)	Autologous chondrocyte implantation (ACI)	Osteochondral allograft transfer (OAT)
Retrograde drilling	Matrix-induced chondrocyte implantation (MACI) Bone marrow-derived cell transplantation	Osteochondral allografting Particulated juvenile cartilage allograft transplantation Micronized cartilage matrix Metallic cap implant

(Modified from Dekker TJ, Dekker PK, Tainter DM et al.: Treatment of osteochondral lesions of the talus a critical analysis review. *JBJS Reviews* 2017;5:e4-e13.)

BOX 16.1 Treatment of Acute OLT

1. Palpate the lesion with a small joint probe
2. Excise chondral fragments with little or no bone and drill/microfracture the base
3. Reattach loose osteochondral fragments with absorbable pins, K-wire, or screws
4. If lesion is displaced, reduce with probe or grasper gently and temporarily fixate with K-wire, then firmly fixate with absorbable pins, screws or K-wire

a Broström approach is performed with a longitudinal incision to expose the torn lateral ankle ligaments, and then the foot is plantarflexed with inversion and an anterior drawer to display the lesion site more clearly. It is critical to remove the displaced fragment and keep it in sterile saline on the back table in the operating room (OR) until it is ready to be reattached. The osteochondral lesion bed needs to be debrided, with good sharp margins. Rarely is a bone graft necessary. The acute OLT is then reduced and secured with pins or screws (Fig. 16.38)

PEARL**Pearls:**

1. The osteochondral fragment absorbs water and is always bigger than the area from where it came. The fragment needs to be carefully trimmed and sized to fit into the osteochondral bed flush and not be proud.
2. A small burr is used to remove a little bit of bone to help with resection and anatomic reduction of the OLT fragment.
3. Place two guide pins or K-wires into the fragment on the back table to use as joysticks to help with reduction and fixation, so that the fragment can be anatomicallly reduced and not fall to the floor accidentally.

Chronic

Most chronic OLT are unstable and require excision. However, more recently, a technique termed “lift and fill” has been developed to reattach these fragments, if the osteochondral fragment is felt to be viable and not avascular.⁸¹ Other techniques have also been used, such as ORIF, with insertion of pins or cortical bone pegs. Kumai et al. showed good clinical outcomes in 24/27 patients at a follow-up of 7 years.⁸²

Principles of treatment of chronic OLT are described in Box 16.2. The choice of treatment for an osteochondral lesion is multifactorial and is determined by factors such as size, depth, location, containment, number, and age of defects, subchondral bone quality, and prior surgery. The primary indicator of success with marrow-stimulating techniques is determined by the size of the lesion. In the past, studies have indicated poor results in lesions that measure $>150 \text{ mm}^2$ in size, or 15 mm in diameter.^{83,84} However, more recently, Ramponi et al. have shown that the optimal size should not exceed 107.4 mm^2 .⁸⁵ Another important factor that determines prognosis is containment, and Choi et al. have shown worse results with uncontained lesions than when the lesions are contained.⁸⁶ Treatment decisions can be developed through an algorithm or diagram (Fig. 16.39).

In general, BMS is still the treatment of choice, using small-diameter microfracture picks or small-diameter K-wires,

as previously described. Chen et al. found that drilling 6 mm deep produced better fill and quality of cartilage than 2 mm deep.⁸⁷ They used a nano-fracture device of 1-mm diameter to stimulate a better healing response. They also found that microfracture results were similar to drilling to the 2-mm depth.

The technique we prefer is with the patient in the supine position, using a nonsterile thigh support and lateral post, with the pad at the foot of the bed removed. This gives excellent access to the anterior and posterior aspects of the ankle. The patient is paralyzed by the anesthesiologist after a popliteal block has also been performed. The tourniquet is inflated at the surgeon's discretion and soft-tissue distraction is applied. Standard anteromedial, anterolateral, and posterolateral portals are established, as previously described (Fig. 16.40).⁴⁸ The entire ankle is debrided and then the osteochondral lesion is palpated. If it is unstable, which is frequently the case in chronic situations, it is excised using different-angled cup and ring curettes, shaver, and a grasper. Good, 90-degree vertical walls are established to hold the clot. Drilling of the lesion is accomplished using a 0.045 or, in some cases, a 0.035 wire throughout the osteochondral lesion. The wire should be irrigated at all times to prevent heat necrosis. Small-diameter microfracture picks can also be selectively used on a limited basis, to penetrate the subchondral bone. If the lesions are more central or posterior, a MicroVector™ aiming device can be used to assist with accurate drilling of the lesion (Fig. 16.41).

Van Bergen et al. described 50 patients followed for a mean of 141 months and reported overall functional scores were 88/100.⁸⁸ Numerous other investigators have also showed good functional outcomes following BMS, with short to mid-term evaluations of athletic populations.^{51,89}

Although successful outcomes have been attained with BMS, deterioration of the regenerated fibrocartilage can occur. Ferkel et al. found 35% of patients had deterioration in their result within 5 years after BMS.⁷⁵ Lee et al. showed only 30% of patients who had BMS had good graft integration on second-look arthroscopy.⁹⁰ Although van Bergen et al. showed good long-term results, one-third of their patients progressed by one grade of arthritis severity on regular x-rays.⁸⁸ It is unclear why the joint deteriorates, but it may be due to the result of a combination of mechanical and biologic factors. Biologic agents that can reduce inflammation and give a good chondrogenic biologic response would be a big advantage to patients with BMS, and will be discussed later in the chapter. Hurley conducted a literature review of 57 studies with 3072 ankles for patients who had marrow stimulation technique for an OLT.⁹¹ The review found a high rate of return to sports of 87%, and the mean time to return to play was 4.5 months. However, there was a significant deficiency in the reported rehabilitation protocols, as well as poor documentation in returning back to play criteria.

OSTEOCHONDRAL LESIONS OF THE TALUS

ALTERNATIVE TREATMENTS

When BMS fails after a good treatment technique or when the OLT is greater than 107.4 mm^2 (particularly with cystic lesions $>6 \text{ mm}$), alternative treatment modalities need to be

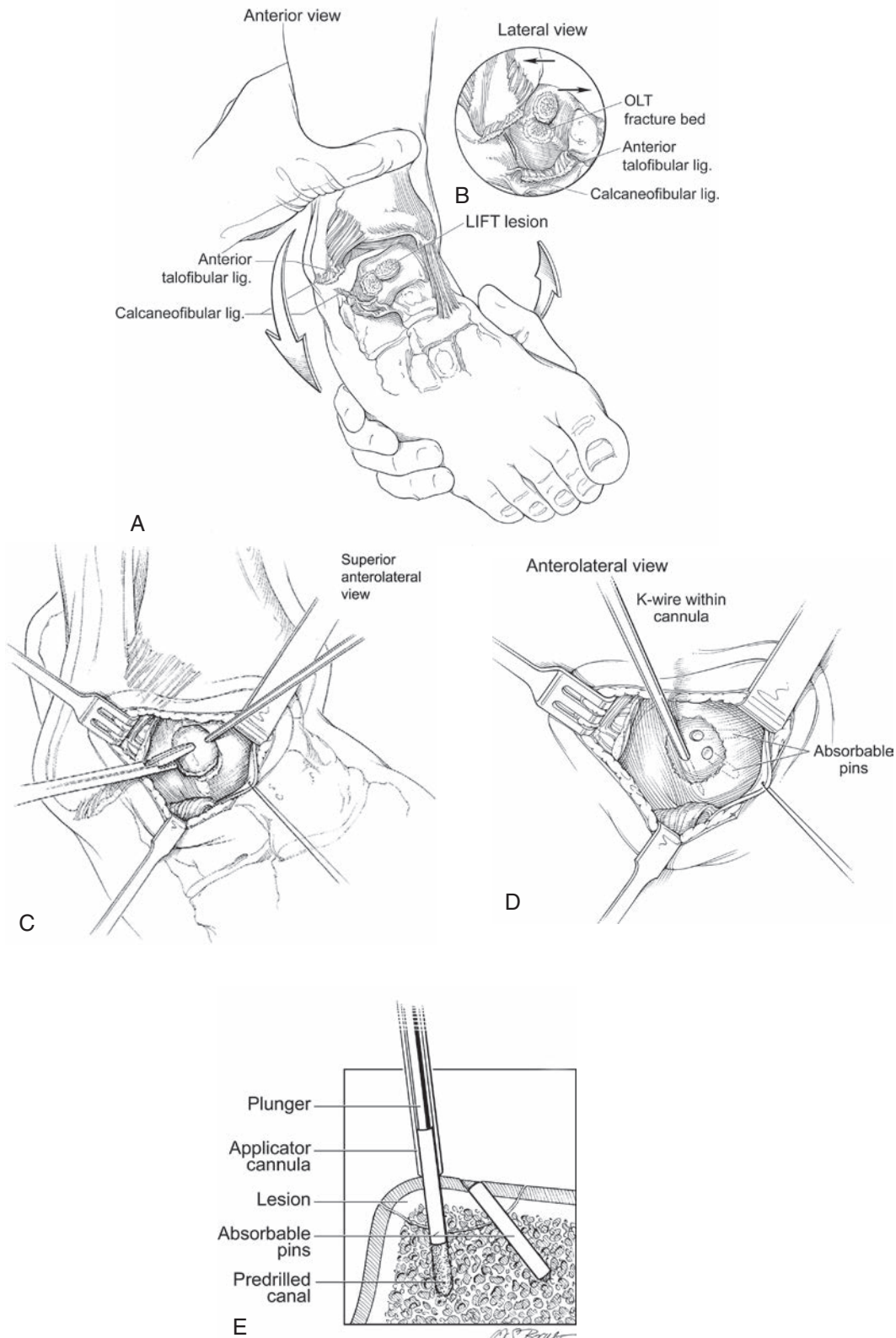


Fig. 16.38 Treatment of an acute osteochondral lesion of the talus (LIFT lesion). (A) A Broström incision is made and the torn lateral ligaments are identified. The foot is then inverted and plantarflexed, and an anterior drawer is applied to access the lateral OLT. (B) Lateral view showing the lateral OLT brought out anteriorly. (C) Oblique view demonstrating the lateral OLT is reduced with a grasper and temporarily fixated with K-wires. (D) Absorbable pins are sequentially inserted to reattach the lateral OLT. (E) Cross-sectional diagram shows the position of the global pin stabilizing the OLT while a second pin is inserted. (Reprinted with permission from Ferkel RD: *Foot and Ankle Arthroscopy*, 2nd edition, Wolters Kluwer, 2017. Illustration by Susan Brust, MS.)

BOX 16.2 Treatment of Chronic OLT

1. Probe with small joint probe
2. Evaluate with palpation whether lesion is loose; if there is any question, use dilute methylene blue to detect staining around the lesion that would suggest whether it is loose or not
3. Drill the lesion if it is not loose
4. Fixate the lesion if loose and if articular cartilage and underlying bone are healthy, with or without inserting bone graft, using absorbable pins, K-wires, or screws
5. Excise the lesion if loose or displaced, and then drill/microfracture the base
6. Bone-graft large cystic areas if cartilage is intact; otherwise, curette the cyst out and drill or abrade the bone (bone grafting of open defects can be considered)

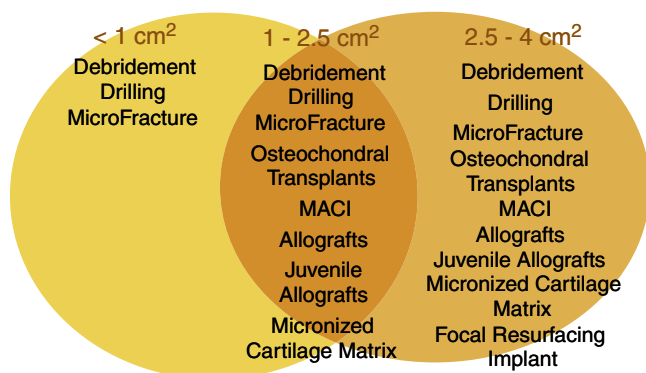
Treatment Options for Cartilage Defects

Fig. 16.39 Treatment options for cartilage defects based on the size of the osteochondral lesion.



Fig. 16.40 Standard setup for ankle arthroscopy in a right ankle. Note the arthroscope is in the anteromedial portal, the shaver is in the antero-lateral portal, and dedicated inflow is placed through the posterolateral portal, with soft-tissue distraction applied via a strap.

considered, such as osteochondral transplants, membrane/matrix autologous chondrocyte implantation (MACI), juvenile allografts, micronized cartilage matrix, or, rarely, a focal resurfacing implant. These alternatives will be discussed in the following sections.



Fig. 16.41 Use of the MicroVector™ drill guide. (A) The MicroVector™ drill guide has two articulating arms with different sized K-wire cannulas. In addition, an offset guide allows a precisely patterned series of drill holes to be made. (B) Transsalleolar drilling with the arthroscope posteromedially into an osteochondral lesion of the medial talar dome. The probe is retracted as the K-wire enters through the distal tibia before going into the talus.

Cartilage Replacement Strategies

Osteochondral Autograft Transplantation (OAT)

Hangody first introduced his clinical technique of mosaicplasty in 1992, to transplant healthy osteochondral plugs from relatively nonweight-bearing areas of the knee to treat osteochondral lesions of the knee.⁹² Over the years, this concept has been carried forth to treat OLT. Osteochondral autograft transplantation (OAT) is a single-stage replacement rather than a reparative treatment option that transfers intact hyaline cartilage with subchondral bone to relatively large defects of the ankle. These osteochondral plugs can be removed from the sulcus terminalis of the knee intercondylar notch, anterior talar head, or medial and lateral nonarticulating portions of the talus. This technique has the advantage of graft availability, absence of disease transmission, lower cost, and ability to use multiple plugs (Fig. 16.42A-D). Disadvantages of the technique include graft availability, nonunion of the osteotomy, donor site morbidity, poor integration of the graft surface with native tissue, cyst formation

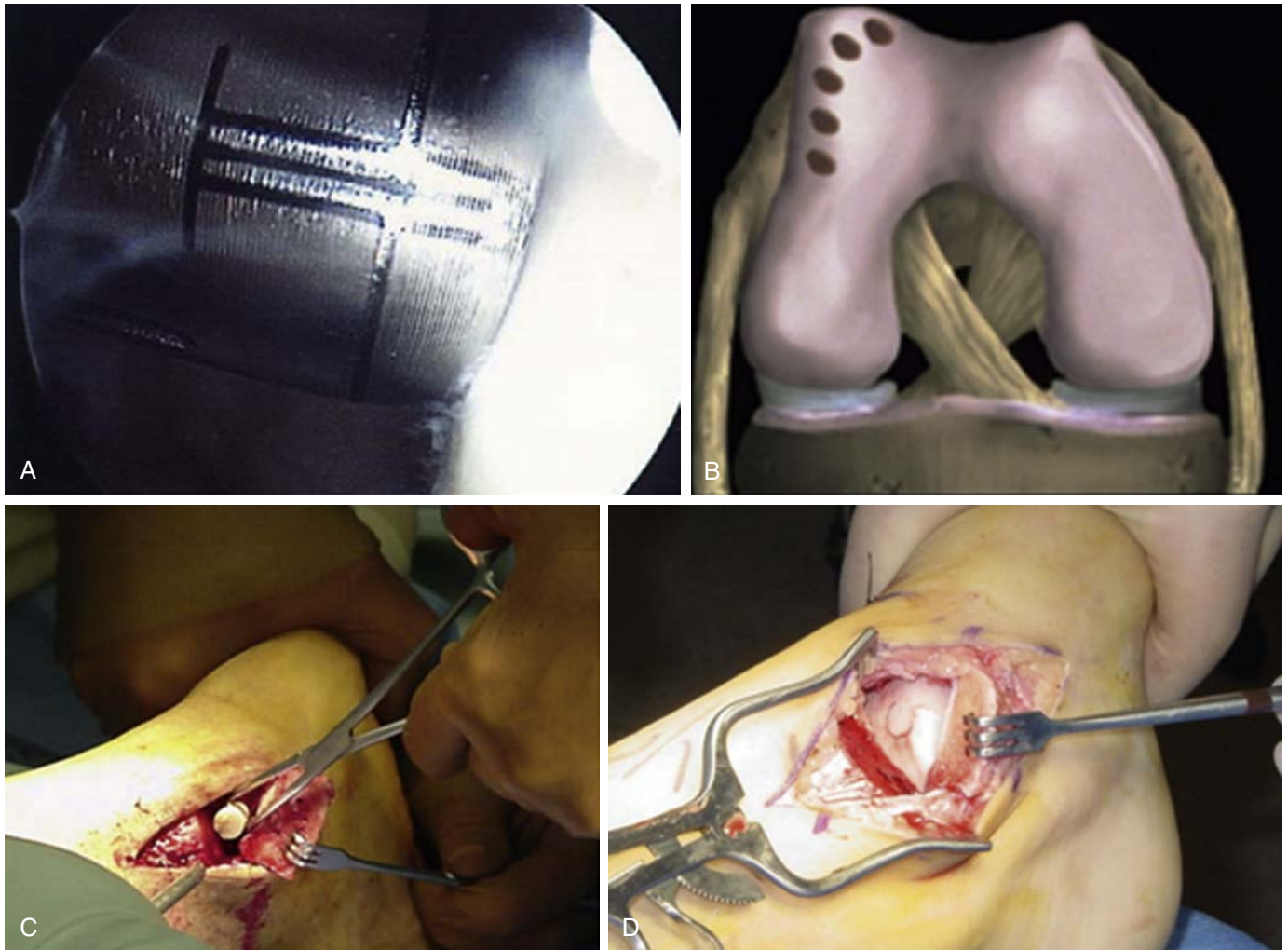


Fig. 16.42 Osteochondral autograft transplantation. (A) The osteochondral autograft is obtained from the lateral femoral condyle in this arthroscopic picture. (B) Note the use of multiple autografts obtained from the lateral knee. (C) After the osteochondral lesion is removed and the holes drilled appropriately, the osteochondral autograft is inserted until it is flush with the remaining articular cartilage. (D) The final result after osteochondral autograft insertion. The medial malleolar osteotomy is then reattached with internal fixation.

around the graft site, and nonunion or deterioration of the graft cartilage.^{93–97} More anterior lesions can be accessed through plantarflexion and ankle distraction, but more central or posterior lesions, medially and laterally, require an osteotomy with subsequent screw and/or plate fixation. If the graft protrudes or sinks below the margin of the normal articular cartilage, contact pressures on the graft surface increase significantly. In addition, biomechanical and structural differences are present between talar and knee articular cartilage that may produce problems long-term.

Sammarco and Makwana treated 12 patients with grafting from the medial or lateral talar facet and reported improvement in AOFAS score from 65 to 91 at 25 months, follow-up.⁹⁸ Scranton et al. performed 50 OAT procedures for OLT, utilizing plugs from the intercondylar notch.⁹⁹ Sixty-four percent of these patients failed one or more surgeries prior to the procedure. These patients achieved 90% good-excellent results, although 15 patients required a second operation. Gobbi et al.

compared chondroplasty and microfracture and OATs in 33 ankles.¹⁰⁰ Although postoperative scores showed significant improvement at 12 months, the ankle-hindfoot scale showed no difference in the rest of the group at 24 months, and the subjective assessment numeric evaluation ratings at 53 months also did not show significant differences in groups. Valderrabano et al. studied 12 patients with OATs and reported significant pain relief and improvement at 72 months, but half the patients subsequently noted knee pain and most patients had developed recurrent ankle lesions with associated joint degeneration.⁹⁶

Flynn et al. reported on autologous osteochondral transplantation in 85 consecutive patients.¹⁰¹ The mean FAOS improved from 50 to 81, and the MOCART score was 86. Lesion size was negatively correlated with the MOCART score, and a quantitative T2 mapping suggested that graft tissue may not always mirror native hyaline cartilage. Hannon et al. did a prospective randomized study comparing osteochondral autograft transfer, chondroplasty, and microfracture.¹⁰² Although the

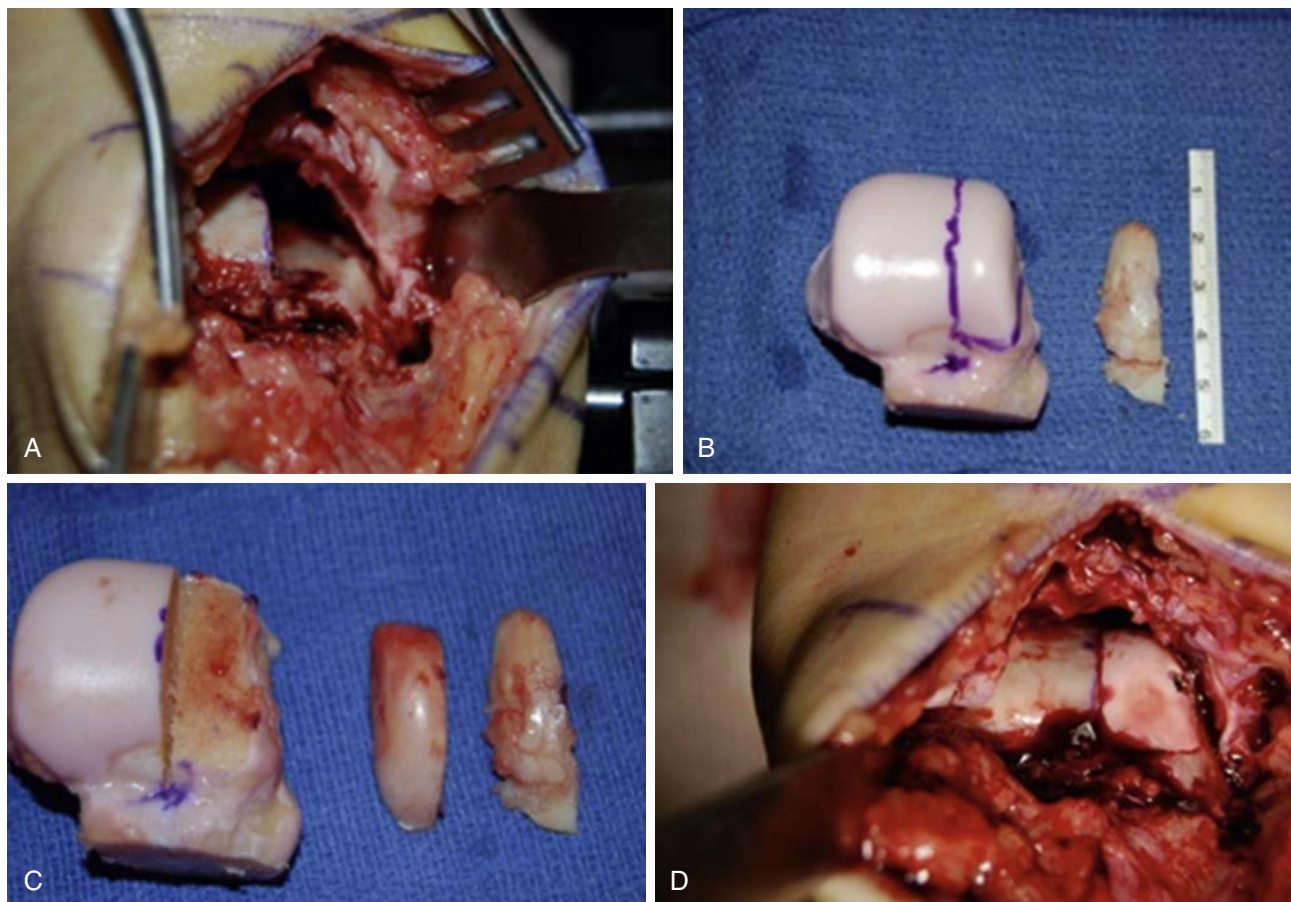


Fig. 16.43 Insertion of osteochondral allograft. (A) Through an anterior approach and distraction, the medial portion of the talus is removed. (B) This shows the size of the lesion that is removed and measurements done to replicate the exact anatomy and size on the allograft. (C) The graft of the talus appears to match nicely the excised portion of the autograft talus removed. (D) Appearance of the allograft after insertion and fixation into the right ankle.

numbers were small, the pain and functional scores improved in all groups, but there was no significant differences in the Ankle-Hindfoot Scale at 12 and 24 months, or the Subjective Assessment Numeric Evaluation.

A number of studies suggest that between 63% and 95% of athletes return to their previous level of activity following OAT. Paul et al. examined sports activity postoperatively in a group of 31 patients after OATs.¹⁰³ Although the patients' time doing sporting activity did not change after surgery, their Tegner scores decreased and the patients did less high-impact and contact sports. Paul et al. also reported on 112 patients with minimum 2-year follow-up.¹⁰⁴ Only 55% were happy with their knee postoperatively. The WOMAC score was 5.5% and the Lysholm Score was 89. The number of grafts, size of the transplant cylinders, and patient age did not influence either the WOMAC or the Lysholm Score.

Osteochondral Allograft Transplantation

Osteochondral allograft transplantation is similar to OATs but avoids some of the disadvantages of the latter procedures. Indications are for failed previous surgery, large lesions, particularly with cysts, and lesions with significant bone loss. Allograft transplantation can use plugs of bone and cartilage or do en bloc

grafts for very large lesions. The allograft can be taken from the exact location on the donor talus as the lesion on the patient's own anatomy. A CT scan of the patient's uninjured talus can be used as a template (Fig. 16.43A-D).

Because of safety concerns over potential infection, allografts are hypothermically stored for a minimum of 14 days to allow for extensive serologic and biologic testing. Since chondrocyte viability decreases after 28 days post-mortem, fresh osteochondral allografts should be utilized between 15 and 28 days post-mortem. Osteochondral allografts, however, also have some concerns, including difficult graft availability, chondrocyte viability, disease transmission, and cost.

Hahn et al. showed improved activity and pain levels in 13 patients who underwent allograft transplantation with internal fixation.¹⁰⁵ Raikin inserted allografts in 15 patients with an average follow-up of 54 months, and 11 of 15 patients graded the result as excellent or good.¹⁰⁶ El-Rashidy et al. used fresh allografts in 38 patients, with a mean age of 44 and a mean lesion size of 1.5 cm².¹⁰⁷ At a follow-up of 38 months, the graft survival was 89% and the AOFAS score went from 52 to 79. More recently, Gortz, DeYoung, and Bugbee did a follow-up report on 11 allograft patients with 12 ankles.¹⁰⁸ All involved partial unipolar grafts inserted through an anterior approach

without osteotomy. Mean age was 36 and the follow-up was 38 months. The mean OMAS improved from 28 to 71. Graft survival rate was 83%, but only 5 of 12 patients achieved good-excellent results. Shimozone et al. compared 25 nonrandomized autograft patients to 16 allograft patients who had an osteochondral transplantation for an OLT.¹⁰⁹ The autograft group provided better clinical and MRI outcomes than the allograft patients. The rate of chondral wear on MRI was higher with allograft than with autograft, and the allograft-treated patients had a higher rate of clinical failure.

Autologous Matrix-Induced Chondrogenesis

Autologous matrix-induced chondrogenesis (AMIC) is a one-step scaffold-based therapy and is done by performing microfracture and/or drilling of the lesion, then inserting a type I/III collagen membrane to cover the defect. Theoretically, this offers a barrier for the clot to be held, and provide an environment for cell differentiation and new cartilage formation. Valderabano showed 26 patients with good clinical results on AOFAS and VAS scores.¹¹⁰ However, MRIs showed complete filling in only 35% of the defects, and 50% of the patients had a hypertrophic cartilage layer. Thermann in 2012 did AMIC with microfracture and platelet-rich plasma (PRP) in 64 patients for an OLT.¹¹¹ The microfracture holes were filled with PRP and the collagen matrix was soaked in PRP and then glued over the defect. Postoperatively, their Hospital for Special Surgery (HSS) results improved from 55 to 81 and their VAS scores also significantly improved.

Autologous Chondrocyte Implantation (ACI)

In 1965, Smith et al. were the first to isolate and grow chondrocytes in a culture medium. Subsequently, Grande, Peterson, and others were the first to introduce a biologic cell-based technique to treat full-thickness cartilage defects with cultured expanded chondrocytes.¹¹² They termed this process “autologous chondrocyte implantation,” and it was first introduced in humans in 1987. The indications for this procedure included patients who failed previous surgery for osteochondral lesions and for larger defects, including cystic lesions. Peterson developed the “sandwich technique” to treat large cystic lesions with bone grafting and insertion of chondrocytes between two biologic membranes. Nam and Ferkel reviewed the results of their first 11 patients who failed previous surgery, and excellent results were seen in 82% of the patients.¹¹³ Kwak, Ferkel et al. published a second report on 29 patients undergoing autologous chondrocyte implantation (ACI) for OLT using a periosteal graft cover.¹¹⁴ Patients demonstrated significant improvement in Tegner scores at a follow-up of 70 months. Giannini et al. studied 46 patients undergoing ACI for large osteochondral lesions.¹¹⁵ Patients with lateral lesions had better AOFAS scores compared with medial lesions. They operated on 29 athletes, and 20 resumed sports at the same level, while 4 could not go back to their sport.

Membrane/Matrix Autologous Chondrocyte Implantation

In 1998, Behrens et al. implanted a type I, III collagen membrane impregnated with harvested cultured chondrocytes (MACI).¹¹⁶ Since that time, Anders et al. performed MACI on 22 patients

for OLT.¹¹⁷ AOFAS scores improved from 70 to 95 at 64 month follow-up. Magnan et al. treated 30 patients with MACI, with a mean OLT size of 2.36 cm².¹¹⁸ The mean AOFAS score improved from 37 to 84 at a follow-up of 45 months. However, only 50% of the patients returned to previous sports activity. Kreulen et al. prospectively studied MACI on 10 patients with an OLT who had failed previous surgery.¹¹⁹ Nine patients were available at 7-year follow-up and compared with their preoperative scores. The SF-36 at 7 years showed significant improvements in physical functioning, lack of bodily pain, and social functioning. The mean AOFAS Hindfoot Score improved from 61 to 78. Giannini et al. used a hyaluronic acid-based membrane for MACI on 46 patients, with mean lesion size of 1.6 cm².¹²⁰ Mean AOFAS scores improved from 57 to 87 at 12 months, and 89 at 36 months. Forty patients were able to resume their normal level of sports; only four were forced to give up athletic activity.

More recently, MACI has been cleared by the FDA to be used in the United States since 2017. The technique we currently use involves usually a medial or lateral malleolar osteotomy with excision of the OLT. If a cyst is present, it is excised and bone grafted, then covered with fibrin, and the membrane impregnated with the patient's cells is placed over the defect. If no cyst is involved, the membrane is placed over the defect with fibrin glue and, in some cases, suture augmentation (Fig. 16.44). Currently, studies are underway to assess its efficacy and results using open surgery in several U.S. centers.

Particulated Juvenile Cartilage

Particulated juvenile cartilage (DeNovo™, Zimmer Biomet) is a relatively new technique that involves the use of allograft cartilage obtained from donors under the age of 13 years. Immature articular cartilage has a much higher cellular density.¹²¹ In addition, there is an increased proliferation rate and improved ability to produce extracellular matrix and retain cartilage phenotype.

The use of particulated juvenile cartilage is a single-stage procedure and involves arthroscopic excision of the osteochondral lesion, drilling or microfracture (this is under debate), and placement of fibrin glue on top of the osteochondral lesion, then insertion of the particulated cartilage, followed by another layer of fibrin glue. This procedure can be done either completely arthroscopically or through an open approach (Figs 16.45A-C). If there is an underlying cyst below the osteochondral lesion, this is curetted, removed, and filled with bone graft, then covered with a layer of fibrin glue prior to inserting the particulated juvenile cartilage. Coetzee et al. reported the first large series results in 2013.¹²² More recently, they have updated their results with 2- to 7-year follow-up on 77 patients and showed 90% had over 70% improvement compared with preoperative scores, and 92% were satisfied without any or minor reservations.¹²³ There were five revisions in this group of patients. Dekker et al. followed 15 patients for a mean follow-up of 35 months and had a 40% failure rate, with the risk factors of failure including preoperative MRI area, intraoperative OLT size, and male patients.¹²⁴ They found that lesions greater than 125 mm² had significantly increased risk of clinical failure. Karnovsky et al. studied 50 patients: 30 with microfracture and 20 with particulated juvenile cartilage.¹²⁵ Both patient groups showed significant clinical

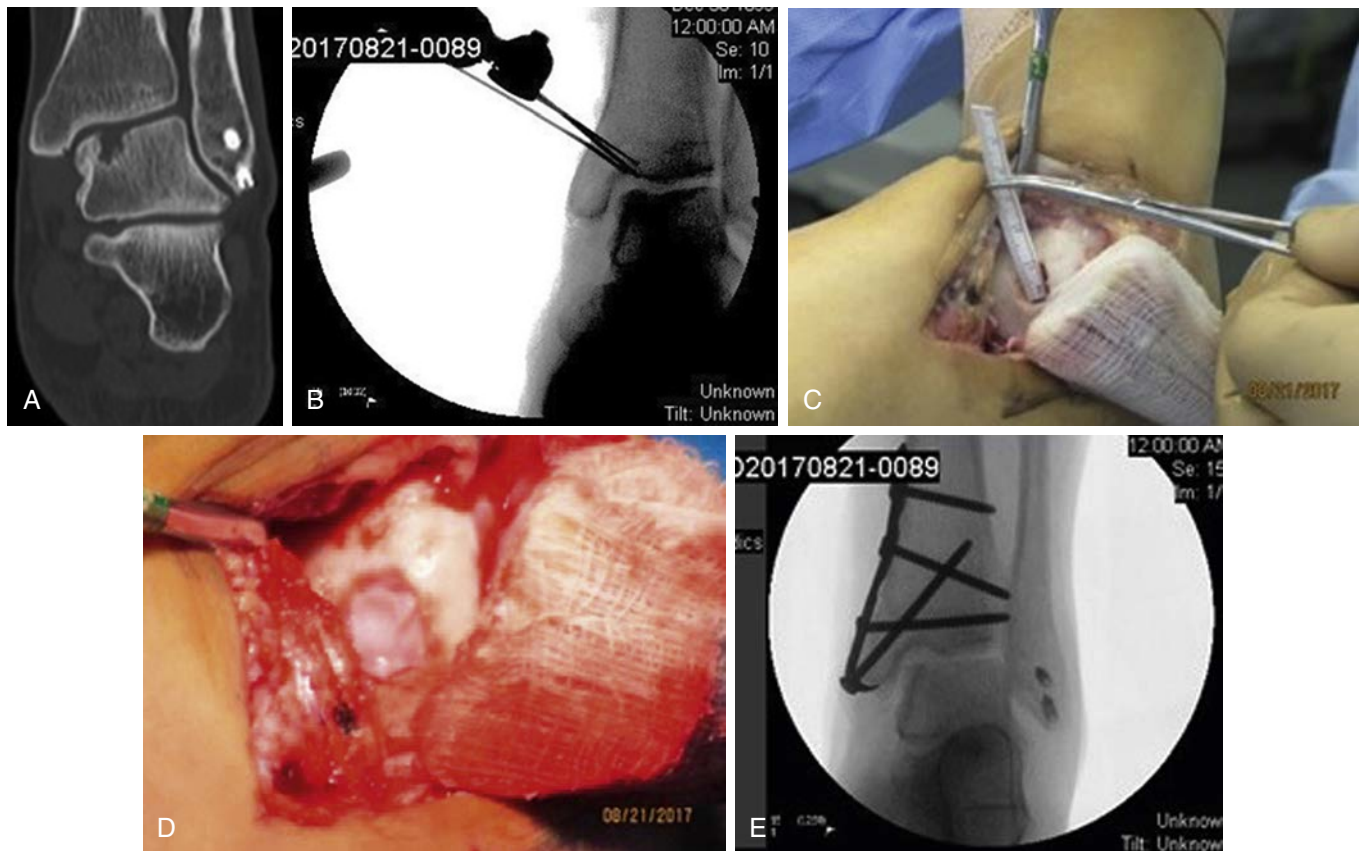


Fig. 16.44 MACI case study. (A) Cystic osteochondral lesion of the talus that has failed previous surgery in a left ankle. (B) Guidepins are used to cut along the medial malleolus to ensure that an accurate osteotomy is performed lateral to the osteochondral lesion. (C) After excision of the osteochondral lesion, careful measurement is made of the exact size of the lesion. (D) Placement of the MACI scaffold onto the medial osteochondral lesion and securing with fibrin glue. (E) Reattachment of the medial malleolar osteotomy in a left ankle with a hook plate device.

improvements, but no differences were seen in patient-reported outcomes between the groups. However, the average OLT size was seen to be larger for the particulated juvenile cartilage patients than the microfracture patients. At SCOI, we recently looked at our results with particulated juvenile cartilage in the first 18 patients with a prospective study and a mean follow-up of 25 months. Scores from the FAOS, FAAM, and SF-36 markedly improved in every subscale category. We continue to monitor this group with longer follow-up. Further Level 1 studies are necessary to determine the long-term effects of its use.

Micronized Cartilage

More recently, micronized cartilage matrix (BioCartilage™, Arthrex) has been introduced as another cartilage transplant alternative. Micronized cartilage matrix is an allograft cartilage extracellular matrix that contains the key components of cartilage, including type II collagen, proteoglycans, and additional cartilaginous growth factors. It is dehydrated, then micronized into small particles and aseptically packaged. Its intended use is to provide a scaffold over the microfracture defect and hopefully improve the quality of cartilage tissue that has formed over the osteochondral lesion defect. The technique is done arthroscopically or through a small mini-arthrotomy. Currently, we do all these arthroscopically,

utilizing the anteromedial, anterolateral, and posterolateral portals. The OLT is excised and drilled and/or microfractured, then the micronized cartilage is inserted (Fig. 16.46). Fortier et al. treated 10-mm cartilage defects in horses with microfracture alone or microfracture plus micronized cartilage and PRP.¹²⁶ The micronized cartilage group safely improved cartilage repair compared with microfracture alone up to 13 months after implantation. The micronized cartilage is mixed with bone marrow aspirate into a syringe, which is then injected through an open or arthroscopic technique into the ankle. Its cost is significantly less than particulated juvenile cartilage or MACI, and it has a shelf life of 5 years. Fansa et al. studied 31 patients with micronized cartilage matrix and bone marrow aspirate concentrate (BMAC), with a mean age of 38 and mean follow-up of 16 months.¹²⁷ The average lesion size was 86 mm². The PROMIS scores improved, and the mean postoperative MRI and MOCART were 69. Dekker and Patel studied 12 patients who had micronized cartilage and BMAC for OLT.¹²⁸ The PROMIS pain and function were assessed at different intervals. No significant differences were seen in the PROMIS pain and function scores at 6 and 12 months or preop scores. The MOCART score was 53. Much more research needs to be done on micronized cartilage to determine its effectiveness in the ankle.

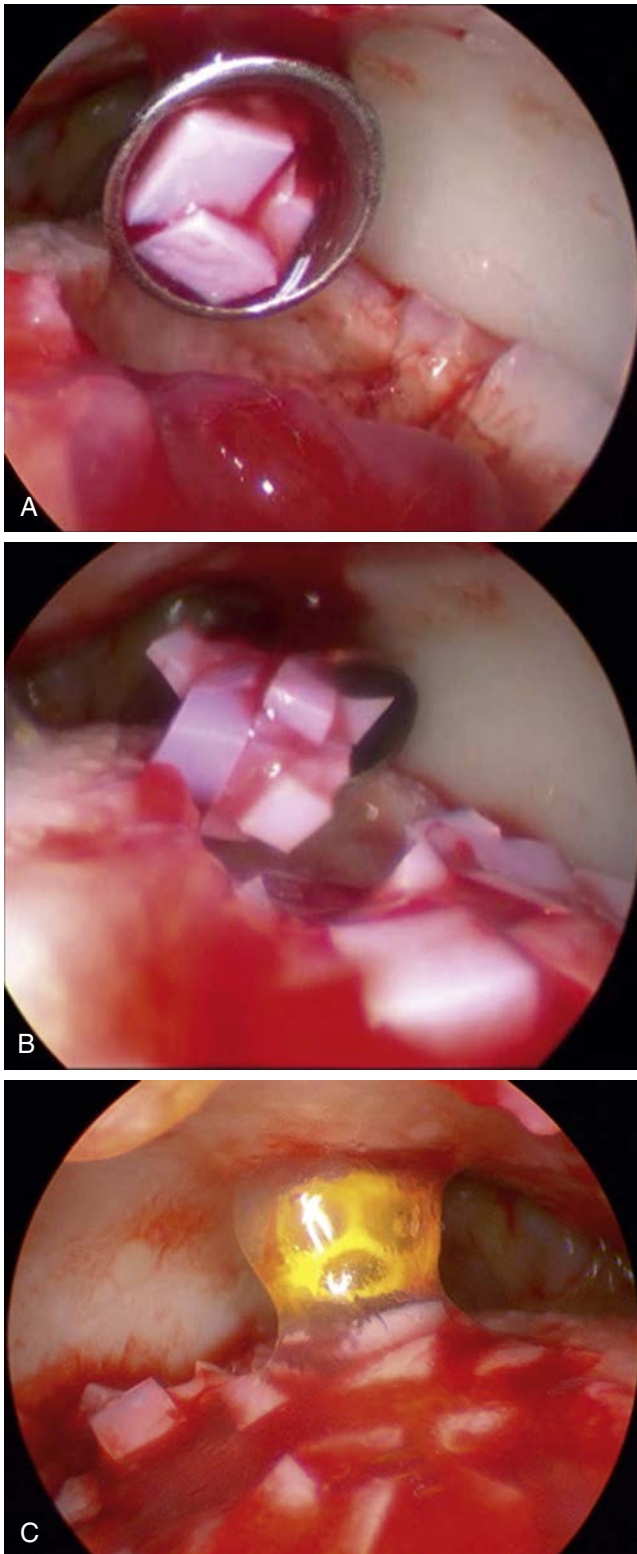


Fig. 16.45 Particulated juvenile cartilage. (A) After the osteochondral lesion is excised in this right ankle, fibrin glue is applied over the osteochondral lesion bed and then the juvenile particulated cells are inserted through the anteromedial portal. Visualization through the posterolateral portal. (B) The entire lesion is carefully filled with the particulated juvenile cartilage and impacted with a freer elevator. Visualization through the posterolateral portal. (C) A small amount of fibrin glue is placed over the juvenile particulated cartilage to ensure stability. Visualization through the posterolateral portal.

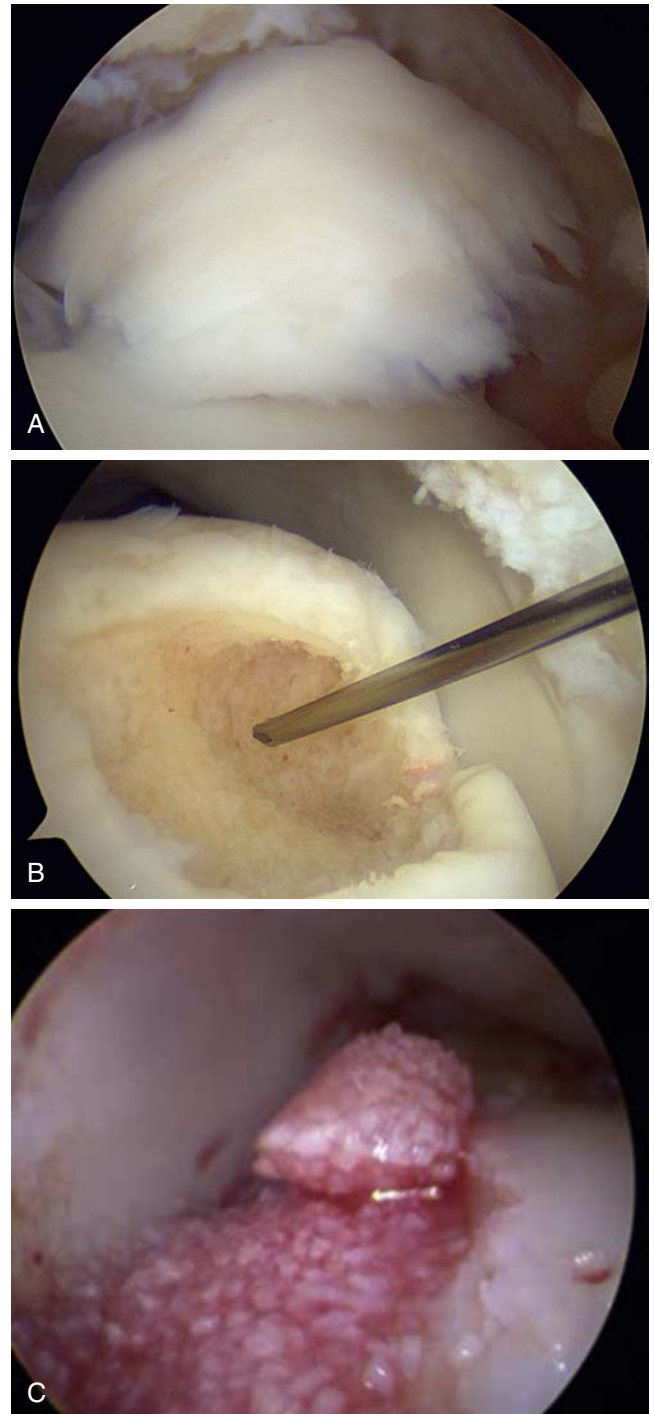


Fig. 16.46 Micronized cartilage matrix. (A) The loose flap of articular cartilage in a right ankle medial talar dome. (B) Percutaneous drilling with a thin wire of the OLT. (C) Insertion of the micronized cartilage matrix through the anteromedial portal in the right ankle, while visualizing from the posterolateral portal.

FUTURE CELL STRATEGIES

The MIAMI Cell

The MIAMI (marrow isolated adult multilineage inducible) cell is a primitive mesenchymal stem cell that expresses many of the genes expressed by embryonal cells but follows a script. Under appropriate experimental conditions, these cells can be induced

to become bone forming or cartilage forming. Tom Temple and his colleagues have done a great deal of work on this, but no results are available at this time.

Metallic Cap Implant

A metallic cap implant has been developed for use in cases of failed OLT surgery. It has a cobalt chrome component and many of the original biomechanical studies were done at the University of Iowa. It is intended to be used for osteochondral lesions of the medial talar dome, and it is hoped that it will redistribute the contact stresses toward normal. Van Bergen et al. reported using a metallic resurfacing implant in 20 patients with an average follow-up of 3 years.⁷⁶ They found the FAOS scores improved and there was decreased pain on rest, walking, stair climbing, and running. More recently, Vuurberg et al. reported on the metallic cap implant on 38 patients with a mean follow-up of 5.1 years.¹²⁹ AOFAS scores increased from 57 to 80 and most of the FAOS scores improved. Return to sport occurred at a mean of 4.1 months, and 77% resumed some sports activity. There were 2 failures and 21 reoperations.

In the future, cartilage transplant material must be implanted easily and quickly, preferably through the arthroscope. It must have reduced surgical morbidity and not require harvesting other tissue. In addition, it must exhibit an enhanced cellular proliferation and maturation, easier phenotype maintenance, and allow for efficient and complete integration with surrounding articular cartilage.

Hopefully in the future, techniques will be developed that allow athletes to return back to their sporting activity with a high percentage of excellent results that hold up after a long time period.

OSTEOCHONDRAL LESIONS OF THE TIBIAL PLAFOND

OLTP or osteochondral lesions of the distal tibia are much less common than OLT. In the past, we looked up our own numbers at the Southern California Orthopedic Institute and found approximately 30% of all ankle arthroscopies involved OLT, while only 3% of our ankle arthroscopies involved OLTP. The etiology can be trauma, spontaneous osteonecrosis, vascular supply problems, and others. Elias et al. used 38 MRIs to determine the most common location and size of OLTPs.¹³⁰ The medial central plafond was most common and the posteromedial plafond was the second most frequent. In our own experience, most of these occur in the central posterior aspect of the distal tibia. Some occur in the medial malleolus.

Technique

The technique for treating an OLTP is the same as an OLT. We use small joint 2.7-mm 30- and 70-degree arthroscopes and use small instrumentation with multiple portals. Occasionally, lesions of the distal tibia will occur with a lesion of the talus, and are called “bipolar” lesions.

The technique for treatment includes unroofing and removing the loose articular cartilage and bone with curettes and a shaver. We then use a thin wire to drill the lesions, either retrograde or antegrade, and may also put a few small microfracture holes in the lesion as well. The MicroVector™ is very helpful to drill these particular lesions retrograde (Fig. 16.47). If there are cysts, these cysts

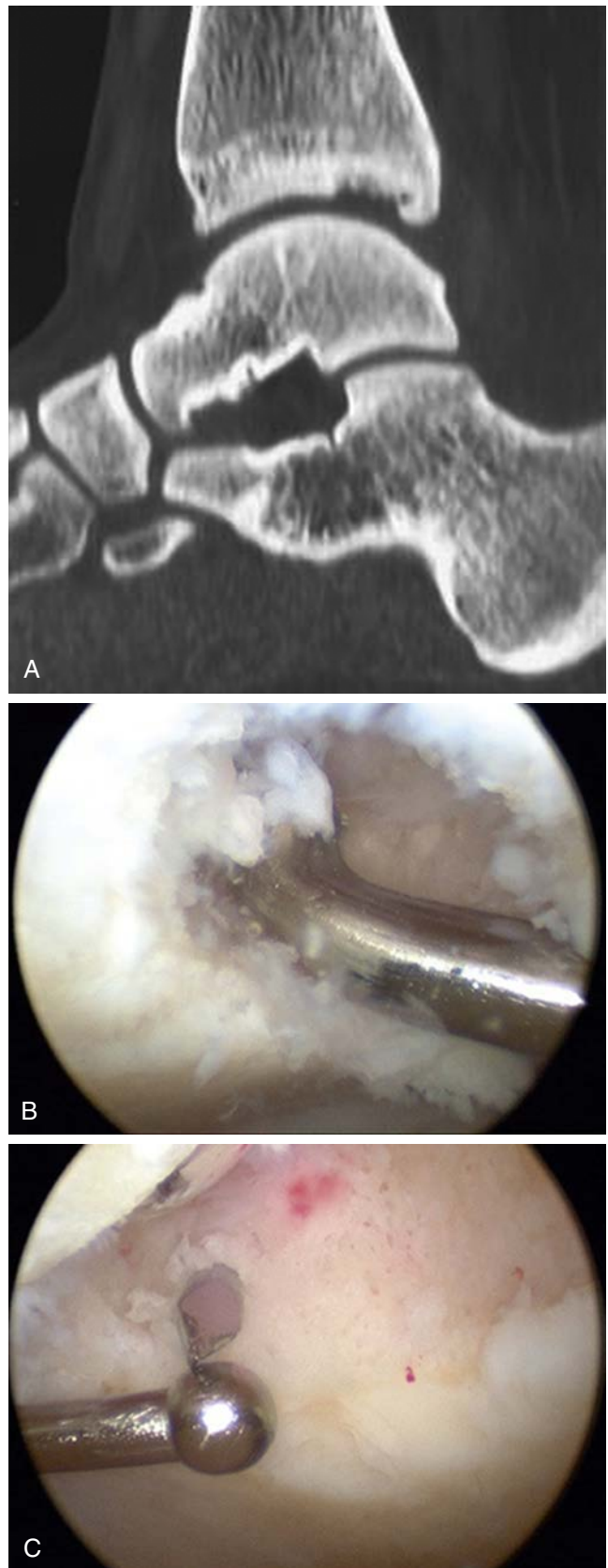


Fig. 16.47 Osteochondral lesion of the tibial plafond. (A) Sagittal CT scan showing the posterior location of the osteochondral lesion of the tibial plafond. (B) Excision of the osteochondral lesion of the tibial plafond with curettes to good bleeding bone and healthy cartilage. (C) Insertion of the MicroVector™ to drill holes into the osteochondral lesion of the tibial plafond.

need to be removed and the area bone grafted as well. Mologne and Ferkel published the first paper on treatment of these lesions in 17 patients and found the AOFAS scores improved from 52 to 87.⁵⁴ Fourteen of 17 patients (82%) had good and excellent results. Cuttica et al. reported on 13 patients who were treated for an osteochondral lesion of the tibial plafond.¹³¹ Nine patients had an isolated lesion and four had a bipolar lesion. Eleven of the 13 patients were available for follow-up, and their AOFAS score went from 35 to 50. Four patients had a poor outcome.

If there is an underlying cyst below the osteochondral lesion, this is curetted, removed and filled with bone graft, then covered with a layer of fibrin glue prior to inserting the particulated juvenile cartilage.

BIOLOGIC ADJUVANTS TO CARTILAGE REPAIR (SEE ALSO CHAPTER 30)

Platelet-Rich Plasma (PRP)

PRP is an autologous blood product that contains at least twice the concentration of platelets compared with baseline values. These platelets contain cytokines and growth factors that participate in tissue healing and can also potentially attract mesenchymal stem cells to the osteochondral lesion. Smyth et al. showed in a literature review that there was a positive effect of PRP on cartilage repair.¹³² A number of the authors showed that PRP increased chondrocyte and mesenchymal stem cell proliferation, type II collagen deposition, proteoglycan deposition, and inhibited the effects of catabolic cytokines.

Hyaluronic Acid

Hyaluronic acid is a carbohydrate component contained within synovial fluid that enhances the viscoelastic properties and experimentally increases the proliferation of cultured chondrocytes in vitro.^{133,134} More recently, Doral et al. showed that

three intraarticular hyaluronic injections performed weekly beginning in the third postoperative week after an arthroscopic debridement and microfracture significantly improved clinical scores, compared with microfracture only control.¹³³ Mei-Dan et al. compared the use of PRP or hyaluronic acid to treat an OLT.¹³⁵ Both groups improved postoperatively, but the PRP group significantly had a better outcome than the hyaluronic acid group.

Bone Marrow Aspirate Concentrate (BMAC)

BMAC is a great source of mesenchymal stem cells, growth factors, and cytokines that may improve the quality of cartilage repair tissue. BMAC is obtained intraoperatively from the iliac crest, proximal or distal aspects of the tibia, or calcaneus. Fortier et al. graded an osteochondral lesion in 12 horses and compared the use of BMAC with microfracture versus microfracture alone.¹³⁶ The group with BMAC plus microfracture resulted in superior healing of cartilage defects than the microfracture alone. Goodrich et al. studied the effect of BMAC on PRP-enhanced fibrin scaffolds and chondral defects in horses.¹³⁷ He found PRP resulted in thicker repair tissue that was not improved by adding BMAC. We usually obtain the BMAC from the iliac crest through a separate setup, then reposition, re-prep, and drape the patient for the arthroscopic ankle procedure. After the osteochondral lesion is excised and marrow stimulation is performed, the joint is suctioned dry and the thickened BMAC is then inserted over the osteochondral lesion (Fig. 16.48).

Bone Marrow–Derived Cell Transplantation

Bone marrow–derived cell transplantation (BMDCT) uses a combination of BMAC and a scaffolding material to fill the osteochondral defect after excision and marrow stimulation. Postoperative scores have been shown with this technique to significantly improve the clinical results.¹³⁸

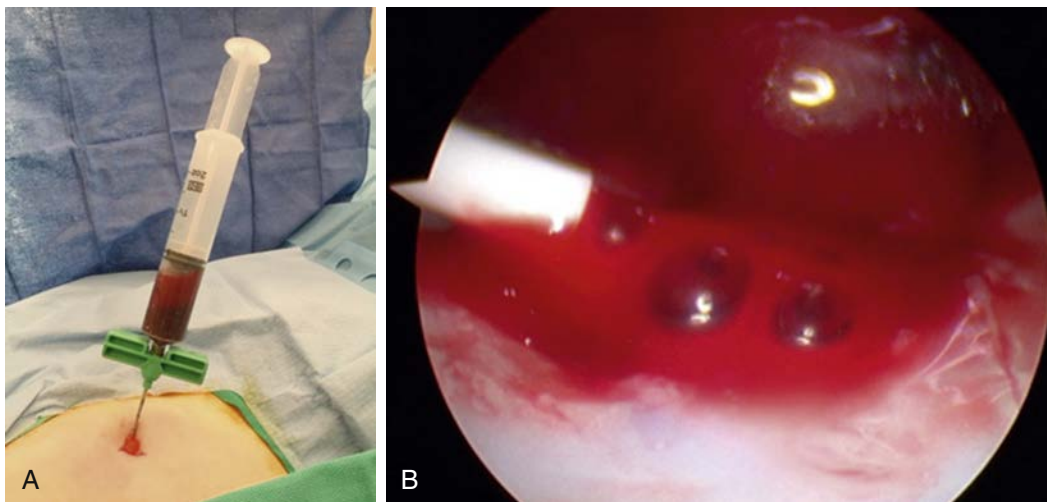


Fig. 16.48 Bone marrow aspirate concentrate. (A) A small incision is made in the iliac crest and a Jamshidi needle is inserted between the inner and outer tables of the iliac crest to obtain 60 ccs bone marrow aspirate. (B) Bone marrow aspirate is then spun down to approximately 5 ccs and injected onto the osteochondral lesion after a bone marrow stimulation procedure has been performed. Visualization from the anterolateral portal in an osteochondral lesion of the left ankle medial talus.

INTERNATIONAL CONSENSUS MEETING ON CARTILAGE REPAIR OF THE ANKLE

In 2011, Drs. John Kennedy, Niek van Dijk, and Richard Ferkel formed the International Society on Cartilage Repair of the Ankle (ISCRA) to study osteochondral lesions. The first meeting was called the International Congress on Cartilage Repair of the Ankle (ICCRA) and was held in Dublin, Ireland, in March 2012. Subsequently, there were five other meetings

on a yearly basis. In November 2017, the first International Consensus Meeting on Cartilage Repair of the Ankle occurred in Pittsburgh, Pennsylvania. Ninety-five participants from 26 countries around the world convened, using the Delphi Process to answer questions on all aspects of cartilage repair of the ankle. These proceedings have been published in a supplement to the *Foot and Ankle International* journal and are available for all to read and review.¹³⁹ Very specific guidelines are presented in this monograph to help guide treatment of OLT.

SUMMARY

Osteochondral lesions of the talus and tibial plafond pose significant challenges for treatment. There are many factors that influence results, including the size of the lesion, location of the lesion, containment of the OLT, subchondral cysts, status of the cartilage cap, as well as other associated pathology. Great research is occurring throughout the world to try to find better techniques and materials to treat these lesions. The future

is very exciting, but much work needs to be done to develop a technique that will produce true type II articular cartilage, hold up for a lifetime, that will be safe, cheap, and easy to perform, preferably arthroscopically. At this time, we have options for our athletes with osteochondral lesions of the tibia and/or talus, but significant questions still exist as to the best technique to return athletes long term to their desired sport.

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Disorders of the Subtalar Joint, Including Subtalar Sprains and Tarsal Coalitions

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INTRODUCTION

Pathology in the subtalar joint can be debilitating, is challenging to diagnose, and can lead to significant lost time for the athlete. In addition to acute bony or cartilage injuries, instability, ligamentous strain, and congenital coalition may all play a role in generating subtalar pain. Subtalar instability as an isolated phenomenon or in conjunction with lateral ankle ligament instability is often subtle and is frequently overlooked. Sinus tarsi syndrome remains a nebulous diagnosis that can typically be correlated with distinct intraarticular pathology. Additionally, pain along the lateral aspect of the subtalar joint may result from a severe planovalgus position of the foot leading to impingement of the calcaneus against the fibula. Subfibular impingement must be distinguished from true pathology of the subtalar joint. Tarsal coalition, although found in up to 2% of the population as a whole, is commonly symptomatic in the running or jumping athlete/dancer.

ANATOMY

The subtalar joint is comprised of three articulating surfaces: the posterior facet, the middle facet, and the anterior facet

(Fig. 17.1). Confluence of the anterior and middle facets is present in over 40% of cases.¹ The bony articulations provide inherent stability and soft tissues provide additional stabilization. The lateral soft-tissue stabilizers have been classified into three separate layers.¹ The superficial layer is composed of the lateral root of the inferior extensor retinaculum, the lateral talocalcaneal ligament, and the calcaneofibular ligament (CFL). The intermediate layer consists of the intermediate root of the inferior extensor retinaculum and the cervical ligament. The deep layer consists of the medial root of the inferior extensor retinaculum and the interosseous talocalcaneal ligament (Fig. 17.2).

The joints move in a triaxial plane, which allows for the motions of flexion/extension, inversion/eversion, and adduction/abduction.

The sinus tarsi is a broadly conical space on the lateral aspect of the foot that lies anterior to the posterior facet between the talus and the calcaneus. It is in continuity with the tarsal canal. The tarsal canal is a narrow opening within the medial subtalar joint and is situated in a posteromedial-to-anterolateral direction. Soft tissues within the sinus tarsi include the artery of the tarsal canal, bursae, nerve endings, and multiple ligaments.



Fig. 17.1 Variations in the anterior and middle facets of the calcaneus. Conjoined, transitional, and distinct facets are all seen in the population. (From Ragab AA, Stewart SL, Cooperman DR. Implications of subtalar joint anatomic variation in calcaneal lengthening osteotomy. *J Pediatr Orthop* 2003; 23:79-83.)

SUBTALAR INSTABILITY

The role that instability of the subtalar joint plays in the patient with lateral ankle instability has been elucidated only recently. Some authors have estimated that 10% to 30% of patients with functional ankle instability marked by a sense of “giving way” of the ankle have evidence of instability of the subtalar joint.^{2,3} It has been suggested that consideration should be given to the concept of global hindfoot instability rather than simply functional instability about the ankle joint.⁴

Instability of the subtalar joint was first proposed in 1962 by Rubin and Whitten.⁵ They proposed a series of stress radiographs to further evaluate this disorder. Brantigan et al.⁶ were the first to detect radiographic evidence of subtalar instability in their series of three patients. Chrisman and Snook⁷ in 1969 were able to document clinical subtalar instability in three of seven patients who were undergoing their tendon transfer procedure for lateral instability. Clanton and Berson⁸ described subtalar injuries as a continuum of other injuries in athletes, particularly sprains of the lateral ankle ligaments.

While most cases of subtalar instability are associated with a more global pattern of injury to the lateral ankle ligament complex including the anterior talofibular and calcaneofibular ligaments, cases of isolated subtalar instability do occasionally present without associated tibiotalar pathology. It is difficult if not impossible to distinguish excess motion on physical examination from the subtalar joint alone from that emanating from the tibiotalar joint or a combination of the two. While stress radiographs have a very limited role in the diagnosis of ankle instability, they may be of utility in diagnosing isolated subtalar instability patterns. Lateral gapping of the subtalar joint on a Broden's view is typically seen in these cases, although the results must be interpreted with caution. The diagnosis is based upon a combination of radiographic and clinical factors; lateral gapping on stress view may be seen in asymptomatic patients as well.⁹

Clinical Presentation

The typical injury that leads to instability of the subtalar joint is a severe supination or supination-inversion force applied to

the hindfoot. This results in a progressive injury to the talonavicular ligament and talonavicular capsule, followed by injury to the calcaneofibular and lateral talocalcaneal ligaments.⁸ The presenting complaint often is a sensation of giving way of the ankle. The patient may report pain localized to the region of the sinus tarsi. Athletic activities can exacerbate the symptoms, resulting in a dependence on bracing or taping. Uneven surfaces may cause pain and a feeling of instability.

It is difficult to differentiate lateral ankle instability from subtalar instability on the basis of patient history. A thorough clinical and radiographic workup can help define the source of the athlete's complaints, but the differentiation still can be elusive.

Physical Examination

The most notable finding on physical examination is increased inversion of the subtalar joint. This should be compared with the presumably uninjured opposite limb. The increased inversion can result from subtalar instability or a combination of subtalar and ankle instability.^{4,8} It is not possible to detect the location of increased inversion by examination. In addition to increased inversion of the hindfoot, an increased translation of the calcaneus in the medial direction has been noted by Thermann et al.¹⁰ In their test, a valgus stress was applied to the calcaneus, followed by an abrupt internal rotation stress. Stress radiographs showed a medial shift of the calcaneus in relation to the talus or an opening of the talocalcaneal angle in patients with subtalar instability.

Following an acute injury, there may be swelling, bruising, and tenderness laterally. In the more chronic setting, increased inversion and lateral tenderness are more likely. It is easier to detect instability clinically in the chronic setting because the athlete will be less apt to guard because of pain.

Radiographic Evaluation

The initial radiographic workup of the patient with subtalar instability involves a weight-bearing anterior-posterior (AP), lateral, and mortise view of the affected ankle, as well as weight-bearing AP, lateral, and oblique radiographs of the affected foot to rule out evidence of bony pathology.

Plain radiographs often are negative, and further investigation must be carried out to arrive at the diagnosis. There have been multiple investigations into the use of stress radiographs in the workup of subtalar instability (Fig. 17.3).^{6,11-13} In a series of three patients, Brantigan et al.⁶ were able to radiographically demonstrate subtalar instability. They attributed the instability to an injured CFL. Heilman et al.¹³ sequentially sectioned ligaments in cadaver limbs and then obtained lateral and Broden's radiographs. They found that sectioning of the calcaneofibular joint caused a 5-mm opening of the subtalar joint. With subsequent sectioning of the interosseous ligament, the joint opened up to 7 mm.

The utility of stress radiographs has been called into question by multiple authors.¹⁴⁻¹⁶ Harper¹⁴ reported a wide range of subtalar tilt with stress radiographs in his group of asymptomatic patients. Louwerens et al.¹⁵ examined 33 patients with chronic ankle instability and 10 control patients who were asymptomatic. Broden's views were checked under fluoroscopy and they detected no difference between symptomatic and asymptomatic

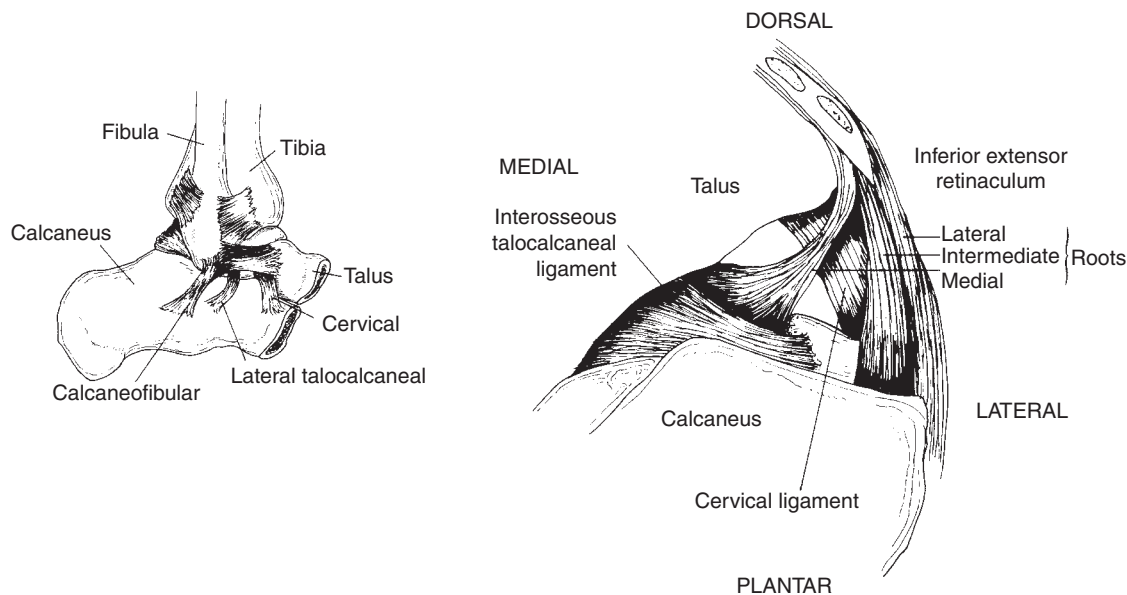


Fig. 17.2 The anatomy of the subtalar joint. (From Mann RA, Coughlin MJ, eds: *Surgery of the foot and ankle*, 7th ed. St Louis: CV Mosby; 1999: p1147, Figure 26-57.)

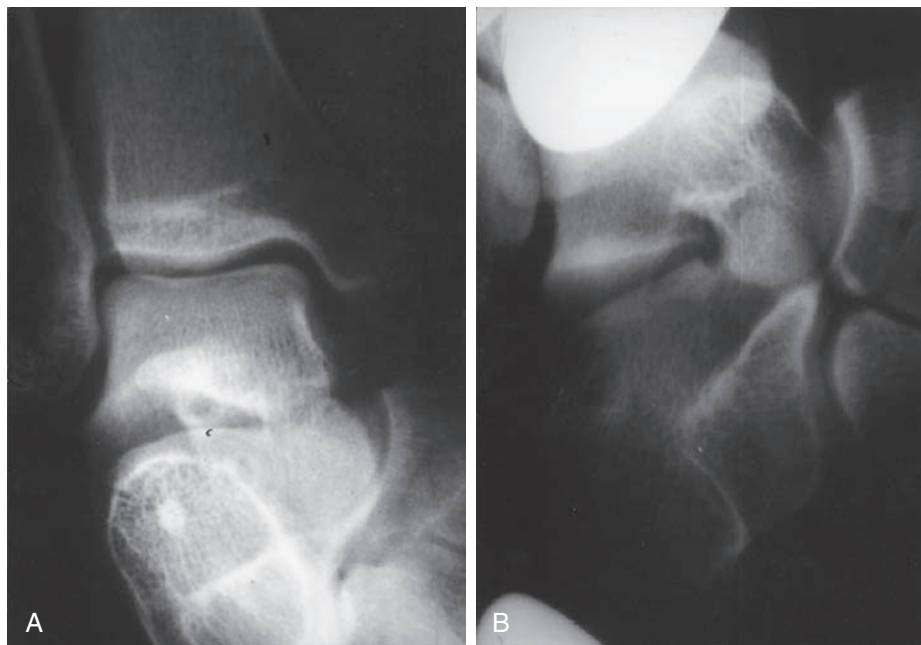


Fig. 17.3 Stress radiographs. (A) Stress anterior-posterior (AP) radiograph with subtalar tilt. (B) Stress Broden view showing subtalar instability.

feet with regard to subtalar tilt or medial shift. Van Hellemond et al.¹⁶ examined both stress radiographs and stress computed tomography (CT) scans in 15 patients with unilateral chronic ankle instability with suspected subtalar instability. Although three of the symptomatic feet and one of the asymptomatic feet had increased subtalar tilt on plain films, there was no significant difference between the symptomatic and asymptomatic sides. None of the patients had increased subtalar tilt on the stress CT scans. The authors therefore doubted that a Broden's stress examination reveals the true amount of subtalar tilt. Frost and Amendola¹⁷ reviewed the collective literature on both talar

tilt and anterior drawer stress radiographs for ankle instability and found no normative values and little agreement between studies. Nevertheless, a markedly positive stress radiograph remains the only definitive means to demonstrate the presence of subtalar instability.

It is most accurate to state that a positive stress radiograph, either in a clinic or operating room setting, is a necessary but not sufficient condition to clearly make the diagnosis of isolated subtalar instability. The false-positive rate is high, and the radiographic findings must be correlated with the physical examination and history.¹⁸

Nonoperative Treatment

In an acute injury, the standard treatment regimen for lateral ankle sprains will suffice for subtalar ligamentous injuries as well. Rest, ice, compression, and elevation (RICE) are part of a good protocol, as well as immobilization and physical therapy, when needed. The same can be said for management of chronic subtalar instability. The routine nonoperative regimen used for chronic lateral ankle instability is initiated. This may include proprioceptive training, peroneal strengthening, and bracing or strapping.^{8,19} With bracing, it is important to understand the delicate balance in providing an athlete with enough support without impeding his or her performance. Taping by an athletic trainer before participation can be effective. Wilkerson²⁰ examined a modification of the standard method of ankle taping with the incorporation of a “subtalar sling.” He found that addition of the sling enhances the protective function of taping but cautioned that it may impede performance of certain activities.

Surgical Treatment

Patients with residual symptomatic instability despite an adequate program of nonoperative management will require a surgical stabilization of their subtalar joint. If both ankle and subtalar instability exist and require surgery, both problems should be corrected at the time of surgery.⁴ Surgical stabilization involves direct ligament repair or lateral tendinosis procedures to substitute for the irreparable ligaments.

Many techniques have been described to concurrently stabilize the ankle and subtalar joint (Fig. 17.4, A through C).^{2,7,10,19-27} Many require some form of extraarticular tendon transfer to provide stability. Kato²⁸ and Pisani²⁹ described techniques focused on the subtalar joint involving intraarticular ligament reconstruction of the interosseous ligament between the calcaneus and talus.

A less invasive technique that, according to Clanton and Berson⁸ and Gould et al.,²⁵ provides a good treatment for subtalar instability is the Brostrom-Gould reconstruction technique for lateral ankle instability (Fig. 17.4, D and E). With the reconstruction of the CFL and anterior talofibular ligament (ATFL) buttressed by the inferior extensor retinaculum, subtalar stability is effectively restored.^{8,25} There is no significant drawback to its routine inclusion in lateral ankle ligament reconstruction; doing so may obviate any concern over subtle subtalar instability in many cases. When the extensor retinaculum and the fibular periosteum are sufficient to hold suture, the Gould modification of the Brostrom procedure should be performed.

No clear criteria exist to guide the use of allograft or autograft reconstruction of the lateral ankle ligaments. In patients with a previous failed reconstruction, documented history of Ehlers-Danlos syndrome, or failed contralateral reconstruction, augmentation of the repair is warranted. The technique originally described by Colville utilizes a split peroneus brevis tendon to recapitulate the ATFL and CFL in an anatomic fashion.³⁰ Rather than split a normal native tendon, O’Neil and Guyton recently described a method of reconstruction utilizing a combination of semitendinosis allograft and a braided suture construct to provide both new tissue and early strength to difficult cases with poor soft-tissue envelopes.¹⁸

The use of the braided suture anchored with interference screws has also recently been described for lateral ankle ligament reconstruction.³¹ The primary utility of the suture construct is immediate strength of the ATFL portion of the reconstruction to allow early mobilization. That same rigidity can create the hazard of limited subtalar motion if it is used across the path of the CFL. While routine use of the suture construct across the ATFL reconstruction may have significant benefit for early mobilization, extension across the subtalar joint is not necessary in most cases.

SINUS TARSI SYNDROME

Symptoms of sinus tarsi syndrome may overlap with those associated with subtalar instability. Some authors consider this syndrome simply a variant of subtalar instability.³² Sinus tarsi syndrome simply describes pain localized to the region of the sinus tarsi. Characteristic findings on clinical and radiographic examination have not been well defined. Likewise, the pathologic changes found at the time of surgery are unclear. The most widely reported description of the pathologic anatomy associated with this condition is degenerative changes to the soft tissues of the sinus tarsi.^{33,34} The majority of cases are posttraumatic but also may be related to inflammatory arthropathies, gout, ganglion cysts, and structural foot abnormalities.^{35,36}

The term *sinus tarsi syndrome* is typically used to refer to pathology emanating from the subtalar joint. It must be distinguished from extraarticular impingement of the fibula against the lateral calcaneus in a fixed or dynamic planovalgus deformity. The pain in the case of subfibular impingement is more typically located underneath the fibula and may only be present with weight bearing. The condition typically does not respond even transiently to injection of the subtalar joint, as the impingement is extraarticular. Magnetic resonance imaging (MRI) may be helpful in making the diagnosis in subtle cases; edema in the lateral talus, distal fibula, or lateral calcaneus is often present.³⁷

Clinical Presentation

The typical complaint is pain over the lateral and anterolateral ankle and hindfoot centered in the region of the sinus tarsi. The patient may report a sensation of mild hindfoot instability. It has been estimated that as many as 70% of patients with sinus tarsi syndrome have had a previous inversion injury to the hindfoot.³⁸

Excessive motion of the subtalar joint may result in temporary impingement against the fibula in the athlete participating in a cutting activity. The symptoms in these cases are transient and localized to the sinus tarsi or lateral calcaneus. The diagnosis of this entity is based primarily upon the same criteria as for subtalar instability. As in subfibular impingement, edema in the distal fibula or lateral calcaneus may be present on MRI.³⁷

Physical Examination

Tenderness over the lateral ankle and hindfoot overlying the sinus tarsi is the most common finding on clinical examination.

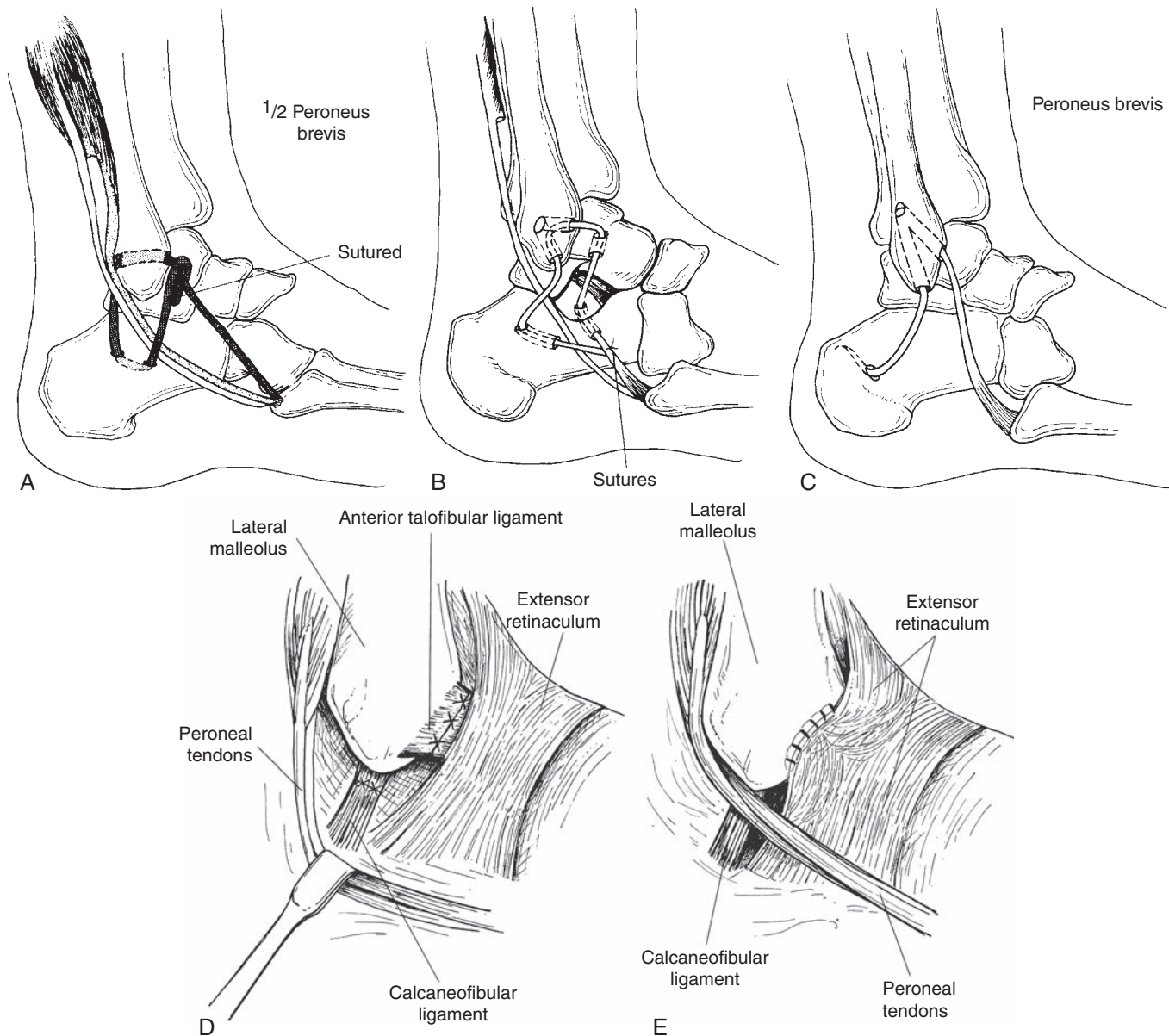


Fig. 17.4 (A) Chrisman-Snook modification of Elmslie procedure. (B) Triligamentous reconstruction. (C) Larsen procedure. (D) Lateral ankle ligament reconstruction. (E) Reinforcing repair with inferior extensor retinaculum. (From Mann RA, Coughlin MJ, eds: *Surgery of the foot and ankle*, 7th ed. St Louis: CV Mosby; 1999, A from p1128, Figure 26-35; B from p1153, Figure 26-64; C from p1127, Figure 26-34; D and E from p1128, Figure 26-36.)

Patients may have findings of mild subtalar instability; however, this is difficult to elicit and often absent. Swelling overlying the sinus tarsi is variably present.

To distinguish sinus tarsi syndrome from subfibular impingement, examination of the foot while standing and walking should be performed to assess hindfoot valgus. In more subtle cases, intraarticular injection of the subtalar joint may be performed to help distinguish the two entities. Subtalar injection is less reliable, however, at distinguishing pathology of the subtalar joint from that of the ankle and peroneal tendons; Kirk et al. demonstrated that communication on injection of the subtalar joint into these structures is remarkably common even in normal specimens.³⁹

Radiographic Evaluation

Plain films are negative in this condition. Stress views may reveal mild subtalar instability, but, as noted in the previous section, these are of uncertain value. Subtalar arthrograms have been described in the evaluation of this condition. The normal subtalar joint will accept 3 ml of contrast dye and will demonstrate multiple recesses and interdigitations within the joint capsule.²¹ Under normal circumstances there is a small recess that projects anteriorly from the subtalar joint. The absence of this synovial recess has been associated with sinus tarsi syndrome.^{35,38}

MRI has also been used in the evaluation of sinus tarsi syndrome. The key MRI features include replacement of the normal fat signal intensity in the sinus tarsi with fluid, inflammatory

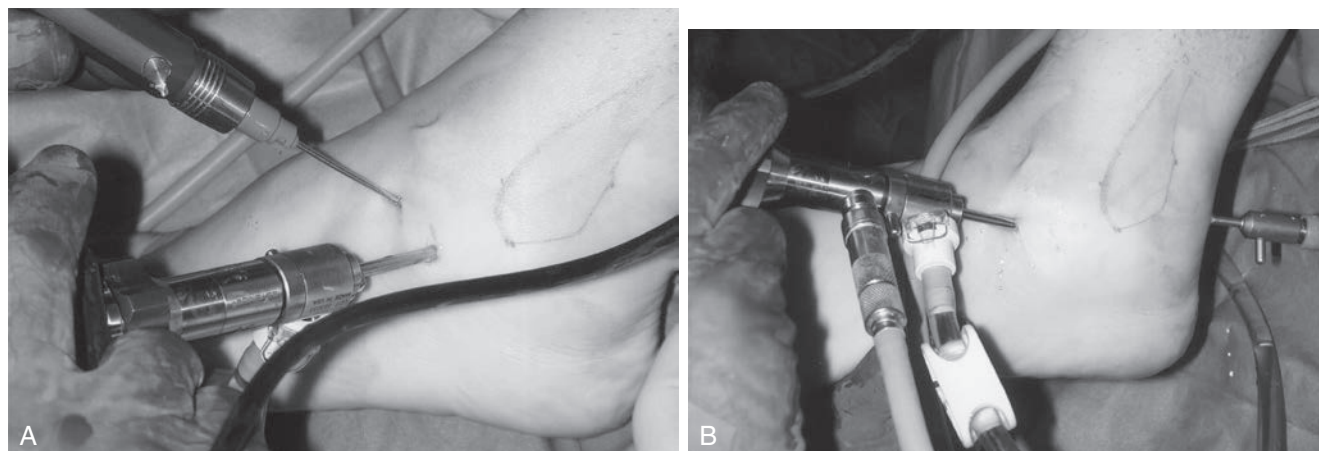


Fig. 17.5 (A) Arthroscopic examination of the subtalar joint with anterior working portal. (B) Posterior working portal.

tissues, or fibrosis.^{36,38} The inflammatory changes often will obscure the ligaments that are normally visualized in the sinus tarsi. Additional findings may include ganglion cysts and chondral changes.⁴⁰

MRI is not typically useful in the setting of acute injury, as it typically shows dramatic postinjury bony edema and is less reliable than CT for demonstrating small bony fragments that may become incarcerated in the joint.

Nonoperative Treatment

Injections of local anesthetic and steroid into the sinus tarsi may be both diagnostic and therapeutic. If the patient does not report even temporary relief following injection, then skepticism must be directed at a diagnosis of sinus tarsi syndrome. In the case of suspected subtalar intraarticular pathology, accurate placement of a diagnostic injection into the capsule of the subtalar joint is possible on a consistent basis.³⁹ Some patients may report permanent resolution of their symptoms after a series of injections.²¹ Surgery is indicated if pain recurs after a series of one to three injections.

Surgical Treatment

Open and arthroscopic techniques are available. Open excision of the tissue filling the sinus tarsi has been reported to have good results.^{21,34,35} Typically a lateral oblique incision is made over the region of the sinus tarsi. The lateral branch of the superficial peroneal nerve is avoided. The inferior extensor retinaculum and the origin of the extensor digitorum brevis are reflected distally. The sinus tarsi is entered, and debridement is performed.

Arthroscopic exploration (Fig. 17.5) of the sinus tarsi for diagnosis and treatment has been described and is preferred. Typically, a tear of the interosseous ligament and associated fibrosis is encountered. Complete decompression and wide exposure of the posterior and middle facet is required.²⁷

SUBTALAR DISLOCATION

A subtalar dislocation involves the dislocation of the talocalcaneal and talonavicular joints. With this injury there is no associated dislocation of the calcaneocuboid or tibiotalar joints. It

was first described separately by DuFaurest⁴¹ and Judcy⁴² in 1811. Broca⁴³ later classified these injuries as medial, lateral, and posterior. Dislocations are named by the direction in which the foot moves relative to the talus. In 1856, Malgaigne⁴⁴ revised this classification and added anterior subtalar dislocations as a specific entity. Frequency of the different subtypes of subtalar dislocations has been reported as 80% medial, 17% lateral, 2% posterior, and 1% anterior.⁴⁵

Clinical Presentation

These injuries may result from high-energy mechanisms such as motor vehicle accidents or falls from a height, but they also can result from a twisting athletic injury. In 1964, Grantham⁴⁶ coined the term “basketball foot” to describe medial dislocations because four of the five patients in his series injured their foot playing basketball. Low- and high-energy mechanisms create two subtypes of subtalar dislocations. High-energy injuries are more likely to be open, more likely to be lateral, have a higher incidence of associated fracture, and have a worse long-term prognosis.⁴⁷

Forced inversion of the foot results in a medial subtalar dislocation, whereas eversion causes a lateral dislocation. During medial dislocations, the sustentaculum tali serves as the fulcrum around which the foot rotates. With lateral dislocations, the foot rotates around the anterior process of the calcaneus.

Significant foot deformity is found in all patients with subtalar dislocation, although this may be somewhat obscured by swelling. Approximately 20% of subtalar dislocations involve open injuries.⁴⁸⁻⁵⁰ However, open injuries are unusual in the athlete.

Physical Examination

The deformity is usually clinically obvious. With medial dislocations, the skin is tented over the lateral malleolus and the dorsolateral talar head. With lateral dislocations, the skin is tented over the prominent medial talar head and the medial malleolus. A thorough neurovascular examination should be performed, although ischemia of the foot is uncommon with these injuries, especially in the athlete. There is a risk of local ischemia to the

soft tissues in the region of the tented skin if reduction is not prompt. Medial dislocations may reduce prior to presentation in the emergency department.

Radiographic Evaluation

The relationship of the talar head to the concave proximal side of the navicular is normally is congruent. On the lateral radiograph, the talar head lies superior to the navicular with medial subtalar dislocations. With lateral subtalar dislocations the opposite is true, and the talar head appears inferiorly displaced.

Associated fractures about the foot and ankle are common. These are better identified on postreduction radiographs. DeLee and Curtis⁵¹ reported a 47% incidence of associated osteochondral fractures of the talonavicular or talocalcaneal joints in their series of 17 patients. Osteochondral fractures were more common with lateral subtalar dislocations in this series. Other series have reported an incidence of associated foot and ankle injuries of 64% to 88%.^{49,50}

Because of the difficulty in identifying associated fractures on plain radiographs, postreduction CT scans have been recommended as a means of identifying associated injuries.⁵¹ Bohay and Manoli⁵² reported four cases of patients who had normal films following reduction of subtalar dislocations. CT scans revealed intraarticular fractures in all four cases. The authors recommended CT scanning in all patients with normal radiographs following reduction of subtalar dislocations. Diagnosis is important because associated intraarticular fractures have been associated with a poor prognosis.^{50,51,53}

Nonoperative Treatment

The majority of subtalar dislocations can be reduced using closed methods. Depending on the time from injury, reduction can be achieved with minimal sedation. The reduction process involves bending the knee to relax the gastrocnemius. Traction is applied to the heel and countertraction is applied to the thigh. As traction is being applied, the deformity is accentuated by inverting the foot for medial dislocations and everting it for lateral dislocations. The deformity then is reversed as direct pressure is placed over the prominent talar head to aid in reduction, particularly those with entrapped fragments within the subtalar joint.

Following reduction, the foot is placed into a bulky splint. Slight eversion of the hindfoot in the splint will help to stabilize medial dislocations, and inversion will hold lateral dislocations. Plain radiographs then are obtained to verify reduction. A CT scan is recommended to rule out associated fractures. Early mobilization of the uncomplicated medial subtalar dislocation with a 2-week progression to weight bearing has been supported.⁵⁴ Injuries with associated fractures will require a longer period of immobilization, typically in the range of 6 to 8 weeks. Following casting, a program of strengthening and range-of-motion exercises is initiated.

Surgical Treatment

The indications for operative intervention are open injuries and inability to achieve a congruent reduction using closed

methods. Lateral dislocations are more likely to require open reduction than medial dislocations.^{50,55} In their series of 25 patients, Bibbo et al.⁵⁰ reported that closed reduction was unsuccessful in eight patients (32%). Four of these cases had identifiable soft-tissue interposition that blocked reduction. None of the patients with a low-energy mechanism of injury required an open reduction.

Blocks to reduction with medial dislocations may include buttonholing of the talar head through the extensor retinaculum or capsule of the talonavicular joint.^{56,57} There have been reports of the deep peroneal nerve interposition blocking reduction as well.⁵⁶ Finally, the lateral edge of the navicular may impact into the medial talar head and thereby block reduction.⁵⁷ With lateral dislocations, reduction may be blocked by impingement of the posterior tibial or flexor digitorum longus (FDL) tendons and by impaction of the medial edge of the navicular onto the lateral talar head.⁵⁷⁻⁵⁹

For open reduction of medial dislocations, a longitudinal anteromedial incision is made along the talar neck extending to the talar head. This allows access to the structures that have entrapped the talar head. At the same time, inspection of impaction fractures of the articular surfaces can be carried out. For lateral dislocations, a more medial longitudinal incision is made over the prominent talar head. Interposed tendons are released and joint surfaces are inspected. Any tears found in the tendons should be repaired.

TARSAL COALITION

Tarsal coalition involves a congenital developmental failure of separation between two or more tarsal bones. This coalition may be bony or fibrous. The two most common locations for coalition are at the talocalcaneal and calcaneonavicular joints. These locations account for approximately 90% of all coalitions.⁶⁰ Less commonly, coalitions have been described at the talonavicular, calcaneocuboid, navicular cuneiform, and cuboid navicular articulations.

Previously it had been suggested that the etiology of tarsal coalition involved the incorporation of accessory ossicles into adjacent tarsal bones.⁶¹ In 1955, Harris⁶² performed microscopic dissection of fetal hindfeet and demonstrated a failure of mesenchymal separation. This failure of segmentation has become the most widely accepted theory regarding the etiology of this disorder. It generally is described as an autosomally dominant disorder with incomplete penetrance.^{63,64}

The incidence of tarsal coalition has been estimated to be less than 1%.⁶⁵ The incidence was likely underestimated before the use of CT scans. Further confounding the incidence is the asymptomatic nature of a large percentage of coalitions. In 1974, Leonard⁶⁶ studied the first-degree relatives of 31 patients with tarsal coalition. He found that 39% of the first-degree relatives had coalitions on radiographs, but all were asymptomatic. Approximately 50% of coalitions are bilateral, with calcaneonavicular coalitions more likely to occur bilaterally.^{65,67}

Clinical Presentation

Children with hindfoot coalition are often asymptomatic until ossification of the fibrous or cartilaginous coalition occurs. Before this time, some degree of motion is preserved at the affected joint. Once the coalition ossifies, the motion at the affected joint is lost and symptoms may arise. The timing of this ossification may vary, depending on the location of the coalition. Patients with calcaneonavicular coalitions may become symptomatic earlier (age 8–12 years) than patients with talocalcaneal coalitions (age 12–16 years).⁶⁸

Patients with tarsal coalition can present with pain, stiffness, and/or a deterioration of athletic performance. Increased stresses are placed on surrounding structures as motion in the hindfoot is restricted, and this may lead to pain. Although a planovalgus position of the foot has been classically described, feet with normal arches or even a cavovarus deformity may contain a coalition.⁶⁹ The symptoms are often low grade and not severe enough to prompt a visit to the doctor until a traumatic event causes a flare-up of pain.

Recurrent ankle sprains often are described in athletes with tarsal coalitions.⁷⁰ Forced motion beyond that which can be accommodated by the abnormal joints may lead to partial or complete ligamentous injuries. The abnormal joints are unable to dissipate the forces generated by athletic activities, and therefore the increased stresses are transferred to the ligamentous structures.

Persistent medial sustentacular pain in the presence of ankle instability may represent a low-grade coalition identifiable only as arthrofibrosis of the middle facet with a positive bone scan.⁷¹

The age of the patient is an important consideration in determining treatment. All isolated calcaneonavicular coalitions should undergo resection regardless of age, but the talocalcaneal coalition often fails with simple resection in patients beyond early adulthood. It is likely that these older patients did not develop symptoms early because they had more extensive or stiffer coalitions to begin with until a later mechanical disturbance occurred. The return of subtalar motion following talocalcaneal resection in the adult is typically poor; subtalar fusion is a more reliable option in these cases.

Whether or not a patient participates in athletics should not be a consideration in surgical decision making if a talocalcaneal coalition is complete. No alternative guidelines for resection versus fusion exist in athletes, and the patient who has already become a successful athlete with a stiff subtalar joint will be no less successful if that joint remains stiff but becomes painless as a result of a fusion procedure.

A dorsal talar beak is often present in cases of a congenitally stiff hindfoot including coalition. There is no evidence that resection of the talar exostosis improves motion or reduces pain from the coalition site itself; however, the prominence may itself be painful with footwear and can be resected if this is the case.

Physical Examination

Patients tend to have a rigid flatfoot involving heel valgus, loss of the midfoot arch, and abduction of the forefoot.

PEARL

This should be differentiated from an asymptomatic flexible flatfoot, in which heel varus and medial arch is restored with single-foot and double-foot heel rise.

The degree of the deformity can be quite variable. Talocalcaneal coalitions are associated with a more severe hindfoot valgus deformity than coalitions at other sites.⁷² Talocalcaneal coalitions typically eliminate motion of the subtalar joint.

PEARL

Calcaneonavicular coalitions may cause only a partial reduction of hindfoot motion.

The patient may be tender about the hindfoot/midfoot, depending on the location of the coalition. Calcaneonavicular coalitions often cause anterolateral tenderness directly over the joint. Talocalcaneal coalitions may cause lateral tenderness over the sinus tarsi and peroneal tendons, as well as medially over the sustentaculum. A bony eminence from talocalcaneal coalitions has been described as a cause of tarsal tunnel symptoms. In one series, 30% of patients with tarsal tunnel syndrome were found to have an eminence from a talocalcaneal coalition as a source of the symptoms.⁷³

Peroneal spasm may or may not be present. This finding has been suggested as part of the classic presentation of this disorder; however, it is found only in the minority of cases.^{65,74}

Radiographic Evaluation

Initial evaluation of the patient should include weight-bearing AP, lateral, and oblique radiographs of the affected foot. An axial heel view should be added to these three views of the foot so that the talocalcaneal joint can be inspected. These may identify the presence of a coalition and degenerative changes in the surrounding joints. A calcaneonavicular bar is best seen on the 45-degree medial oblique view. A lateral x-ray may show the “anteater nose,” a projection from the anterior process of the calcaneus to the navicular (Fig. 17.6, A) that is a sign of the calcaneonavicular coalition.⁷⁵ The axial heel view is the best plain radiograph for diagnosing coalitions of the middle facet of the subtalar joint, but a CT is required to exclude the diagnosis. Secondary signs of a talocalcaneal coalition also may be detected on the lateral view. These include narrowing of the posterior facet of the subtalar joint, blurriness of the middle facet of the subtalar joint, beaking of the dorsal head of the talus, and rounding of the lateral process of the talus.⁷⁶

CT has been established as the gold standard study for the identification of talocalcaneal coalitions.^{77,78} A CT scan (Fig. 17.6, B) allows one to identify the coalition, determine the extent of joint involvement, and assess any areas of surrounding degenerative changes. It can be particularly useful for preoperative planning and determining whether a coalition is resectable. It also may be used postoperatively to assess the completeness of resection, progressive degenerative changes, and recurrence of the coalition.

Less commonly, MRI has been used in the workup of tarsal coalitions (Fig. 17.6, C). It may better identify nonosseous coalitions.⁷⁹ The surrounding joints and soft tissues can be evaluated as well. A radionuclide bone scan also may be useful in the diagnosis of the symptomatic patient with suspected tarsal coalition, particularly as a screening procedure.⁸⁰ This test can be positive when the patient is symptomatic. Accumulation of

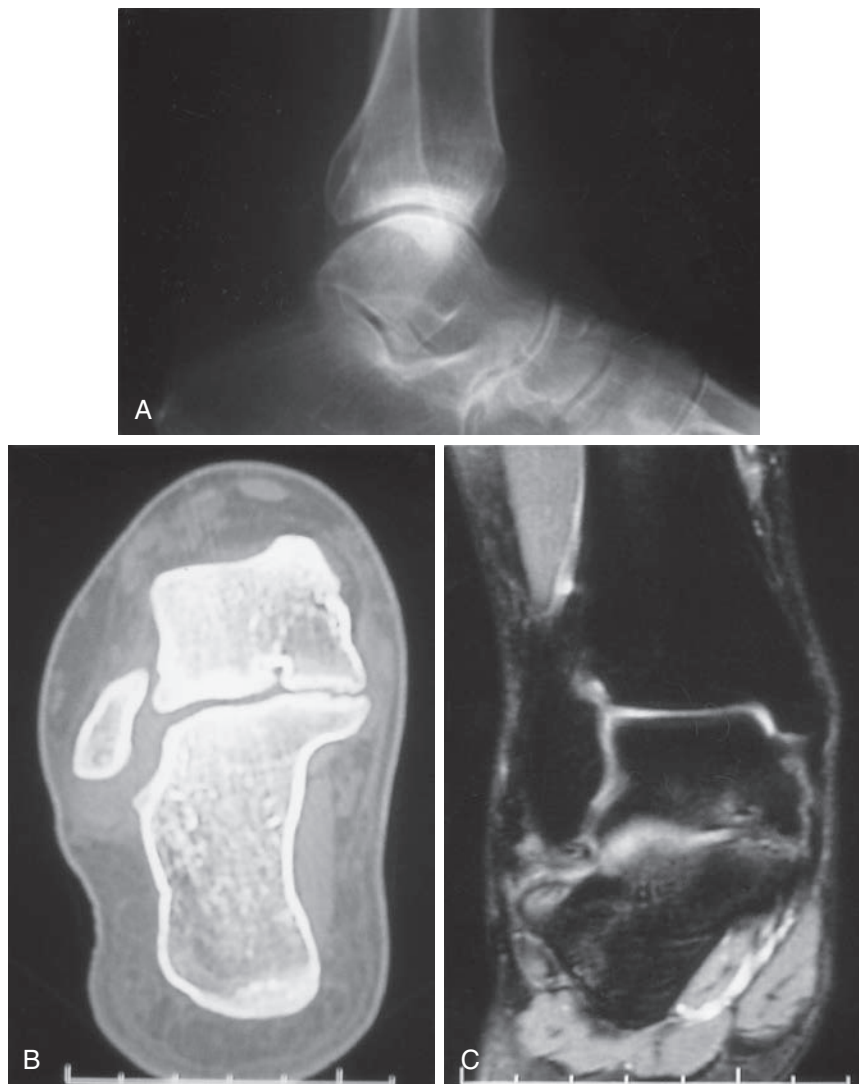


Fig. 17.6 (A) Radiograph of calcaneonavicular coalition, with “anteater nose” projection from anterior process of calcaneus to navicular. (B) Computed tomography scan. (C) Magnetic resonance imaging of middle facet coalition.

the radionuclide most likely is the result of increased stresses at the surrounding joints or within the coalition itself.

Nonoperative Treatment

Typically, a trial of nonoperative management is indicated in the treatment of tarsal coalitions. A study by Jayakumar and Cowell⁸¹ in 1977 found that one-third of their patients responded favorably to conservative treatment. When the diagnosis is made in the adolescent who is a competitive athlete, definitive treatment on a more expedient basis may be appropriate. In this manner, the time off from competition may be reduced. Morgan and Crawford⁸² looked at 12 adolescent athletes with coalitions (8 calcaneonavicular and 4 talocalcaneal). Nonoperative treatment was successful in none of the patients, and 8 of the 12 elected to undergo surgery.

The usual regimen of nonoperative management for patients with mild symptoms includes antiinflammatory medications and orthotics. For more severe symptoms, patients may undergo a trial of a short-leg walking cast for a

period of 6 weeks. If the patient responds favorably to immobilization, then orthoses are used. The patient is considered to have failed nonoperative treatment if pain persists after two cast applications.

Surgical Treatment

The most common procedures performed for tarsal coalition include resection of the coalition, selected arthrodesis, and triple arthrodesis. Previous reports have examined resection of tarsal coalitions in adolescent athletes. In Morgan and Crawford's⁸² review of 12 adolescent athletes, they reported their results in 8 athletes who underwent resection of tarsal coalitions. They found that five out of six athletes who had calcaneonavicular bars were able to return to play. Both athletes with talocalcaneal bars were also able to return to play following resection. Elkus⁸³ examined 15 feet with calcaneonavicular coalitions and 8 with talocalcaneal coalitions in a population of young athletes. All patients underwent resection of their coalitions with or without soft-tissue interposition. The majority of the patients

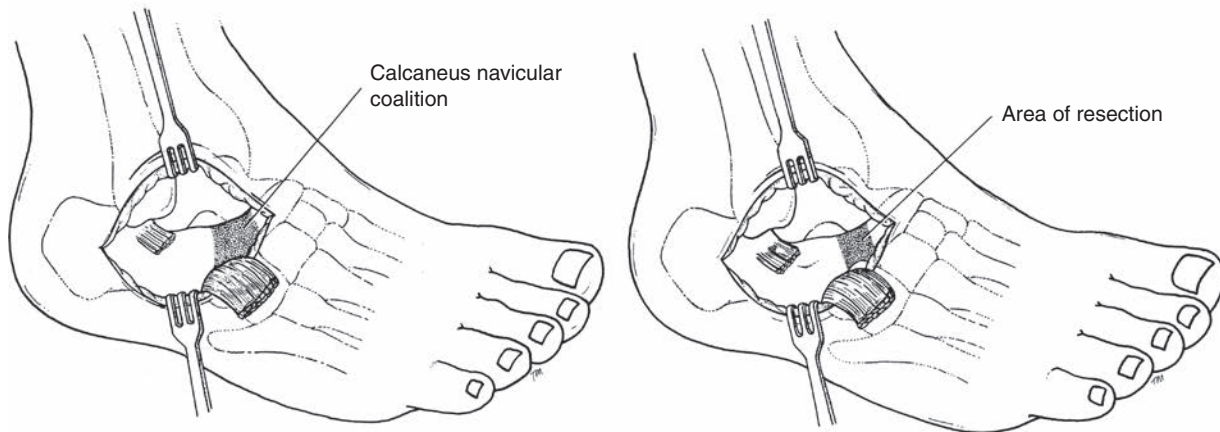


Fig. 17.7 Drawing demonstrating excision of calcaneonavicular coalition and interposition of extensor brevis. (A) Skin incision. (B) Exposure of the extensor brevis. (C) Reflection of the extensor brevis forward demonstrates the area of coalition. (D) Demonstration of the area of coalition to be resected. (E) Interposition of the extensor brevis muscles. (From Mann RA, Coughlin MJ, eds: *Surgery of the foot and ankle*, 6th ed. St Louis: CV Mosby;1992.)

had relief from pain (no numbers reported) with variable return of subtalar motion. The author did note that all eight cases of talocalcaneal bar resection had improvement in motion, had decreased pain, and were able to return to athletic activity.

CALCANEONAVICULAR COALITIONS

The primary treatment for calcaneonavicular coalitions is resection unless degenerative changes are present in the subtalar or midtarsal joints. Although talar beaking previously was thought to be evidence of degenerative changes, it is not a contraindication to resection.⁸⁴ One contraindication to resection is the presence of a concomitant talocalcaneal coalition. Generally, the bar should be resected during adolescence, but resection of bars in the adult population has been shown to be beneficial as well.⁸⁵ There is evidence that results are better after resection of fibrous coalitions rather than bony bars.⁸⁶

The reported outcomes following surgical excision of the coalition have been variable. Cohen et al.⁸⁵ in 1996 reviewed their results when resecting coalitions in adults. They examined 12 patients, 77% of whom displayed degenerative changes before resection. All but two of the patients reported subjective relief of the preoperative symptoms. Gonzalez and Kumar⁸⁶ reported on 75 feet in 48 patients with calcaneonavicular coalitions. Their results with resection and interposition with the extensor digitorum brevis muscle was good or excellent in 77% of the patients. The authors noted that their best results were in patients who had a fibrous coalition and who were younger than 16 years. In contrast, Andreasen⁸⁷ reported results of 31 bar resections that were examined 10 to 22 years following surgery. He found 30% of the patients had mild pain and 26% had severe pain. A recurrence of the bar was seen in 67% of patients, and 96% of feet had osteoarthritic changes. Six patients required triple arthrodesis.

No material for interposition into the resection site has been demonstrated to be superior to any other or to simple resection.

Technique of Resection of Calcaneonavicular Coalition

A 4-cm incision is made over the calcaneonavicular interval, exposing the fascia overlying the extensor digitorum brevis. One should avoid branches of the superficial peroneal and sural nerves. The extensor digitorum brevis is reflected distally, exposing the calcaneocuboid joint, sinus tarsi, and calcaneonavicular coalition. The coalition is resected in parallel cuts from each surface, avoiding convergence. K-wires can be used to plan the cuts. A total resection of at least 1 cm is desirable. The hind-foot is mobilized to test for adequate subtalar motion. Bone wax is generously packed into the bony surfaces. Closure is done in layers (Figs. 17.7 and 17.8).

TALOCALCANEAL COALITIONS

Resection of the coalition also is the treatment of choice in patients with symptomatic talocalcaneal coalitions. Skeletally immature patients with smaller bars and no evidence of degenerative changes in the subtalar joint are most likely to benefit from resection.⁷⁸ Contraindications to resection include patients with rigid flatfeet or degenerative changes of the subtalar and transverse tarsal joints. These patients are better served with a subtalar or triple arthrodesis. In carefully selected patients, generally 80% to 90% will report satisfactory results following a resection.⁸⁸⁻⁹⁰

The decision whether to resect the coalition or perform a fusion may be influenced by the size of the bar. Some feel that involvement of more than one-half of the joint will preclude a successful resection.⁹⁰ Wilde et al.⁹¹ reported unsatisfactory outcomes with middle coalition resection and fat interposition in the presence of middle facet coalition area greater than 50% of the area of the posterior facet. On the other hand, Kumar et al.⁸⁹ did not find a correlation between the extent of middle facet coalition and the postoperative results in 18 feet on which resection was performed. In general, the quality of the residual

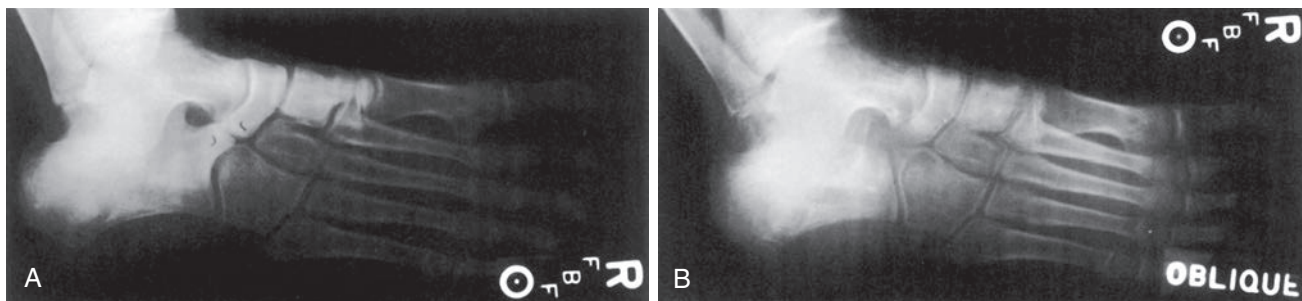


Fig. 17.8 Calcaneonavicular coalition. (A) A 45-degree oblique view of the foot demonstrates the calcaneonavicular coalition. (B) Postoperative 45-degree oblique view of the foot demonstrates adequate excision of the calcaneonavicular coalition.

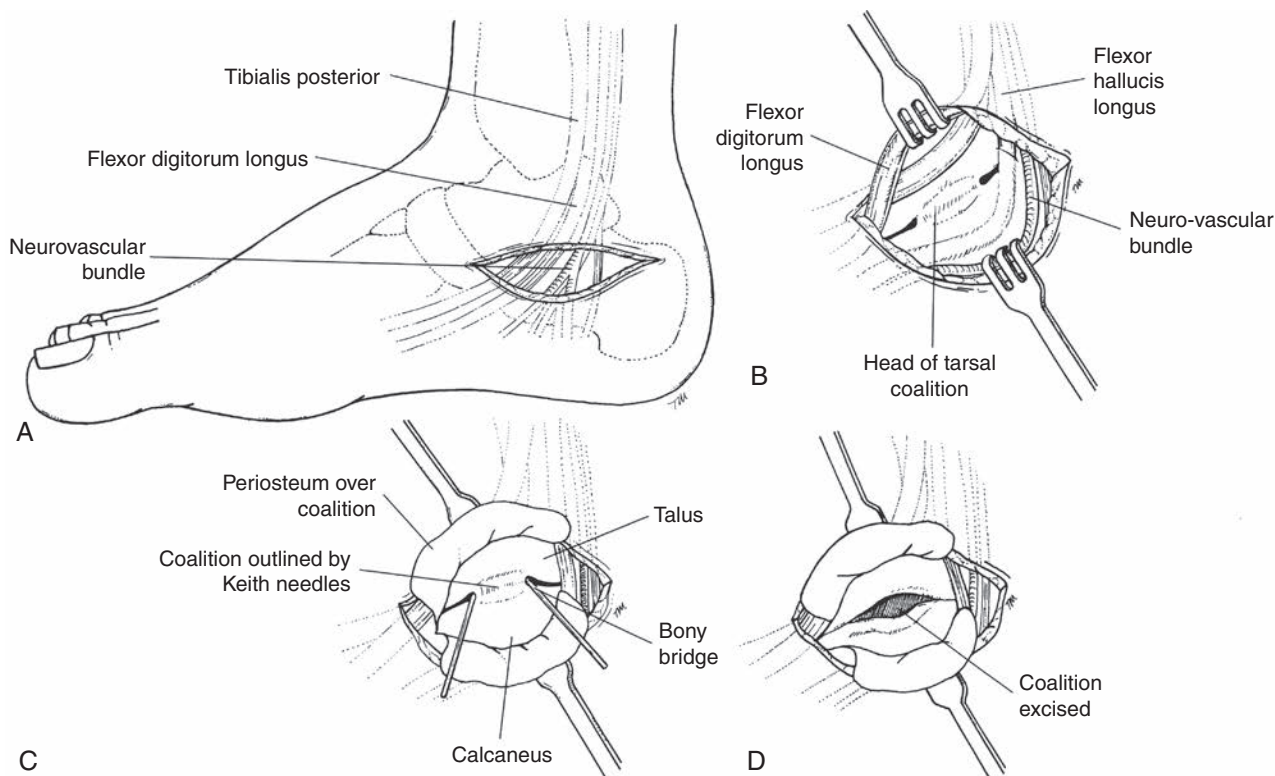


Fig. 17.9 Excision of talocalcaneal coalition. (A) Skin incision. (B) Reflection of structures dorsally and plantarward to expose the area of coalition. (C) Outlining the coalition with needles. (D) Postexcision appearance of the coalition. (From Mann RA, Coughlin MJ, eds: *Surgery of the foot and ankle*, 6th ed. St Louis: CV Mosby;1992.)

posterior facet of the subtalar joint is likely more important than the extent of the area involved with the coalition.

Mosca and Bevan⁹² reported the concurrent correction of pes planus deformity using a calcaneal lengthening osteotomy in eight patients. They emphasized the importance of concurrent deformity correction, but no comparative group undergoing resection alone was reported.

Technique of Resection of Talocalcaneal Coalition

A 6- to 7-cm linear incision is made just below the medial malleolus, just above the sustentaculum tali (Figs. 17.9, A and 17.10, A). Tenotomy scissors are used to dissect and identify the

posterior tibial tendon, FDL, and tibial neurovascular bundle. The middle facet lies just under the FDL, often covered by minimal periosteum. The middle facet with coalition is identified and dissected, showing the extent of the coalition (Figs. 17.9, C and 17.10, B). The tarsal tunnel can be entered with a wire or probe anteriorly to assist with orientation. Once the corners of the coalition are identified, excision is done using small straight osteotomes and rongeurs. The excised surfaces should be parallel to prevent contact and potential osseous fusion (Figs. 17.9, D and 17.10, C). Bone wax may be packed on the bony resection surfaces to reduce bleeding. Closure of the FDL sheath is included in the layered closure.

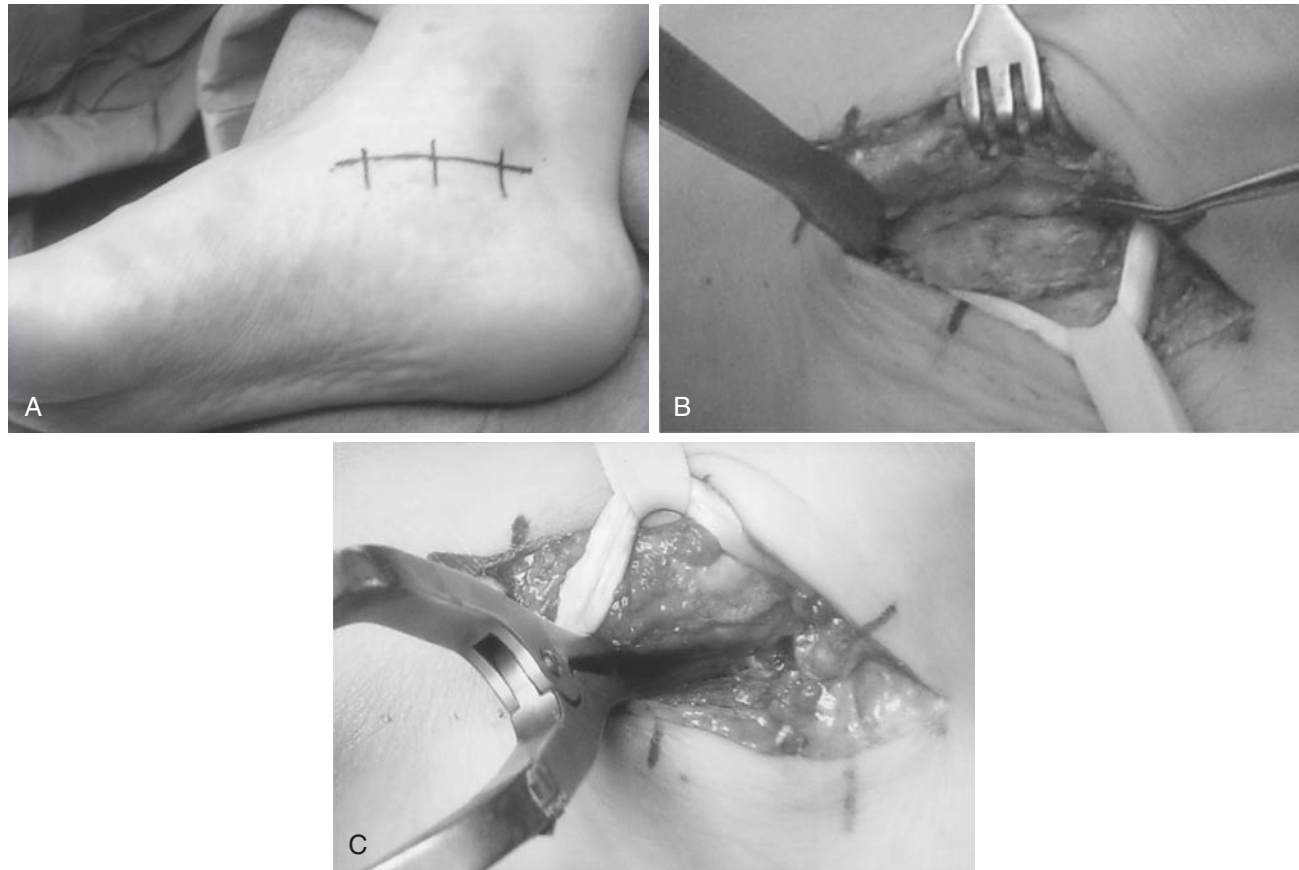


Fig. 17.10 (A) Incision marked on medial hindfoot. (B) Middle facet coalition with corners defined, flexor digitorum longus retracted inferiorly. (C) Coalition excised, flexor digitorum longus retracted superiorly.

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Diagnostic and Operative Ankle and Subtalar Joint Arthroscopy

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HISTORY OF THE TECHNIQUE

Arthroscopy has revolutionized the practice of orthopedic surgery since the mid-1970s. After a long history of sporadic attempts at arthroscopy, technologic breakthroughs in Japan and several surgical pioneers in North America launched widespread interest in percutaneous joint surgery. In 1939, Tagaki was the first to introduce systematic arthroscopic assessment of the ankle in the literature.¹ More than 30 years later, Watanabe published a series of 28 ankle arthroscopies in 1972,² followed by Chen in 1976 and several publications in the 1980s.^{3,4} The rapid rise of the popularity of foot and ankle arthroscopy over the last 20 years is partly because other noninvasive techniques cannot adequately diagnose disorders in these joints. To operate in the central and posterior ankle, some type of distraction device is needed. Invasive external distraction was tried in the early 1980s. The first noninvasive technique was described by Yates and Grana in 1988.⁵ With the advent of better small-joint arthroscopes and instrumentation, the production of more efficient noninvasive distraction devices, the development of

tendoscopic surgery, and the introduction of a two-portal technique for posterior ankle problems, ankle arthroscopy further developed to the current state.

Nowadays, arthroscopy of the ankle joint has become the most important diagnostic and therapeutic procedure for chronic and posttraumatic complaints of the ankle joint and became an integral part of modern orthopedic surgery. The dynamic nature of arthroscopy, moreover, necessitates constant improvements that will continue to allow this field to grow. In order to optimize the practice of arthroscopic procedures, a firm understanding of their subtle refinements, limitations, and risks is fundamental.

INDICATIONS AND CONTRAINDICATIONS

The key in assessment of ankle joint pathology is clinical assessment of the patient. By means of a clinical diagnosis, an indication is set for an arthroscopic intervention and the clinical diagnosis is essential for preoperative planning.⁶ The clinical

diagnosis is based on history, symptoms and signs, and radiographic examination. Anterior problems of the ankle include soft-tissue or bony impingement, synovitis, loose bodies, or ossicles. More centrally located complaints can originate from an osteochondral lesion or arthrosis, whereas posterior problems can be caused by intra-articular pathology, such as posterior impingement syndrome (os trigonum); posttraumatic calcifications; loose bodies or synovitis; or by periarticular posterior ankle pathology, such as peroneal tendon, posterior tibial tendon, or flexor hallucis longus (FHL) pathology. In posterior ankle disorders, especially, differentiation from subtalar pathology is sometimes difficult.

The relative contraindications for ankle arthroscopy include moderate degenerative joint disease with restricted range of motion, a significantly reduced joint space, severe edema, and tenuous vascular status.⁷ The absolute contraindications for ankle arthroscopy include absence of a clinical diagnosis, severe degenerative joint disease, and localized soft-tissue infection. However, if septic arthritis is present, ankle arthroscopy is indicated, since it is a useful tool for drainage, debridement, and lavage of the joint.⁸

SURGICAL TECHNIQUE—PREPARATION

Operative Setup

In general, the procedure is carried out as outpatient surgery under general anesthesia or epidural anesthesia. Patients can be placed in various positions according to the surgeon's preference, with a supine position with slight elevation of the ipsilateral buttock being mostly used. The heel of the affected foot rests on the very end of the operating table in order to make it possible for the surgeon to fully dorsiflex the ankle by leaning against the sole of the patient's foot. For the treatment of posterior ankle problems, the patient is placed in prone position and a small support is placed under the lower leg, making it possible to move the ankle freely (Fig. 18.1). In both settings, a tourniquet is placed around the upper thigh.

There are some important considerations in deciding whether to use dorsiflexion or traction for routine anterior ankle arthroscopy. When saline is introduced in the dorsiflexion position, the anterior working area and any bony or soft-tissue impediment in front of the medial malleolus, in front of the lateral malleolus, at the talar neck, or at the distal tibia can be adequately visualized and treated. For the treatment of anterior impingement lesions, synovitis, ossicles, and loose bodies, it therefore is beneficial to perform the procedure without distraction. In this dorsiflexed position, the talus is concealed in the joint, thereby protecting the cartilage from potential iatrogenic damage. Moreover, loose bodies usually are located in the anterior compartment of the ankle joint and with dorsiflexion creating an anterior working area, removal is facilitated.⁹ On the other hand, distracting the joint makes it possible for the loose body to fall into the posterior aspect of the joint, thus making removal more difficult or impossible by an anterior approach. The same is true for the removal of ossicles and bony spurs by a chisel or burr. Distraction of the joint results in tightening of the anterior capsule, thus making it more difficult to identify



Fig. 18.1 For posterior ankle arthroscopy, the patient is placed in prone position. A tourniquet is applied and a small support is placed under the lower leg, making it possible to move the ankle freely.

anterior osteophytes, ossicles, loose bodies, and soft-tissue impediments. Furthermore, when portals are created and instruments are introduced in the distracted position, this may result in iatrogenic cartilage damage at the talar dome.⁶

The main reason for inspection of the talar dome and tibial plafond is for treatment of an osteochondral lesion. A clinical diagnosis must be established preoperatively using history, physical examination, and standard x-rays. In case of doubt about the existence or the exact location and size of a defect, a preoperative spiral computed tomography (CT) scan or magnetic resonance imaging (MRI) can be performed. Knowing the exact location of a defect makes it possible to decide preoperatively whether distraction will be necessary or whether the osteochondral lesion can be approached in a forced plantarflexed position of the foot. In our experience, more than 90% of medial and lateral talar dome lesions can be treated in a hyperplantarflexed position.⁶ Distraction may be beneficial when an osteochondral lesion is located in the posterior part of the medial or lateral talar dome, the tibial plafond, or when a soft-tissue impediment, ossicles, or an impregnated loose body is located in the joint space between fibula and tibia (intrinsic syndesmotic area).^{10,11} For posterior ankle problems, for example an osteochondral lesion in the posterior quarter of the talar dome or in the posterior part of the tibial plafond, two-portal posterior ankle arthroscopy is an important alternative (Fig. 18.2).¹²

Arthroscopic equipment

Both a 4.0-mm and a 2.7-mm arthroscope with 30-degrees obliquity can be used for ankle arthroscopy, depending on the indication. Small-diameter, short arthroscopes yield an excellent picture that is difficult to distinguish from a standard 4.0-mm scope. The small-diameter arthroscope sheath, however, cannot deliver the same amount of irrigation fluid per time as the standard sheath, causing an important drawback when motorized instruments are used, since these cases must benefit from an adequate amount of irrigation fluid. For routine arthroscopic procedures such as anterior impingement syndrome, loose body removal, treatment of synovitis, and the vast



Fig. 18.2 Two-portal posterior ankle arthroscopy is an important alternative for the treatment of posterior ankle problems.

majority of osteochondral defects, it is beneficial to use the 4.0-mm arthroscope. A 2.7-mm arthroscope should be reserved for the treatment of osteochondral lesions of the posterior third of the talar dome (when not approached by a posterior ankle arthroscopy), pathology of the articular part of the tibiofibular joint, such as a soft-tissue impediment or impregnated ossicles or loose bodies, or other posterior ankle problems that are treated by an anterior approach. Use of a 2.7-mm scope usually necessitates the creation of a third posterolateral portal to maintain adequate flow in the joint.

Instrumentation

An 18-gauge spinal needle is used to distend the joint and to locate the anterolateral portal by allowing precise positioning under direct vision of the portals. The probes used in ankle arthroscopy should be about 1.5 mm in diameter to reach the small recesses of the gutters and to lift up under loose articular cartilage. An angled tip is desirable to touch over the dome-shaped talus and flat tibia. Another important instrument is the grasper. For the removal of small, loose bodies in soft tissue, a flat-tipped grasping forceps with fine teeth can be used. For larger loose bodies and soft-tissue fragments, a cup-shaped, jaw-grasping forceps with serrated edges is better. Small-joint basket forceps with different tip designs help to remove soft-tissue and chondral fragments. Various small-joint curettes, either straight or curved, are particularly valuable for removing osteochondral lesions and trimming of articular cartilage edges. Small-joint osteotomes and chisels are available to remove osteophytes and ossicles and can facilitate tissue elevation. Sometimes a small periosteal elevator can be useful. Motorized instruments can excise larger volumes of tissue than conventional hand instruments and suction it quickly out of the joint. They also can be used for debridement of large osteochondral lesions. A power burr is useful for abrading or excising hard bone fragments.

Irrigation

Different fluids can be used for arthroscopic irrigation during ankle and foot arthroscopy. Lactated Ringer is used most



Fig. 18.3 Left ankle. The anteromedial portal is placed just medial to the anterior tibial tendon at the joint line. Care must be taken not to injure the saphenous vein and nerve transversing the ankle joint along the anterior edge of the medial malleolus.

common because it is physiologically compatible with articular cartilage and is rapidly reabsorbed if extravasated from the joint. Other options include glycine and normal saline. When a 4-mm arthroscope is used, gravity inflow usually is adequate if the fluid is introduced through the arthroscope sheath. When a 2.7-mm arthroscope is used, the gravity inflow should be introduced through a separate (posterolateral) cannula. An alternative is to use an arthroscopic pumping device.

SURGICAL TECHNIQUE—PORTALS

Portals provide an entry to visualize the structures of the ankle and foot. In order to perform adequate diagnostic and therapeutic arthroscopy, proper portal placement is critical.¹³ Numerous portals for arthroscopy of the ankle have been described in literature. In general, these portals can be grouped into a) anterior, b) posterior, c) transmalleolar, and d) transtalar.

In routine ankle arthroscopy, two primary portals are used: the anteromedial and the anterolateral portal. Some authors, however, recommend routine placement of posterior portals in ankle arthroscopy. In these cases, a posterolateral portal is recommended. Because of the potential for serious complications, most authors feel that the posteromedial portal is contraindicated when performing anterior ankle arthroscopy.¹⁴

Anterior Portals

Anteromedial Portal

The anteromedial portal should be made first, since it is easy to access. This is especially true with the ankle in hyperdorsiflexion. The exact point of entry in this position is easily reproducible, and the risk of neurovascular damage is minimal. The anteromedial portal is placed just medial to the anterior tibial tendon at the joint line (Fig. 18.3). In the hyperdorsiflexed position, a local depression can be palpated. In the horizontal plane, this depression is located between the anterior tibial rim and the talus. During palpation the thumb first detects the interval in the horizontal plane and subsequently locates the vertical

position. In the vertical position, the anterior tibial tendon is the landmark. One should palpate the anterior tibial in the dorsiflexed position. In this dorsiflexed position the anterior tibial tendon moves 1 cm lateral. The location of the anteromedial portal now can be marked onto the skin just medial from the anterior tibial tendon. Care must be taken not to injure the saphenous vein and nerve traversing the ankle joint along the anterior edge of the medial malleolus. By moving the ankle joint from the plantarflexed position to the dorsiflexed position, the talus can be felt to move in relation to the distal tibia. The thumb gets locked into the soft spot in the hyperdorsiflexed position. A small longitudinal incision is made through the skin only just medial from the anterior tibial tendon. Blunt dissection is performed with a mosquito clamp through the subcutaneous layer and through the capsule into the ankle joint. With the ankle in the forced dorsiflexed position, cartilage damage is avoided. In this forced dorsiflexed position, the arthroscope shaft with the blunt trocar is introduced. When the trocar is felt to contact the underlying bony joint line, the shaft with the blunt trocar is gently pushed further into the anterior working area in front of the ankle joint toward the lateral side. The anterior compartment is irrigated and inspected. The next portal to make is the anterolateral portal.

Anterolateral Portal

The anterolateral portal is the second standard anterior portal. In general, it is placed just lateral to the tendon of the peroneus tertius at or slightly proximal to the joint line (Fig. 18.4) and is made under direct vision by introducing a spinal needle. In the horizontal plane, it is situated at the level of the joint line. In the vertical plane, the anterolateral portal is located lateral to the common extensor tendons and the peroneus tertius tendon. Care must be taken to avoid the superficial peroneal nerve because it runs subcutaneously. This subcutaneous nerve often can be palpated or visualized by placing the foot in forced hyperplantarflexion and supination. The intermediate dorsal cutaneous branch of the superficial peroneal nerve crosses the anterior aspect of the ankle joint superficial to the common extensor tendons. Damage to this branch can be avoided by staying lateral to the extensor tendons. Once the lateral branch is identified, its position can be marked with a marking pen on the skin.

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

Accessory Inferior Anteromedial and Anterolateral Portals

Accessory portals can facilitate instrumentation or the introduction of fluid in specific indications. Separation between the main portal and the accessory portal should be at least 5 mm in order to allow proper instrumentation and triangulation while

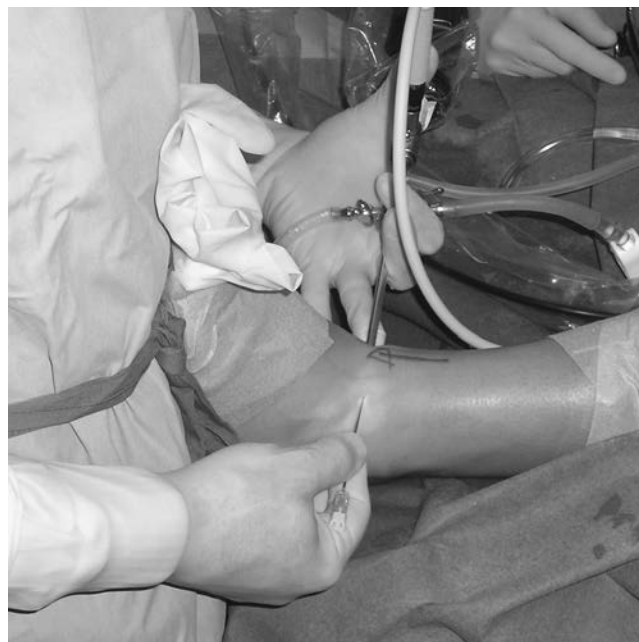


Fig. 18.4 Left ankle. The anterolateral portal is placed just lateral to the tendon of the peroneus tertius at or slightly above the joint line.

minimizing the risk of necrosis. The lateral accessory portal is placed just below the anterior talofibular ligament. After introduction of a spinal needle, a skin incision is made under direct vision in line with the anterior talofibular ligament. The medial accessory portal is placed in. On the medial side, after locating the portal with the spinal needle, the incision is made in line with the fibers of the deltoid ligament. The knife can be introduced directly into the joint under direct vision.

Posterior Portals

Posterolateral Portal

The posterolateral portal is made 1.2–2.5 cm proximal to the tip of the lateral malleolus, just lateral to the Achilles tendon (Fig. 18.5, A). Both the small saphenous vein and the sural nerve are in close proximity and therefore may be at risk for damage.¹⁴ After making a vertical stab incision, the subcutaneous layer is split by a mosquito clamp. The mosquito clamp is directed anteriorly, pointing in the direction of the interdigital webspace between the first and second toe (Fig. 18.5, B). When the tip of the clamp touches the bone, it is exchanged for a 4.5-mm arthroscope shaft with a blunt trocar pointing in the same direction. By palpating the bone in the sagittal plane, the level of the ankle joint and subtalar joint most often can be distinguished because the prominent posterior talar process can be felt as a posterior prominence between both joints. It is not necessary to enter either joint capsule. The blunt trocar is situated extra-articular at the level of the ankle joint. Next, the blunt trocar is exchanged for a 30-degree, 4.0-mm arthroscope. In order to prevent damage to the lens system, the direction of view should be lateral.

Posteromedial Portal

This portal is made second, just medial to the Achilles tendon. In the horizontal plane, it is located at the same level as the

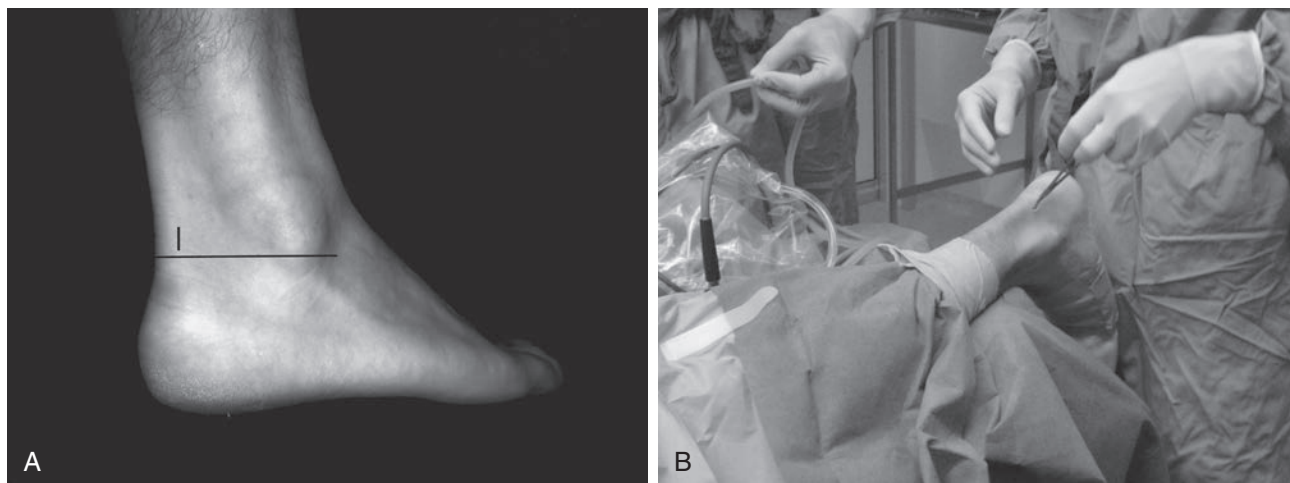


Fig. 18.5 (A) Right ankle. The posterolateral portal is made level to or slightly above the tip of the lateral malleolus just anterior to the Achilles tendon. (B) Left ankle. The mosquito clamp is directed toward the first interdigital web space.

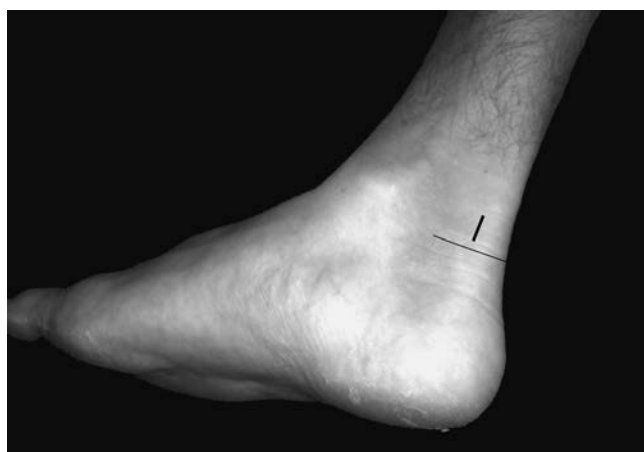


Fig. 18.6 Right ankle. The posteromedial portal is made just medial to the Achilles tendon. In the horizontal plane, it is located at the same level as the posterolateral portal.

posterolateral portal (Fig. 18.6). Care should be taken, since the neurovascular bundle and its branches are at risk.^{13,14} After the skin incision has been made, a mosquito clamp is introduced and directed toward the arthroscope shaft, which already was introduced through the posterolateral portal. When the mosquito clamp touches the shaft of the arthroscope, the shaft is used as a guide to travel anterior in the direction of the ankle joint. All the way, the mosquito clamp must touch the shaft of the arthroscope until the mosquito clamp touches the bone. The arthroscope is pulled slightly backward and slides over the tip of the mosquito clamp until the tip of the mosquito clamp comes to view. The clamp is used to spread the extra-articular soft tissue in front of the tip of the camera and, when present, scar tissue or adhesions; the mosquito clamp is then exchanged for a 5-mm, full-radius shaver.

Transtibial and Transmalleolar Portals

A transmalleolar portal may be used for debridement and drilling of lesions of the talar dome and is used most often in combination with distraction of the ankle. A special guide facilitates

the placement of the portal and the Kirschner wires that are used to drill the defect. Transtibial or transmalleolar drilling with a guiding system is especially useful for tibial plafond lesions. For the treatment of talar dome lesions, the transmalleolar portal has the disadvantage of causing damage to the cartilage of the medial malleolus opposite the osteochondral talar defect and therefore is not recommended to perform on a routine basis.

SURGICAL TECHNIQUE—ARTHROSCOPIC ANATOMY

The ankle joint can be divided into anterior and posterior cavities, each of which can then be subdivided into three compartments for methodologic inspection of the ankle joint. Ferkel¹⁴ developed a 21-point systematic examination of the anterior, central, and posterior ankle joint to increase the accuracy and reproducibility of the arthroscopic examination (Box 18.1). For posterior ankle problems, Van Dijk et al.¹² reported on a two-portal approach with the patient in the prone position, specifically for close visualization of the posterior compartment of the ankle and subtalar joint. He developed a 14-point systematic examination for the hindfoot and posterior ankle joint (Box 18.2).

The Anterior Ankle

The anterior arthroscopic examination is initially performed through the anteromedial portal, followed by evaluation through the anterolateral portal. From medial to lateral, several structures that can be visualized. First examined is the deep portion of the deltoid ligament as it arises from the tip of the medial malleolus. Its fibers run vertically down to the medial trochlear surface of the talus, an area where ossicles may be hidden and that should be evaluated carefully for pathology. Next, the articular surface of the tip of the medial malleolus as it corresponds and articulates with the medial talar dome, the posterior recess and posterior ligaments can be evaluated. The medial gutter includes the area from the deltoid ligament to below the medial dome of the talus. Areas of articular damage here should be carefully noted.

BOX 18.1 The 21-Point Arthroscopic Examination of the Ankle

Anterior:

Deltoid ligament
Medial gutter
Medial talus
Central talus and overhang
Lateral talus
Trifurcation of the talus, tibia, and fibula
Lateral gutter
Anterior gutter

Central:

Medial tibia and talus
Central tibia and talus
Lateral tibiofibular or talofibular articulation
Posterior inferior tibiofibular ligament
Transverse ligament
Reflection of the flexor hallucis longus

Posterior:

Posteromedial gutter
Posteromedial talus
Postero-central talus
Posterolateral talus
Posterior talofibular articulation
Posterolateral gutter
Posterior gutter

From Ferkel RD. *Arthroscopic surgery. The foot and ankle*. Philadelphia, PA: Lippincott-Raven; 1996.

BOX 18.2 The 14-Point Hindfoot Endoscopic Examination

1. Lateral talocalcaneal articulation
2. Flexor hallucis longus retinaculum
3. Flexor hallucis longus tendon
4. Posterior talar process
5. Posterior talofibular ligament
6. Posterior tibiofibular ligament
7. Transverse tibiofibular ligament
8. Tip of the medial malleolus/medial malleolus
9. Posteromedial gutter
10. Posteromedial talus/tibia
11. Postero-central talus/tibia
12. Posterolateral talus/tibia
13. Posterolateral gutter
14. Tip of lateral malleolus

Additional (when indicated): Posterior tibial tendon, Flexor digitorum tendon, Peroneal tendons.

From Van Dijk CN, Scholten PE, Krips R. *Arthroscopy*. 2000;16:871-876.

The tibia articulates with the medial dome of the talus, forming the medial corner of the ankle. In this region, the anterior articular margin of the tibia deviates from its more horizontal configuration centrally and laterally to a more convex configuration in the coronal plane. At this medial articular notch, the arthroscope may be maneuvered most easily into the central and posterior aspects of the joint without damaging the articular surfaces.

The distal portion of the tibial lip directs slightly anteriorly in the sagittal plane. This portion of the tibia articulates within a depression in the talar surface and is called the sagittal groove. The groove lies between the medial and lateral shoulders of the talus and projects from anterior to posterior. At the area between the anterior tibial lip and the capsule is a periosteum-covered subchondral bone, the synovial recess, which extends from medial all the way to the lateral portion of the ankle. This is where tibial osteophytes develop and synovium and capsule become adherent at the margins of the osteophyte. More laterally, the trifurcation includes the distal lateral tibial plafond, the lateral dome, and the fibula and is bounded by the anterior inferior tibiofibular ligament superiorly. This relation is important in the ankle because it is often the site of soft-tissue pathology. The syndesmotic or anterior inferior tibiofibular ligament runs at approximately a 45-degree angle from the lateral portion of the distal tibia to the fibula, just below the level of the lateral talus. The anterolateral talar dome also is the site of osteochondral lesions of the talus, and access into ankle joint usually is easy in this region. The lateral gutter is the space between the medial border of the fibular articulation and the lateral border of the talar articulation. It extends from below the anterior inferior tibiofibular ligament to the anterior talofibular ligament. This often is the site of chondromalacia and ossicles at the tip of the fibula within the ligament substance. The anterior talofibular ligament lies intracapsular and runs from the tip of the fibula to the inferior lateral portion of the talus. It can be easily reached for a shrinkage procedure in case of laxity. The anterior gutter represents the capsular reflection anteriorly of the ankle as it inserts along the talar neck. There is a normal bare area proximal to the capsular insertion, similar to the area on the central portion of the distal tibia. A synovial recess also can be found at the anterior inferior aspect of the talar dome. In this area, anterior talar osteophytes may articulate or butt against osteophytes of the anterior tibial lip.⁹

The Posterior Ankle

Using a posterolateral and posteromedial portal with the patient in the prone position, one first approaches the fatty tissue overlying the joint capsule (Fig. 18.7). This tissue can be partially removed. At the level of the ankle joint, the posterior tibiofibular ligaments and the posterior talofibular ligament can be recognized. After removal of the very thin joint capsule of the subtalar joint, the posterior compartment of the subtalar joint can be visualized. The posterior talar process can be freed of scar tissue and the FHL tendon can be identified. The FHL tendon is an important landmark to prevent damage to the more medially located neurovascular bundle (Fig. 18.8). When manual distraction is applied to the os calcaneus, the posterior compartment of the ankle joint opens up and can be visualized. The arthroscope and instruments can be introduced into the posterior ankle compartment. Procedures such as a synovectomy and/or capsulectomy of both ankle and subtalar joint can be performed. On the medial side, the tip of the medial malleolus, as well as the deep portion of the deltoid ligament, can be visualized. Opening the joint capsule from inside out at the level of the medial malleolus permits the tendon sheath of the posterior tibial tendon to be opened and the

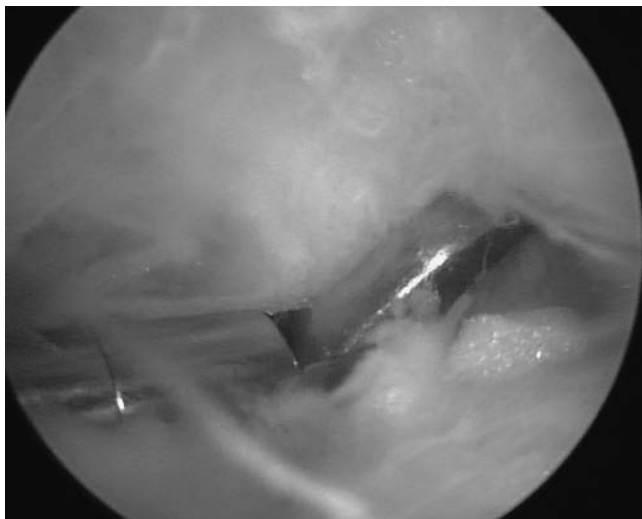


Fig. 18.7 Right ankle. Using a posterolateral and posteromedial portal with the patient in the prone position, one first approaches the fatty tissue overlying the joint capsule. This tissue can be partially removed.

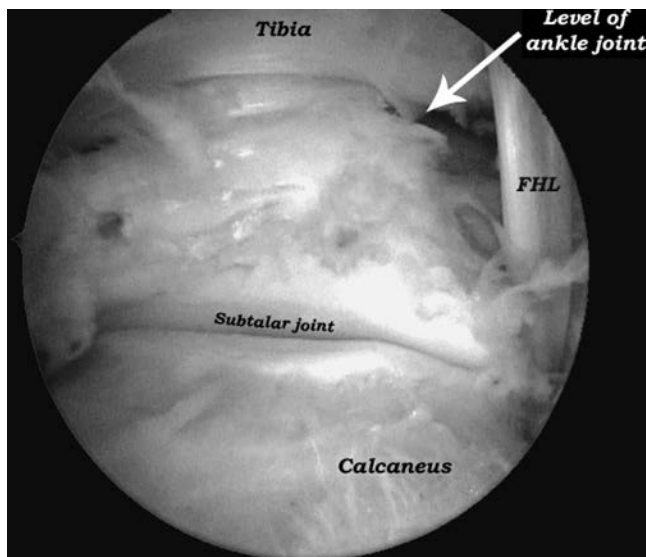


Fig. 18.8 Left ankle. After removal of the thin joint capsule, the posterior ankle and subtalar joint can be visualized. The posterior talar process can be freed of scar tissue and the flexor hallucis longus tendon can be identified. This is an important landmark to prevent damage to the more medially located neurovascular bundle.

arthroscope to be introduced into the tendon sheath to inspect the posterior tibial tendon (Fig. 18.9). The same procedure can be followed for the flexor digitorum longus (FDL). Next, the talar dome and nearly the entire surface of the complete tibial plafond can be inspected. An osteochondral lesion or subchondral cystic lesion can be identified, debrided, and drilled. The posterior syndesmotomic ligaments can be inspected and, if hypertrophic, partially resected. Moreover, the intrinsic syndesmotomic area and the posterior talofibular ligament can be examined (Figs. 18.10 and 18.11). Removal of a symptomatic os trigonum or a nonunion of fracture of the posterior talar process involves partial detachment of the posterior talofibular ligament and release of the flexor retinaculum, both of which attach to the posterior talar prominence. Release of the FHL tendon involves detachment of the

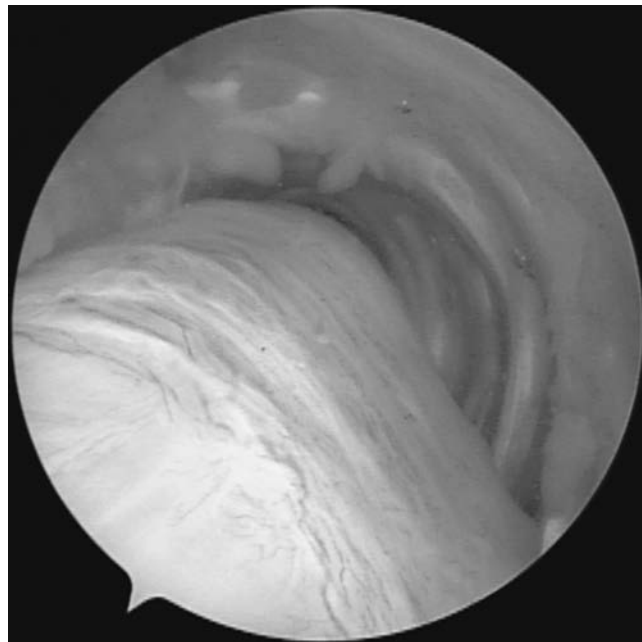


Fig. 18.9 Right ankle. By opening the joint capsule from inside out at the level of the medial malleolus, the tendon sheath of the posterior tibial tendon can be opened and the scope can be introduced into the tendon sheath of the posterior tibial tendon. This patient has a tendinitis of the posterior tibial tendon, recognized by the increased vascularity on and around the tendon. Higher up, a vincula is identified. The direction of the view is from distal to proximal.

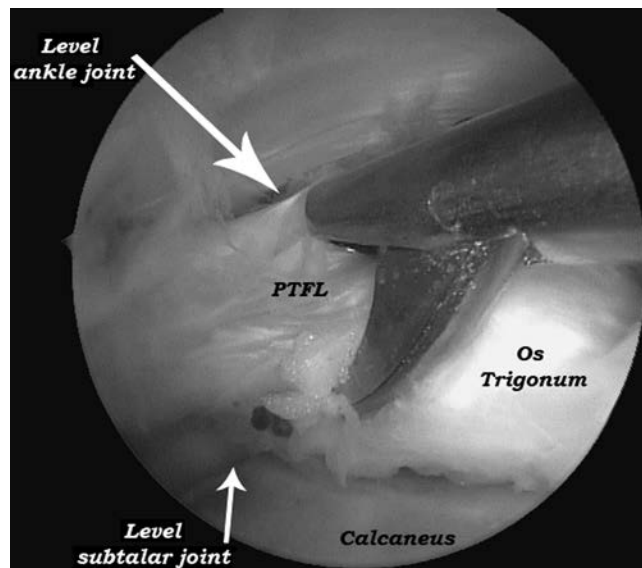


Fig. 18.10 Left ankle. Removal of a symptomatic os trigonum or a nonunion of fracture of the posterior talar process involves partial detachment of the posterior talofibular ligament and release of the flexor retinaculum, both of which attach to the posterior talar prominence.

flexor retinaculum from the posterior talar process (Fig. 18.12). Adhesions surrounding the flexor tendon can be removed. On the lateral side, the peroneal tendons can be inspected (Fig. 18.13). A tight and thickened crural fascia can hinder the free movement of instruments; it can be helpful to enlarge the defect in the fascia by means of a punch or shaver. Bleeding is controlled by electrocautery at the end of the procedure.

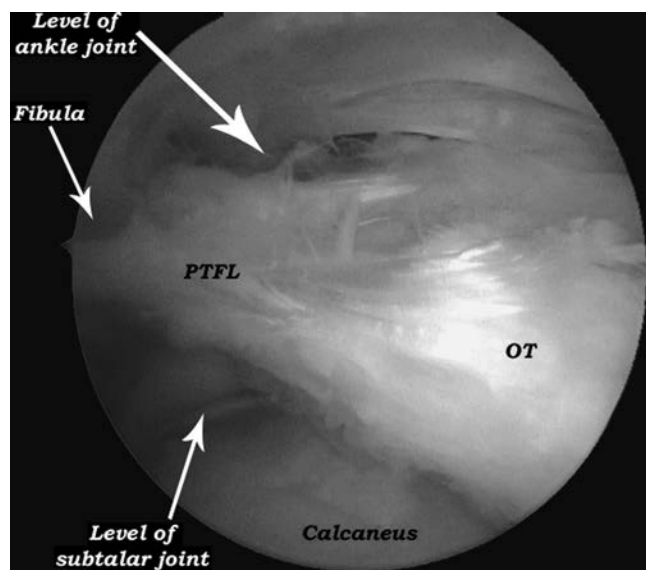


Fig. 18.11 Left ankle. Removal of a symptomatic os trigonum involves partial detachment of the posterior talofibular ligament (PTFL) and release of the flexor retinaculum, both of which attach to the posterior talar prominence (see also Fig. 18.12).

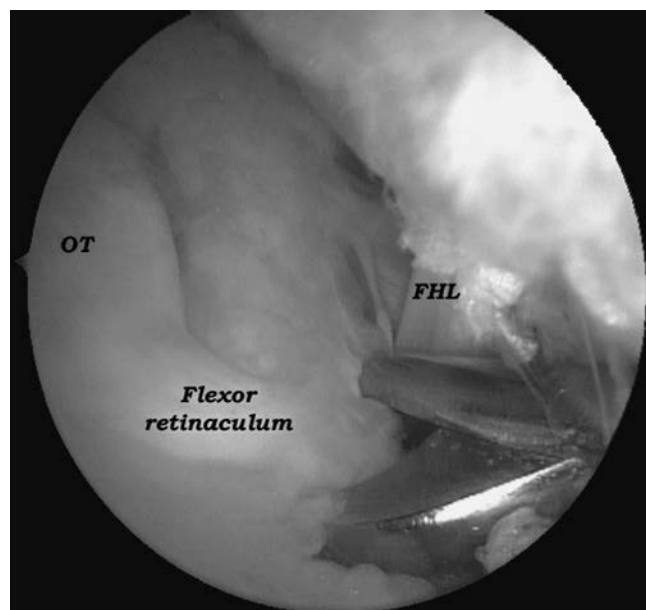


Fig. 18.12 Left ankle. Release of the flexor hallucis longus retinaculum from the posterior talar process to remove a symptomatic os trigonum (see also Figs. 18.11 and 18.13).

DIAGNOSTICS, CONSIDERATIONS, AND SURGICAL TECHNIQUES PER SPECIFIC INDICATION—ANTERIOR ANKLE

Anterior Ankle Impingement

Arthroscopic scoring systems for anterior impingement use the location (tibia or talus) and size of osteophytes as prognostic factors for postoperative success. A study by Scranton et al.¹⁵ compared open resection with arthroscopic resection of painful anterior impingement spurs, ranging from grade I through IV according to the size of spurs and degree of involvement of

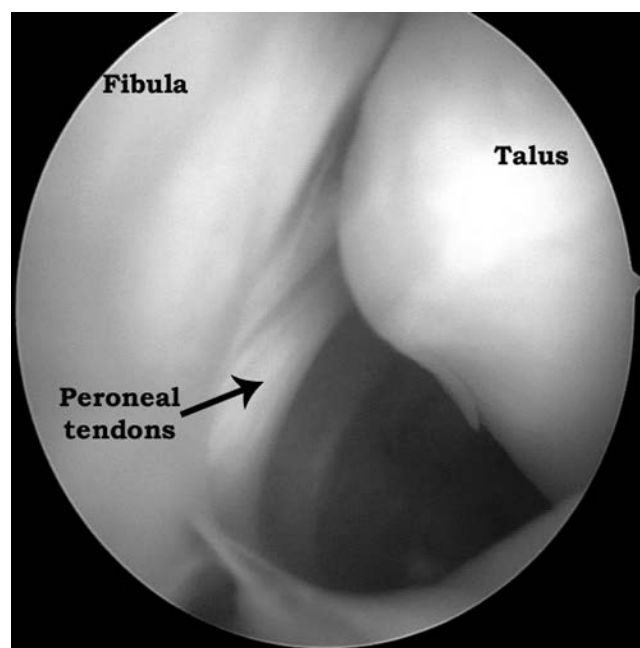


Fig. 18.13 Left ankle. During posterior ankle arthroscopy, the peroneal tendons can be inspected on the lateral side

BOX 18.3 Classification System for Degenerative Changes of the Ankle

Grade	X-ray Finding
1	Normal joint or subchondral sclerosis
2	Osteophytes without joint space narrowing
3	Joint space narrowing with or without osteophytes
4	(Sub)total disappearance or deformation of the joint space

From Van Dijk CN, Tol JL, Verheyen CC. *Am J Sports Med.* 1997;25:737-745.

the ankle. They demonstrated that the treatment and recovery correlated with the grade, with all grades being able to be resected arthroscopically. The reproducibility of this classification system may be doubtful, however, because the correlation was assessed with outcomes at short-term follow-up (10 weeks postoperatively).

Other studies determined that the degree of osteoarthritic changes also influences the outcome of treatment.⁹ Osteophytes without joint space narrowing are not a manifestation of osteoarthritis; subsequently a normal joint remains after removal of these spurs. A classification for anterior ankle impingement based on the degree of degeneration was developed (Box 18.3).⁹ The results at long-term follow-up show that the use of this osteoarthritic classification is more discriminating than the impingement classification of Scranton and McDermott as a predicting value for the outcome of arthroscopic surgery for anterior ankle impingement.^{9,15}

Surgical Technique

The patient is placed in a supine position with slight elevation of the ipsilateral buttock. The heel of the affected ankle rests on the very end of the operating table, thus making it possible



Fig. 18.14 The heel of the affected ankle rests on the end of the operating table, thus making it possible for the surgeon to dorsiflex the ankle joint fully by leaning against the sole of the patient's foot.

for the surgeon to fully dorsiflex the ankle joint by leaning against the sole of the patient's foot (Fig. 18.14). After making an anteromedial skin incision, the surgeon bluntly divides the subcutaneous layer with a hemostat. A 4-mm, 30-degree arthroscope routinely is used. The anterolateral portal is made under arthroscopic control. Additional portals just anterior to the tip of the lateral or medial malleolus are used only when indicated. Osteophytes are removed by a 4-mm chisel and burr. These spurs can be identified easily when the ankle is in a fully dorsiflexed position to prevent the anterior joint capsule from covering the osteophytes. Another advantage of the forced dorsiflexion position is the fact that the talus is concealed in the joint, thereby protecting the weight-bearing cartilage of the talus from potential iatrogenic damage. The contour of the anterior tibia is first identified by shaving away the tissue just superior to the osteophyte. An overcorrection of the medial malleolus generally is pursued by shaving some of it away after resection of the osteophyte.

Visualization of the anterior ankle joint can be improved by bringing the ankle into a forced dorsiflexion position because in this position the anterior working area opens up. Distraction makes the anterior capsule more tense over the osteophyte, and its use therefore is not recommended.⁹ It is important to identify the anterior and superior borders of the osteophyte, and this often requires careful elevation or peeling of soft tissues from the confines of the osteophyte.

After an arthroscopic intervention, a hematoma will be formed postoperatively that subsequently will develop into scar tissue. The scar tissue that fills the defect will act instantly as a new anterior soft-tissue impediment. It therefore is important to remove the osteophytes to enhance more anterior and anteromedial space and diminish the chance for a recurrence of symptoms.¹⁶⁻¹⁸

Synovitis

Synovitis can be a noninflammatory, inflammatory, or septic process of the synovium, which is most characterized by joint swelling and tenderness. A generalized or localized synovitis can occur, most often with fibrous bands and adhesions. Synovitis of the ankle may be a difficult diagnostic problem. Even after careful history, physical examination, and diagnostic testing, the diagnosis may not be readily apparent. During arthroscopy, localized or generalized inflammation of the synovia can be present. It may contain hemosiderin or fibrin debris. Scarring, fibrosis, and adhesions often are seen in relation to the synovitis.

In 1997, Cheng and Ferkel¹⁹ proposed the following classification system for synovial disorders:

- Congenital: plicae or congenital bands within the ankle; plicae, or shelves, have been demonstrated in the knee but are difficult to find in the ankle. Congenital bands are seen as an incidental finding when examining the ankle for other types of pathology.
- Traumatic: sprains, fractures, and previous surgery
- Rheumatic: rheumatoid arthritis, pigmented villonodular synovitis, crystal synovitis, hemophilia, and synovial chondromatosis
- Infectious: bacterial and fungal
- Degenerative: primary and secondary
- Neuropathic: Charcot joint
- Miscellaneous: ganglions, arthrofibrosis

Osteochondral Lesions (see also Chapter 16)

An important cause of residual pain after an ankle sprain is an osteochondral lesion of the talus. It is defined as the separation of a fragment of articular cartilage, with or without subchondral bone. The incidence of an osteochondral lesion after an ankle sprain probably is underestimated because these lesions often remain undetected and has been reported to be as high as 6.5% after ankle sprains. In the acute situation, symptoms depend on the amount of damage to the periarticular tissues and the involvement of afferent pain fibers in the subchondral bone. Usually the lesion is located in the anterolateral or posteromedial aspect of the talar dome. Histologically the medial and lateral lesions are identical, but morphologically they differ; the lateral lesions are shallow and more wafer shaped, indicating a shear mechanism of injury. In contrast, medial lesions generally are deep, cup shaped, and located posteriorly, indicating a mechanism of torsional impact. From an etiologic point of view, trauma is the most common cause of osteochondral lesions of the talus, but idiopathic osteonecrosis often may be the underlying pathologic process. In the literature, the latter has been associated with alcohol abuse, use of steroids, endocrine disorders, and some hereditary conditions. Although initial



Fig. 18.15 A heel-rise view (*left*) demonstrates a posteromedially located osteochondral defect. Because of the relative posterior location of the defect, a plain anterior-posterior view (*right*) is not able to demonstrate this lesion.

symptoms may be absent, in chronic cases most patients present with intermittent pain located deep in the ankle joint that increases on weight bearing. On physical examination, signs are often absent. A discrete limitation of range of motion with some synovitis may be present. Local tenderness on palpation with recognition is absent in most cases. Since there are no specific pathognomonic signs or symptoms, it is essential for the examining physician to be aware that an osteochondral lesion can be present. The frequent absence of radiographic changes on conventional radiographs has led to the use of more sensitive methods for detection. A heel-rise view doubles the change to detect an osteochondral lesion (Fig. 18.15).²⁰ Furthermore, both CT scan and MRI can be helpful for the diagnosis and preoperative planning with a lack of significant difference between the sensitivity and specificity of both modalities.²⁰

Pritsch et al. developed an arthroscopic staging system that correlated well with the CT classification of Ferkel and Sgaglione²¹⁻²³ and the MRI classification of Anderson et al.²¹⁻²⁶ In 1999, a new arthroscopic staging system was developed by Taranow et al.,²⁷ who classified cartilage as viable and intact (stage A) or breached and nonviable (stage B). The bone component was determined as follows: (1) stage 1 is a subchondral compression or bone bruise, (2) stage 2 lesions are subchondral cysts and are not seen acutely (these develop from stage 1 lesions), (3) stage 3 lesions are partially separated or detached fragments in situ, and (4) stage 4 represents displaced fragments. The condition of the cartilage and bone together determines the type of surgical treatment.

Despite the existence of classification systems, few authors base their treatment decision on these systems. A meta-analysis of Tol et al.²⁸ showed that the value of preoperative radiologic staging systems was of minor value in the preoperative planning because they hardly correlate with the perioperative findings. This demonstrates the shortcoming of preoperative radiologic staging systems as a guide for the treatment strategy. Perioperative staging of osteochondral lesions therefore seems more appropriate. Eventually, the most rational way of preoperative assessment of

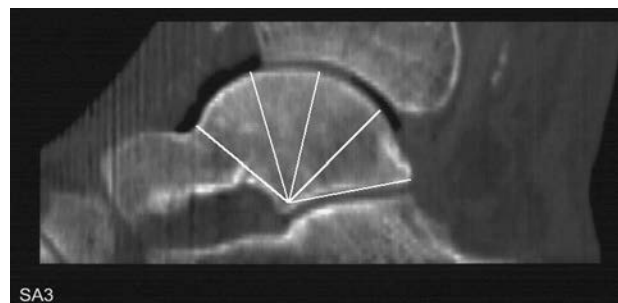


Fig. 18.16 From anterior to posterior the talar dome can be divided into four equal parts. When an osteochondral defect is located in one of the three anterior parts, it can be reached by a routine anterior ankle arthroscopy. Soft-tissue distraction might be necessary. When the lesion is located in the most posterior quarter, it can be reached only by posterior ankle arthroscopy.

osteochondral lesions is to determine whether they are symptomatic or asymptomatic.

Surgical Technique

From anterior to posterior the talar dome can be divided into four equal parts (Fig. 18.16). When the osteochondral lesion is located in one of the three anterior parts of the talar dome, it can be treated by a routine anterior ankle arthroscopy. When it is located in the most posterior quarter of the talar dome, the defect should be approached by a posterior ankle arthroscopy (see “Osteochondral lesions” under 18.6) or by means of a medial malleolar osteotomy. The current routine treatment consists of removal of dead bone and overlying cartilage.²⁸ After debridement, the subchondral sclerotic zone is perforated with a burr or K-wire or by microfracture. Preoperatively, it is desirable to decide whether to use mechanical distraction in combination with a 2.7-mm arthroscope or to use a standard 4-mm arthroscope and to treat the osteochondral lesion in the anterior working area by forcing the ankle into full plantarflexion. The osteochondral lesion in the posterior quarter of the talar dome is difficult to reach in the hyperplantarflexed position in

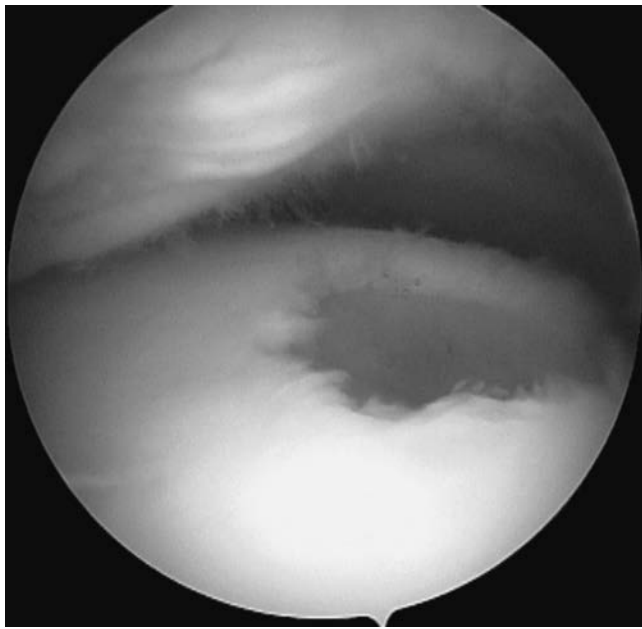


Fig. 18.17 An anterolateral located osteochondral defect of the talus. By bringing the ankle in full plantarflexion, the defect can be fully debrided. Completeness of the debridement can be checked by switching portals.

patients having a diminished plantarflexion or in case of anterior osteophytes.^{27,29}

Routinely the procedure is performed without distraction. The standard anteromedial and anterolateral approaches are created as described earlier. In an osteochondral lesion located medially, the 4-mm arthroscope is moved over to the anterolateral portal and the instruments are introduced through the anteromedial portal. For an anterolateral defect, the arthroscope remains in the anteromedial portal and the instruments are introduced through the anterolateral portal. If osteophytes are present, they are removed first by chisel and/or burr. Synovitis located anterolaterally (in case of an anterolateral defect) or anteromedially (in case of an anteromedial defect) is removed by a 4.5- or 5.5-mm synovator. The extent of removal of osteophytes and synovitis is checked by bringing the ankle into plantarflexion. It now should be possible to palpate and visualize the osteochondral lesions (Fig. 18.17). If this is not the case, then a further synovectomy should be performed in the dorsiflexed position. After sufficient synovectomy, it is possible to identify the lesion in the forced plantarflexed position by palpating the cartilage with a probe. Not only can the lesion be palpated with a probe, but it also should be possible to visualize at least the most anterior part of the lesion. It can be helpful to add a soft-tissue distraction (Fig. 18.18, A and B).¹⁸ If possible, a 3.5- or 4.5-mm synovator is now introduced into the lesion. After it has been debrided by the synovator or curette, the arthroscope is moved over to the portal opposite the defect to check the completeness of the debridement. The scope then is brought back to the opposite portal and further debridement is performed. It is important to remove all dead bone and overlying, unsupported, unstable cartilage. Every step in the debridement procedure should be checked by regularly switching portals in order to

perform a precise and complete debridement, with removal of all loose fragments. Introduction of the instruments and the arthroscope is performed with the ankle in the fully dorsiflexed position, thus preventing iatrogenic cartilage damage. After full debridement, the sclerotic zone is approached with a microfracturing technique, or multiple drill holes are made with a 2-mm burr or a 1.6-mm K-wire. A K-wire has the advantage of flexibility, whereas a 2-mm drill can break more easily if the position of the ankle is changed during drilling. When a 2-mm drill is used, a drill sleeve is necessary to protect the tissue.

In posteriorly located lesions for which an anterior approach is chosen, a noninvasive traction device that allows the surgeon to change quickly from the fully dorsiflexed position (introduction of the instruments) to the distraction position offers obvious advantages. The distraction device consists of a belt around the waist of the surgeon that is connected to a noninvasive distraction loop placed around the ankle. The amount of distraction can be adjusted by leaning more or less backward (see Fig. 18.18, B).

Loose Bodies and Ossicles

Loose bodies can be either bony, chondral, or osteochondral and can arise from osteophytes or defects in the talus or tibia. Sometimes, a loose body is attached to the capsule or other structures with scar tissue, better known as a corpus liberum pendulans.¹⁴ A small, loose body may cause catching symptoms along with pain, swelling, and limitation of motion. Symptoms of internal derangement may resolve if a small loose body becomes fixed to the synovial lining, ceasing to cause joint irritation. A loose body may grow by proliferation of chondroblasts/osteoblasts or may shrink because of the action of chondroblasts/osteoclasts.

Physical examination may not be very revealing, with vague areas of tenderness, possible limitation of motion, and catching. Rarely is a loose body palpable. As with all ankle problems, a careful physical examination must rule out extra-articular entities that can cause symptoms similar to intra-articular lesions. Peroneal subluxation, posterior tibial tendon attrition or rupture, tarsal tunnel syndrome, sinus tarsi syndrome, stress fracture, and tendinitis must be carefully excluded by both physical examination and ancillary studies. Plain radiographs usually reveal an osseous loose body, but chondral loose bodies are not visible on routine studies. A CT or MRI study is best suited to make the distinction between an intra-articular versus an extra-articular or intracapsular abnormality.

Surgical Technique

The arthroscopic approach to loose bodies is straightforward. Loose bodies localized to the anterior compartment, particularly in patients with ligamentous laxity, can be approached with a routine setup using anteromedial and anterolateral portals. However, the posterior joint also should be examined for the presence of loose bodies, which can hide in the posterior recess of the joint.^{12,29} A posterolateral portal can be made for inspection and posteriorly located loose bodies can be removed best by means of a two-portal posterior approach.

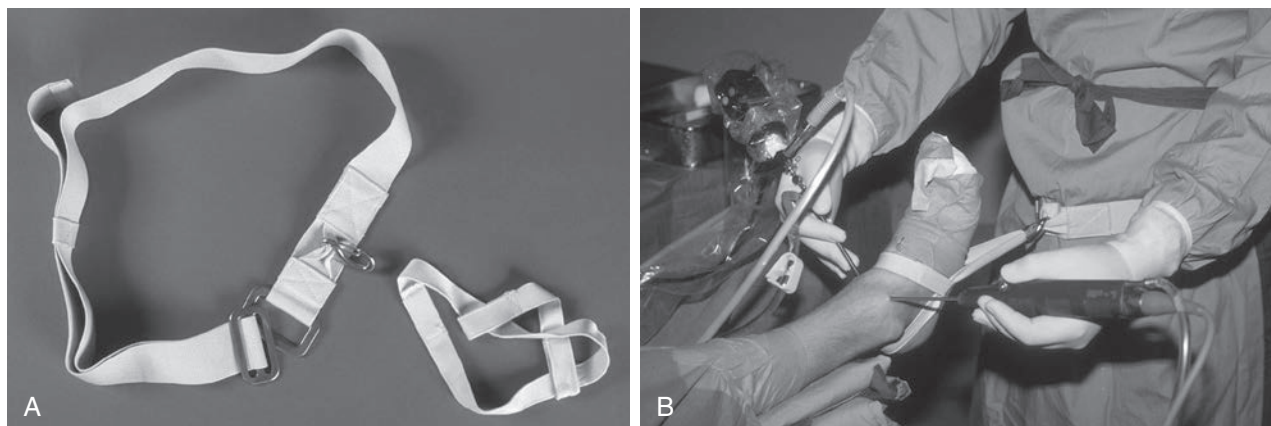


Fig. 18.18 (A) A resterilizable soft-tissue distractor can be helpful to visualize lesions that are located more posteriorly in the ankle joint. (B) The amount of soft-tissue distraction can be adjusted by leaning more or less backward.

SURGICAL TECHNIQUES AND CONSIDERATIONS PER SPECIFIC INDICATION—POSTERIOR ANKLE

Posterior ankle syndrome involves pain syndrome. It presents mainly on forced plantarflexion. Posterior ankle impingement is caused by overuse or trauma, mainly found in ballet dancers and runners.¹² Distinction between these two disorders seems important because posterior impingement through overuse has a better prognosis.

There are at least 10 specific causes for posterior ankle pain: os trigonum syndrome; posttraumatic calcifications, loose bodies, and bony avulsions; osteochondral defects; tendinitis of the FHL tendon; tendinitis of the posterior tibial tendon; tendinitis the peroneal tendons; tendinitis of the Achilles tendon; and ankle and subtalar arthrosis.^{12,29}

Os Trigonum Syndrome

The os trigonum is an inconsistently present accessory bone of the foot situated at the posterolateral aspect of the talus. It appears between the ages of 8 and 11 as a secondary center of ossification and usually fuses to the talus within 1 year after its appearance. When this ossification center remains separate from the talus it is referred to as the os trigonum. According to Sarrafian, the prevalence of this ossicle ranges from 1.7% to 7.7%.³⁰ A recent study by Zwiers et al. found a prevalence of 23.7% on CT-scans of non-affected ankles.³¹ When fusion does occur and there is a large, intact posterolateral process, it is referred to as a fused os trigonum. Since Rosenmuller first described the os trigonum in 1804, there has been controversy concerning its origin.³² McDougall³³ believed it to be a secondary ossification center of the talus, whereas other authors state that it is a non-united fracture of the posterolateral talar process. The os trigonum usually remains asymptomatic, but an otherwise normal os trigonum can become symptomatic during or after strenuous physical activities or following an injury to the ankle. Sometimes an acute trauma in plantarflexion may result in contusion, compression, or fracture of the os trigonum or posterior process of the talus. These injuries may

cause an overload posttraumatic syndrome of the os trigonum. In this condition, the os trigonum becomes painful but appears undisrupted on the lateral x-rays (Fig. 18.19). On the other hand, chronic impingement of the posterior process of the talus against the tibia caused by chronic microtrauma or overuse by repeated hyperplantarflexion movements can lead to an inflammatory process of the os trigonum. It also can result in degenerative changes in the posterior capsule of the ankle joint, adjacent ligaments, tendon, and chondrosynovial surface.

Clinically, the patient complains of pain during push-off while running. The pain often is absent during walking on level ground but appears on uneven terrain. Usually pain is complained of posterolaterally in the ankle joint, but sometimes it may be located in the posteromedial region. Physical examination can reveal the presence of moderate swelling on only the medial or on both sides of the Achilles tendon, with tenderness on palpation.

A forced passive plantarflexion of ankle and foot will reproduce the recognizable symptoms. With this test the examiner performs repetitive, quick, passive forced plantarhyperflexion movements. The test can be repeated in slight external rotation or internal rotation of the foot relative to the tibia. The investigator should apply this rotation movement on the point of maximal plantarflexion, thereby grinding the posterior talar process/os trigonum in between the posterior tibial rim and calcaneus (Fig. 18.20). A negative test rules out a posterior impingement syndrome. A positive test in combination with pain on posterolateral palpation can be followed by a diagnostic infiltration. The infiltration is performed from the posterolateral position between the prominent posterior talar process and the posterior edge of the tibia. If the pain disappears on forced plantarflexion, the diagnosis is confirmed. After clinical examination, a routine lateral radiograph of the ankle should reveal an os trigonum. Bone scanning can effectively localize osseous injuries in and around the talus by demonstrating increased uptake in the posterior talar region but is not very specific. A CT scan enables the surgeon to determine the exact location, size, and shape of the ossicle and therefore is valuable for preoperative planning (Fig. 18.21, A and B).

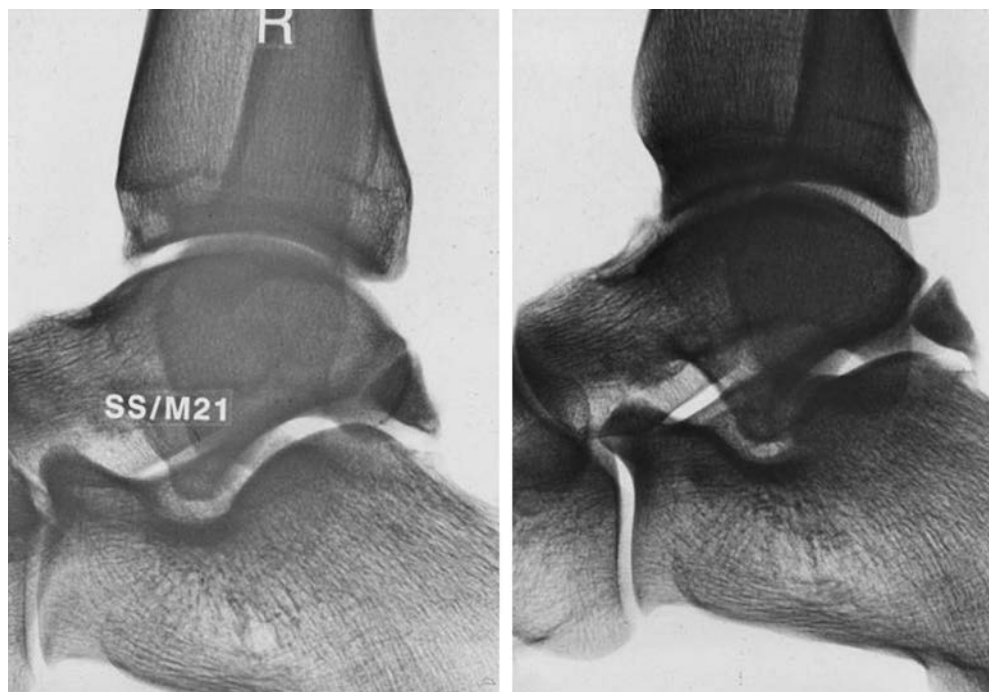


Fig. 18.19 Posttraumatic syndrome of the os trigonum of the right ankle. Plain lateral x-rays (*left*) reveal an undisrupted os trigonum. Additional posteromedial impingement views (PIM) with the foot in 25-degrees external rotation in the same patient show that the os trigonum is disrupted.

FHL tendinitis is often present in patients with a symptomatic os trigonum and pain located posteromedially. The FHL tendon can be palpated behind the medial malleolus. By asking the patient to flex the toes repetitively with the ankle in 10- to 20-degree plantarflexion, the FHL tendon can be palpated in its gliding channel behind the medial malleolus. During palpation there may be crepitus and recognizable pain.

Surgical Technique

A two-portal (posterolateral and posteromedial) approach with the patient in the prone position gives excellent access to the posterior ankle compartment of the ankle joint. The posterior compartment of the ankle joint thus can be visualized, and the subtalar joint, os trigonum, and FHL can be inspected. After inspection, the posterior talofibular ligament must be detached from the posterior talar process. The superior border of the posterior talar process is cleaned with the shaver, after which the FHL tendon can be inspected (Fig. 18.22). The flexor retinaculum can be cut. After this has been performed, the posterior talocalcaneal ligament must be cut. Finally, the os trigonum can be detached with a chisel or small osteotome and subsequently removed (Fig. 18.23).

Posttraumatic Calcifications, Loose Bodies, and Bony Avulsions

Calcifications, bony avulsions, and loose chondral- or osteochondral fragments may result from major trauma to the ankle joint.¹⁴ When the fragments are located in the posterior compartment of the ankle, they are most likely the result of a hyperplantarflexion trauma or a combination of strong inversion, plantarflexion, and external rotation of the tibia. In either case,

an unsuspected chondral or osteochondral lesion may occur and result in a loose body floating in the posterior compartment of the ankle or subtalar joint.

Osteophytes of the posterior tibial rim, an os trigonum, and even part of the posterior talar process may break off during a hyperplantarflexion trauma and act as a loose body. After a severe inversion trauma, the posterior talofibular ligament may avulse a bony fragment from the posterior talar process, possibly causing posterior ankle impingement. Multiple loose cartilaginous or osteocartilaginous bodies also may form in synovial chondromatosis.

A small, loose body may cause catching symptoms with joint motion along with pain. Plantarflexion may be limited and painful during the hyperplantarflexion test. Plain lateral radiographs usually reveal an osseous loose body, but when located posteromedially it may over-project. An additional posteromedial impingement view (PIM) with the foot in 25-degree external rotation relative to the tibia is helpful when there is suspicion for bony pathology in posteromedial compartment of the ankle joint (see Fig. 18.19). Lesions that appear to be loose bodies on routine radiographs may actually be intra-articular, intracapsular, or extra-articular in location, particularly in the posterior ankle joint compartment. The location of the lesions should be determined preoperatively to avoid embarrassment of performing an arthroscopic examination for loose body removal only to find the joint free of any abnormality. A CT scan is best suited to make the distinction between an intra-articular abnormality versus an extra-articular or intracapsular abnormality and to determine the exact location in the posterior ankle joint compartment.

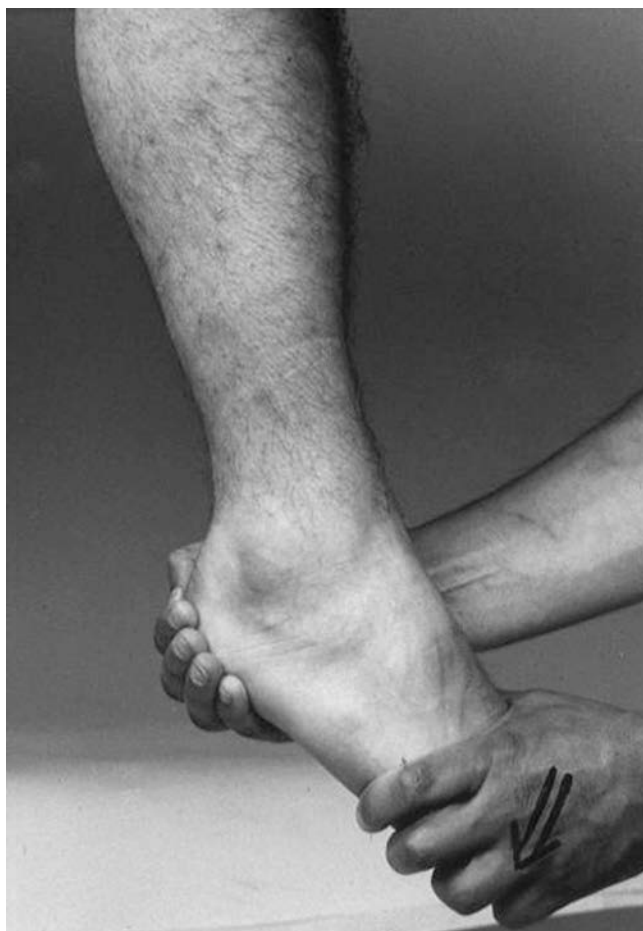


Fig. 18.20 Forced passive plantarflexion test. This test will reproduce the recognizable symptoms. The examiner performs repetitive, quick, forced passive plantarhyperflexion movements. The investigator should apply rotational movements on the point of maximal plantarflexion; thereby, the posterior talar process/os trigonum is pinched between the posterior tibial rim and calcaneus.

Osteochondral Lesions

On the sagittal plane, the talar dome can be divided into four equal quarters. When an osteochondral lesion is located in one of the anterior three quarters of the talar dome, which included the majority of the lesions, the pathology should be approached by routine anterior ankle arthroscopy. In case the lesion is located in the most posterior quarter, however, the lesion is better treated by posterior ankle arthroscopy. A preoperative CT scan with sagittal image reconstructions is important to determine the exact location of the lesion (Fig. 18.24). In case of a posteromedially located osteochondral defect, the FHL tendon also should be inspected routinely. The tendon can be affected because of shredding of the tendon against the defect during flexion of the great toe while walking. When the tendon is affected, the flexor retinaculum should be cut and thus the tendon released and debrided. Osteochondral lesions of the tibial plafond can best be treated by means of posterior ankle arthroscopy, since the joint opens up quite well with soft tissue distraction. Treatment includes debridement and microfracturing. A 90-degree microfracture probe is very well suited for this purpose. Cystic lesions that are too large to reach by



Fig. 18.21 (A and B) A computed tomography scan enables the surgeon to determine the exact location, size, and shape of loose ossicles and is therefore valuable for preoperative planning. (A) A loose fragment posterolateral in the ankle joint on a sagittal reconstruction. (B) Loose fragments between the distal fibula and talus of the left ankle.

microfracturing are best be approached by retrograde drilling using a retrograde aiming device. Cysts larger than 1-cm diameter demand filling with bone graft.

SURGICAL TECHNIQUES AND CONSIDERATIONS PER SPECIFIC INDICATION—PATHOLOGY OF THE TENDONS AND TALAR JOINT

FHL Tendon Pathology

Tendinitis of the FHL tendon is caused most often by posterior overuse and posttraumatic injuries.³⁴ It is typically found in athletes performing repetitive, forceful push-offs such as gymnasts, skaters, long-distance runners and swimmers,³⁵⁻³⁸ and in

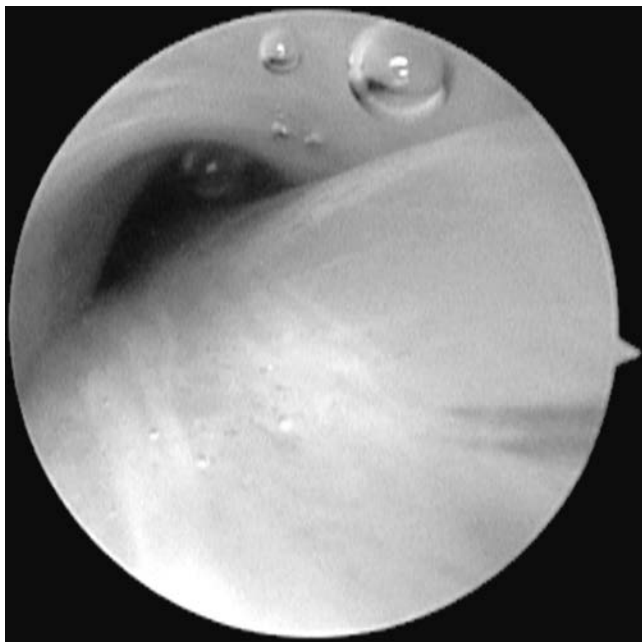


Fig. 18.22 Right ankle. Inspection of the flexor hallucis longus tendon in its channel. The view is from proximal to distal. (See also Figs. 18.6 and 18.12.)

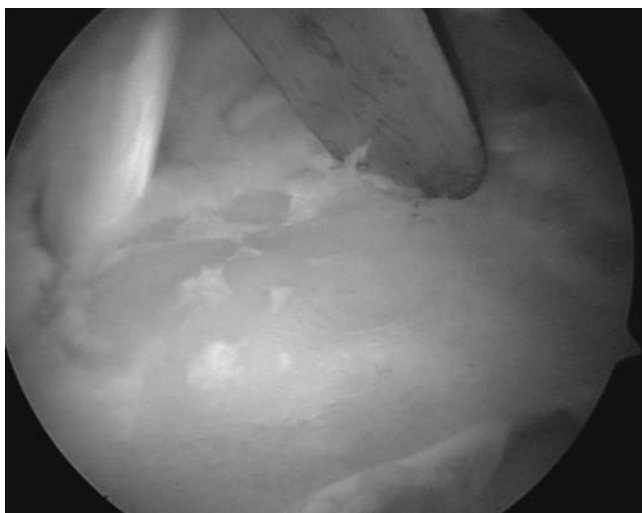


Fig. 18.23 Removal of an os trigonum with a chisel. On the left the flexor hallucis longus tendon is located.

ballet dancers.^{35,36} Moreover, it is a concomitant finding with other pathology, such as an os trigonum, loose bodies, bony avulsions, ankle and subtalar arthrosis, or a combination. An unexpected but consequent finding that may cause FHL tendinitis is a posteriorly located osteochondral lesion, with the defect being located in the posterior quarter of the talar dome on the medial side. In a case series reporting on 50 patients with FHL tendinitis, only three patients had an isolated tendinitis. A concomitant posteromedial osteochondral defect was found in seven patients, and in 40 patients the tendinitis was accompanied by os trigonum syndrome, bony avulsions, calcifications, and localized synovitis.³⁹ The tendinitis is maintained during the stance phase when walking. During this phase, the ankle joint is in dorsiflexion and the posterior talar dome is in closest

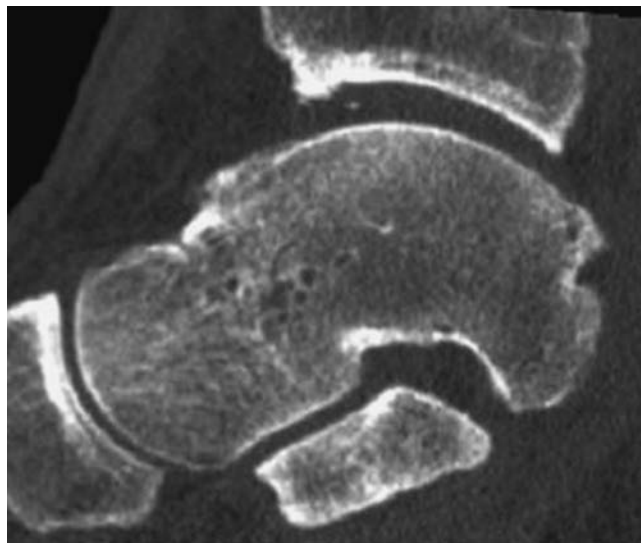


Fig. 18.24 A preoperative computed tomography (CT) scan with sagittal image reconstructions is important to determine the size and location of posterior located osteochondral defects. A sagittal CT reconstruction of a left ankle with an osteochondral defect that is located at the posterior end of the talar dome.



Fig. 18.25 In a cadaver specimen it is shown that the flexor hallucis longus tendon shreds against the posterior part of the talus (often the place where osteochondral defects are located) during dorsiflexion (see text).

contact with the tendon. The tendon, meanwhile, is moving in the opposite direction because the toes are actively flexed to start with the push-off phase. Here, the tendon may shred against the osteochondral lesion, leading to irritation and inflammation (**Fig. 18.25**). In this way a posteriorly located osteochondral lesion can cause tendinitis of the FHL.

The pain of an FHL tendinitis is located posteromedially. The tendon can be palpated behind the medial malleolus. By asking the patient to flex the toes repetitively with the ankle in 10- to 20-degrees plantarflexion, the FHL tendon can be palpated in its gliding channel behind the medial malleolus. The tendon glides up and down under the palpating finger of the examiner.

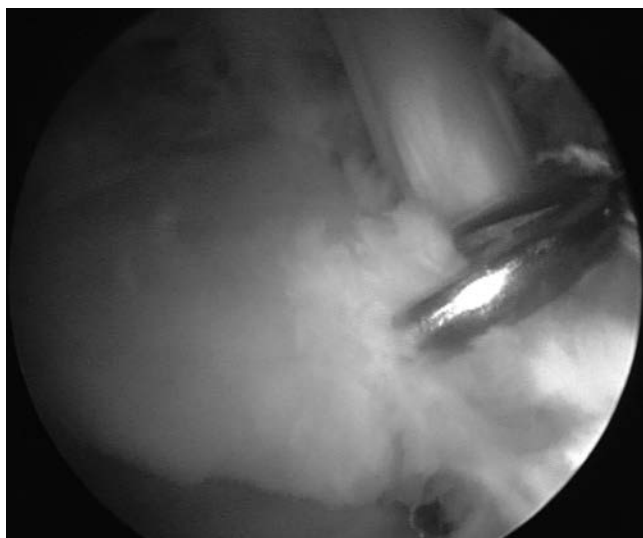


Fig. 18.26 Release of the flexor retinaculum in a left ankle. Adhesions surrounding the flexor hallucis longus tendon are removed.

In case of stenosing tendinitis or chronic inflammation, there may be crepitus and recognizable pain. Sometimes a nodule in the tendon can be felt to move up and down under the palpating finger.³⁹

Surgical Technique

The routine two-portal approach with the patient in the prone position is used when evaluating and treating flexor hallucis longus pathology. In general, during posterior ankle arthroscopy the FHL tendon is an important landmark to prevent damage to the more medially located neurovascular bundle.^{12,29} When a tendinitis is present, it is treated by performing a release of the flexor retinaculum (Fig. 18.26). The tendon sheath can be opened distally all the way up to the level of the sustentaculum tali. Here, the arthroscope may enter the tendon sheath in order to inspect the tendon tissue. Adhesions surrounding the FHL tendon, nodules, and scar tissue can be removed and inflammation, tears, and thickness can be debrided. In case of a FHL tendon tear, treatment includes reduction of any nodule and debridement of the tendon with removal of irregularities by means of radiofrequency. In our experience, converting to an open procedure is not necessary.

Posterior Tibial Tendon Pathology

The posterior tibial tendon plays an important role in normal hindfoot function. It plantarflexes and supinates the foot and thus prevents valgus deformity. Different stages of posterior tibial tendon dysfunction have been described as the disease progresses from peritendinitis to elongation and degeneration of the tendon.³⁴ Tenosynovitis is often seen in association with flatfoot deformity or a prominent navicular tubercle and, to a lesser extent, in association with psoriatic and rheumatoid arthritis. In the early stage of posterior tibial tendinitis, tenosynovectomy can be performed if conservative treatment fails. Postsurgical and post-fracture adhesions and irregularity in the contour of the posterior aspect of the tibia/medial malleolus can account for a symptomatic posterior tibial tendon.



Fig. 18.27 Right ankle. The distal portal for posterior tibial tendoscopy is located directly over the tendon 2 cm distal to the posterior edge of the medial malleolus. A 2.7-mm arthroscope is introduced.

Posttraumatic calcification in the posteromedial joint capsule can produce symptoms of posterior tibial tendinitis because of the close connection of joint capsule and posterior tibial tendon sheath in this region. In a cadaveric study, a consistent membranous mesotendineal structure was found between tendon and tendon sheath.³⁴ This thin, vincula-like structure runs between the posterior tibial tendon and tendon sheath and attaches to the tendon sheath of the flexor digitorum tendon. It runs from the proximal end all the way with a free edge approximately 4 to 5 cm above the level of the posteromedial tip of the malleolus. After traumatic injury to the ankle, these mesotendineal structures may have clinical implications.

Surgical Technique

The main portal for posterior tibial tendoscopy is located directly over the tendon, 2 cm distal to the posterior edge of the medial malleolus. The distal portal is made first, with an incision through the skin. The tendon sheath is penetrated by the arthroscope shaft with a blunt trocar. A 2.7-mm arthroscope with an inclination angle of 30 degrees is introduced (Fig. 18.27). After a spinal needle is introduced under direct vision, an incision is made through the skin into the tendon sheath to create a proximal portal. Instruments such as shaver system can be introduced. Through the distal portal a complete overview can be obtained of the posterior tibial tendon, from its insertion (navicular bone) to approximately 6 cm above the level of the tip of the medial malleolus.

The complete tendon sheath can be inspected by rotating the scope over the tendon. Special attention should be given to inspect the tendon sheath covering the deltoid ligament, the posterior medial malleolus surface, and the posterior joint capsule. More proximal, the free edge of the vincula is inspected. The posterior joint capsule can be palpated and removed with a shaver system. The arthroscope is placed from the distal portal between tendon and medial malleolus. The shaver comes down from the proximal portal. Once the arthrotomy is made, the arthroscope and instruments can be manipulated into the posteromedial compartment of the ankle joint. Synovectomy or



Fig. 18.28 Left ankle. The main portal for peroneal tendoscopy is located directly over the tendons 2 cm distal to the posterior edge of the lateral malleolus. The distal portal is made first. A 2.7-mm scope can be introduced. The proximal portal is created under direct vision.

loose body removal thus can be performed. In case of a posterior tibial tendon tear, after arthroscopic debridement, the procedure must be converted to a mini open procedure for tubularization of the tendon

Peroneal Tendon Pathology

Tenosynovitis of the peroneal tendons, (recurrent) dislocation, rupture, and snapping of one of the peroneal tendons account for most of the symptoms at the posterolateral side of the ankle joint.^{34,39} This disorder must be differentiated from fatigue fractures of the fibula, lesions of the lateral ligament complex, and posterolateral impingement (os trigonum syndrome). Peroneal tendon disorders often are associated and secondary to chronic lateral ankle instability.⁴⁰ Because the peroneal muscles act as lateral ankle stabilizers, more strain is placed on their tendons in the presence of chronic lateral instability, resulting in hypertrophic tendinopathy, tenosynovitis, and ultimately in (partial) tendon tears. The diagnosis may be difficult in patients with lateral ankle pain. Recurrent peroneal tendon dislocation and tenosynovitis can be established by clinical examination. In the case of tears or ruptures of the peroneus brevis or longus tendon, supplemental investigations such as MRI or ultrasonography can be helpful to establish the diagnosis. Postsurgical and post-fracture adhesions and irregularities in the posterior aspect of the fibula where the gliding channel of the tendon is located can be responsible for symptoms in this region.

The primary indication for peroneal tendoscopy is posterolateral ankle pain with a high clinical suspicion for tenosynovitis of the peroneal tendons, subluxation or dislocation, a tear or postoperative adhesion,^{41,42} independently of whether or not positive MRI findings are present.⁴³

Surgical Technique

The main portal for peroneal tendoscopy is located directly over the tendons, 2 cm distal to the posterior edge of the lateral malleolus. The distal portal is made first, with an incision through the skin. The tendon sheath is penetrated by the arthroscope shaft with a blunt trocar. A 2.7-mm arthroscope with an inclination angle of 30 degrees is introduced (Fig. 18.28). After a spinal

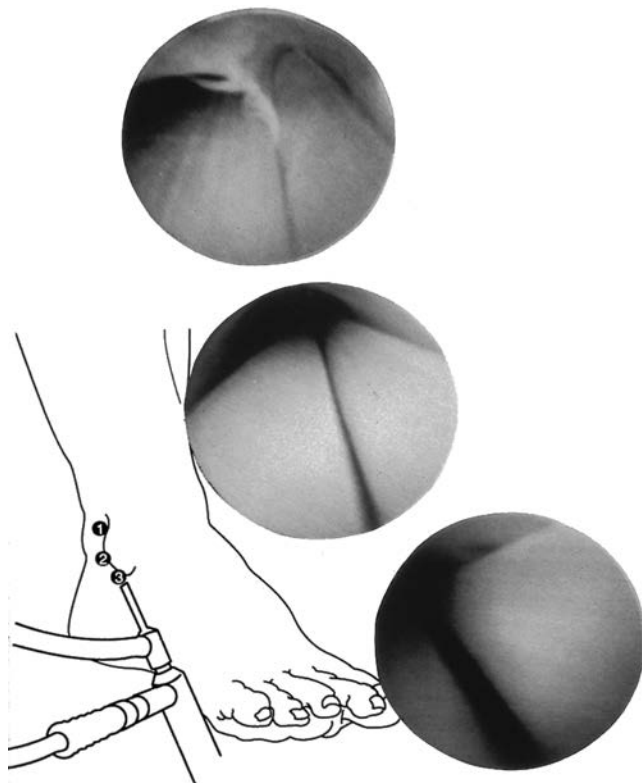


Fig. 18.29 Through the distal portal a complete overview can be obtained of both peroneal tendons. The inspection starts approximately 6 cm proximal from the posterior tip of the lateral malleolus where a thin membrane splits the tendon compartment into two chambers (1). More distally (2, 3) both tendons lie in one compartment.

needle is introduced under direct vision, an incision is made through the skin into the tendon sheath to create a proximal portal. Instruments such as shaver system can be introduced. Through the distal portal on the lateral side, a complete overview can be obtained of both peroneal tendons. The inspection starts approximately 6 cm proximal from the posterior tip of the lateral malleolus, where the tendon compartment is split into two chambers by a thin membrane (Fig. 18.29). More distally, both tendons share one compartment. The complete compartment can be inspected by rotating the endoscope over and between the two tendons. A vincula-like membrane by which both tendons are attached to the tendon sheath allows the arthroscope to rotate freely all around each tendon. The muscle fibers of the peroneus brevis can be recognized in the thin membrane up to the tip of the fibula. At this location both tendons cross the calcaneofibular ligament, which usually gives some fibers to the anterior talofibular ligament. Approximately 3 to 5 cm distal from the fibula the tendons cross each other and again get divided by a membrane and a bony prominence. Here, a pathologic thickened vincula or tendon sheath can be released, adhesions can be removed, and a symptomatic prominent tubercle can be removed. When a total synovectomy of the tendon sheath is to be performed, it is advisable to create a third portal more distal or more proximal from the previously described portals. In case of treatment for recurrent peroneal tendon dislocation, it is possible to deepen the groove of the peroneal tendons with a burr.⁴¹ In case of a peroneal tendon tear, the tendon can be



Fig. 18.30 Left ankle. In patients with paratendinitis and tendinosis there is localized swelling approximately 4 to 7 cm above the insertion of the Achilles tendon.

arthroscopically debrided followed by a mini open procedure for tubularization of tendon. Several authors suggest that when >50% of the cross-sectional area of the tendon is involved in the tear, the tendon is considered irreparable requiring more advanced surgical treatment. A recent international consensus statement, however, concluded that this threshold remains quite arbitrary and a surgeon should always try to repair the tear in case some reasonable native tendon is present.⁴⁴ In case one or both tendons are nonreconstructable, grafting of the tendon(s) is favoured.⁴⁴

Achilles Tendon Pathology—Paratendinitis and Tendinosis (see also Chapter 9)

Overuse injuries of the Achilles tendon can be divided into insertional and noninsertional pathology.^{45,46} Noninsertional tendinopathy can be divided into three subgroups:

- Paratendinitis, characterized by inflammation on the lining of the tendon. In case of acute paratendinitis, there is diffuse swelling around the tendon. Most cases of isolated paratendinitis respond well to conservative treatment.
- Paratendinitis combined with tendinosis, most often presenting with localized swelling 4 to 7 cm above the insertion of the Achilles tendon (Fig. 18.30). On examination there is pain, particularly when the tendon is squeezed. Most often the pain is localized predominantly on the medial side. MRI demonstrates marked thickening of the tendon.
- Tendinosis, where fields of local degeneration in the tendon are present. Since no evidence is available showing inflammation in these patients, the term *tendinitis* has been exchanged for *tendinosis*.^{34,47} With advanced tendinosis, the tendon elongates because of chronic degeneration and is no longer in functional continuity. There often is an increase in passive range of dorsiflexion.



Fig. 18.31 For peritendinitis of the Achilles tendon, the portals are created 2 cm distal and proximal of the lesion. After a spinal needle is introduced under direct vision, an incision is made at the location of the proximal portal.

When treating Achilles tendinopathy, several treatment options are proposed in literature. Heavy-load eccentric calf-muscle training has been demonstrated to be an effective treatment for chronic Achilles paratendinitis. When treating paratendinitis surgically, the diseased and thickened paratenon is excised. Operative treatment of chronic tendinosis consists of debridement of the paratenon and removal of degenerative necrotic tissue. The thickened degenerative portion of the tendon is excised, and the defect is closed primarily. Revascularization is stimulated by making multiple longitudinal incisions into the tendon. Open surgery, however, produces a guarded prognosis. In fact, Maffulli et al.⁴⁸ reported poor results in more than 60% of patients. Therefore, an arthroscopic approach is recommended.

Surgical Technique

For peritendinitis of the Achilles tendon, the portals are created 2 cm proximal and 2 cm distal of the lesion (Fig. 18.31). The distal portal is made first: an incision is made through to the skin only. The crural fascia is penetrated by the arthroscope shaft with a blunt trocar, and a 2.7-mm arthroscope with an inclination angle of 30 degrees is introduced. After a spinal needle is introduced under direct vision, an incision is made at the location of the proximal portal. An instrument such as a probe is then introduced and the pathologic paratenon is removed by a

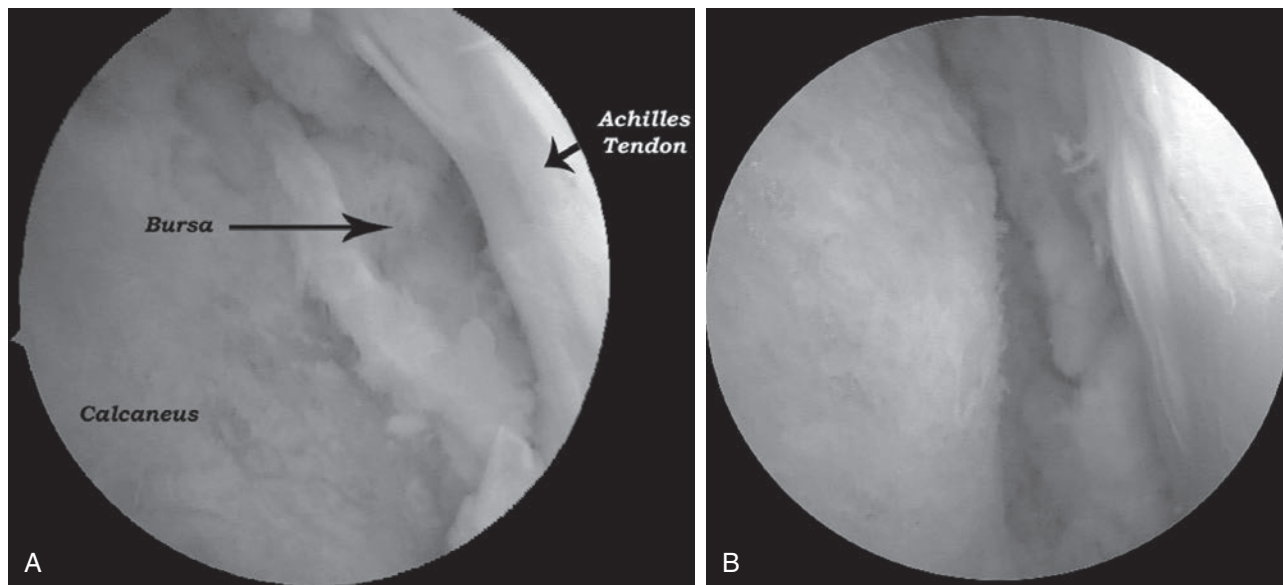


Fig. 18.32 (A) Achillotendoscopy: retrocalcaneal bursitis. (B) After removal of the bursa and inflamed soft tissue, the calcaneal prominence is removed with a full-radius resector and small acromionizer.

small shaver. The Achilles tendon can be inspected by rotation of the scope over the tendon. The plantaris tendon can be recognized and released, or resected when indicated.

Achilles Tendon Pathology—Insertional Tendinitis and Retrocalcaneal Bursitis

Insertional tendinitis can be subdivided into three categories:

- (Chronic) retrocalcaneal bursitis, accompanied by deep pain and swelling of the posterior soft tissue just in front of the Achilles tendon (Fig. 18.32). The prominent bursa can be palpated medially and laterally from the tendon at its insertion. The lateral radiograph demonstrates the characteristic prominent superior calcaneal deformity. Operative treatment involves removal of the bursa and resection of the lateral and medial posterosuperior aspect of the calcaneus.
- Retrocalcaneal bursitis, frequently combined with midportion insertional tendinosis. Often a partial rupture of the midportion of the tendon is present at its insertion. When operative treatment for retrocalcaneal bursitis is indicated, debridement of the midportion of the Achilles insertion should be considered in case of a partial rupture.³⁴
- Isolated insertional tendinosis, leading to pain at the bone-tendon junction that worsens after exercise. The tenderness is specifically located directly posterior to the junction. Radiographic signs of ossification at the most distal extent of the insertion of the tendon (bone spur) are typical signs of insertional Achilles tendinosis. Most patients can be managed with nonoperative measures, such as widening and deepening of the heel counter of the shoe. When operative treatment is indicated, the pathologic ossifications and spurs can best be approached by a central heel-splitting incision.

Open surgery for insertional tendinitis with removal of the chronically inflamed bursa and the posterosuperior prominence of the calcaneus can be associated with a poor outcome. Moreover, open surgical treatment requires plaster immobilization to prevent equinus malformation and to stimulate wound

healing. Angermann and Hovgaard⁴⁹ reported a cure rate of only 50% after open surgery for chronic retrocalcaneal bursitis. Endoscopic treatment offers the advantage of less morbidity, reduced postoperative pain, and outpatient treatment. It provides easy access to the narrow anatomical space around the Achilles tendon and is associated with a lower rate of complications and requires a shorter rehabilitation period.⁵⁰

Surgical Technique

Achilles tendinosis for retrocalcaneal bursitis is performed with the patient in the prone position. Two portals are created, medial and lateral to the Achilles tendon, at the level of the superior border of the os calcis. A 4-mm arthroscope with an inclination angle of 30 degrees is introduced through the posterolateral portal. A probe and subsequently a 5-mm, full-radius shaver are introduced through the posteromedial portal. After removing of the bursa and inflamed soft tissue, the surgeon uses a full-radius shaver and small acromionizer to remove the calcaneal prominence.

Subtalar Joint Arthroscopy and Intraosseous Talar Cysts

Subtalar arthroscopy was first described in 1985,⁵¹ and may be applied as both a diagnostic and therapeutic tool. As with any other joint, the subtalar joint should be compartmentalized and examined. Indications for subtalar arthroscopy include the evaluation of subtalar instability, debridement of osteochondral lesions, and excision of avulsion fragments or loose bodies. The anterior subtalar joint consists of the anterior facet, middle facet, talonavicular joint, and spring ligament. The dividing axis through the subtalar joint consists of the sinus tarsi, tarsal canal, cervical ligament, talocalcaneal interosseous ligament, inferior extensor retinaculum, and fat pad. The posterior subtalar joint consists of the posterior facet that is 40 to 45 degrees lateral to the longitudinal axis of the foot, the capsule, the posterior recess, the lateral recess (thickened by the calcaneofibular ligament), and calcaneus.



Fig. 18.33 Subtalar joint arthroscopy: the posterior lateral portal is made approximately 1 cm posterior and 1 cm proximal to the tip of the fibula.

Surgical Technique

The patient may be positioned supine or laterally with a bolster under the foot at the edge of the table. A 1.9- to 2.7-mm arthroscope with 30-degree wide angle is used. Small joint shavers and burrs can be introduced. When needed, soft-tissue distraction can be performed. Four portals have been described.⁵¹ The anterior lateral portal is made in the sinus tarsi 2 cm anterior and 1 cm inferior to the tip of the lateral malleolus. Caution should be taken not to injure the superficial peroneal nerve. The inframalleolar portal is made anterior to the calcaneofibular ligament. Caution should be taken not to injure the peroneal tendons. The posterior lateral portal is made 1 cm posterior and 1 cm proximal to the tip of the fibula (Fig. 18.33). Caution should be taken not to injure the sural nerve, lesser saphenous vein, and peroneal tendons. The medial portal is made in the sinus tarsi approximately 2 cm anterior to the tip of the lateral malleolus. A blunt trocar is introduced through the deep fascia and guided gently through the tarsal canal to the medial skin surface. The foot is placed in equinus to relax the neurovascular structures. An incision is made over the trocar. A blunt trocar is introduced from the medial portal. The joint is insufflated and the arthroscope is introduced to view the anterior lateral and posterior medial subtalar joint. Caution is taken to avoid the neurovascular structures, which are approximately 2.5 cm distal to the tip of the medial malleolus. A systematic examination of the subtalar joint is performed by varying the portal placement of the scope. An arthroscope in the anterior lateral portal enables evaluation of the sinus tarsi, interosseous ligament, cervical ligament, and lateral and posterior gutters. Placing the arthroscope in the posterior lateral portal enables evaluation of the lateral gutter and lateral compartment. An arthroscope in the medial portal enables evaluation of the anterior lateral and posterior medial compartments. The major complications specific to this procedure are sural nerve injury at the posterior lateral portal, superficial peroneal nerve injury at the anterior lateral portal, and peroneal tendon disruption at the inframalleolar portal. The arthroscope may be placed inadvertently in the ankle joint or may penetrate the capsule of the ankle and enter

the lateral ankle gutter. For this reason, fluoroscopic confirmation of position can be useful.

Assessment of the posterior articulation of the subtalar joint can best be performed by means of a two-portal endoscopic approach of the hindfoot with the patient in the prone position.¹³ The therapeutic indications include debridement of chondromalacia, excision of osteophytes, the removal of a loose body, lysis of adhesions with posttraumatic arthrofibrosis, and synovectomy. Intraosseous talar cysts can be approached through the subtalar joint.⁵² Retrograde curettage of these lesions with destruction of the surrounding zone of sclerosis, along with bone grafting, is the treatment of choice. Lesions with a communication to the subtalar joint can be treated with the patient in the prone position. For proper preoperative planning, a CT scan is indispensable.

A shaver is introduced through the posteromedial portal. After identification of the FHL tendon, the posterior talar process is freed from its capsular attachments. The joint capsule of the subtalar joint is resected, and the opening of the cyst in the subtalar joint is identified by direct vision and palpation by means of a small probe (Fig. 18.34, A and B). With the endoscope in the posteromedial portal and the probe in place through the same posterolateral portal, the drill guide is introduced. The drill guide is parallel to the probe of which the curved tip is in place in the opening of the cyst in the subtalar joint. The drill guide is positioned onto the posterior talar process and a hole is drilled into the cystic lesion with a 4.5-mm drill (Figs. 18.35 and 18.36). The lesion is curetted and debrided with a closed-cup curette. The opening of the cyst is enlarged to 6.5 mm, and a 6.5-mm blunt trocar is introduced. Multiple drill holes are made through the cystic wall from inside the lesion with a K-wire (Fig. 18.36). Cancellous bone obtained from the iliac crest is packed into the cystic lesion through the trocar (Fig. 18.37). This two-portal endoscopic approach offers an excellent alternative to open techniques, with obvious advantages. An arthrotomy or malleolar osteotomy is prevented. The articular origin of a cyst can be identified under direct arthroscopic vision. A second portal makes it possible to probe and subsequently treat the lesion by debridement, drilling, and transtrocar bone grafting. Excellent results at follow-up have been reported by using this technique.^{12,52}

Combined Anterior and Posterior Ankle Arthroscopy

In case of combined anterior and posterior ankle pathology, anterior arthroscopy can be combined with posterior arthroscopy of ankle in the same operative setting. Indications include rheumatoid arthritis, pigmented villonodular synovitis, chondromatosis, or ankylosis.

Surgical Technique

First, the patient is placed in the prone position for the posterior approach. A tourniquet is applied and a small support is placed under the lower leg. A two-portal endoscopic approach of the hindfoot is performed and the pathology can be treated. When the procedure has been finished, the portals are closed and the wound is draped, a strap is placed around the foot, the knee is flexed approximately 90 degrees, and the strap is attached with

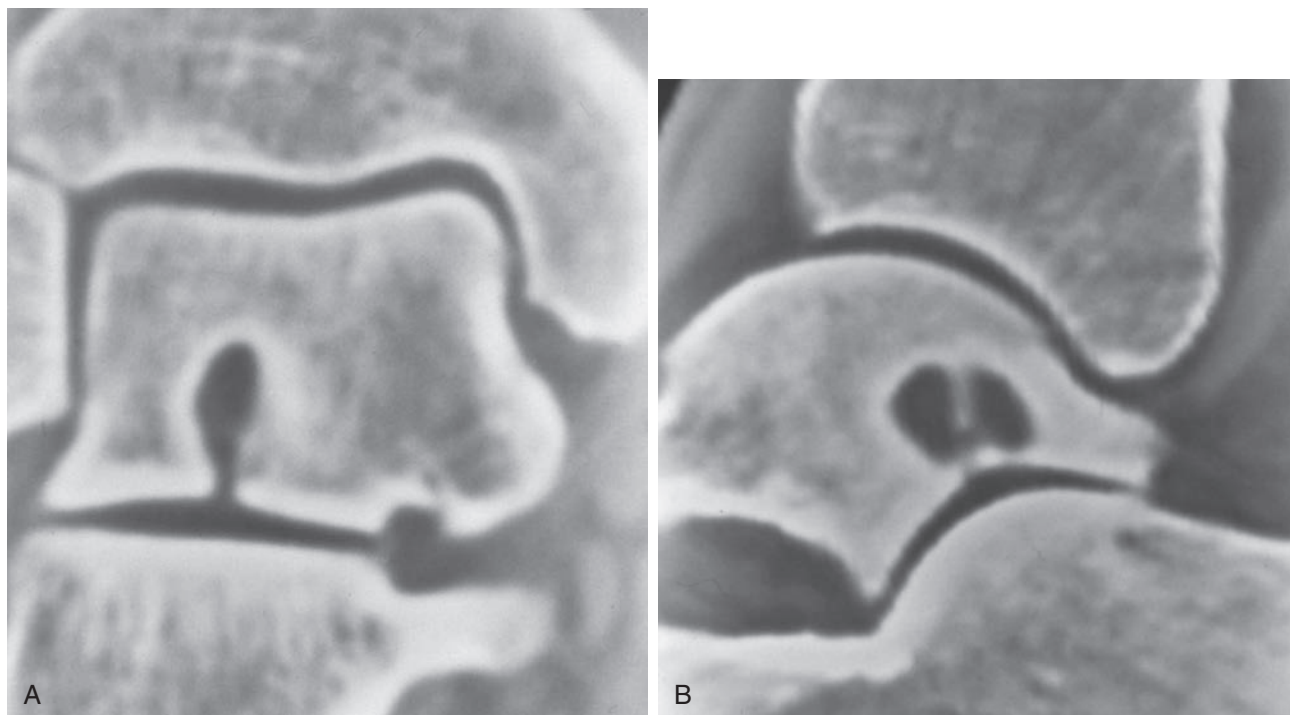


Fig. 18.34 (A and B) A computed tomography (CT) scan is indispensable for proper preoperative planning. This CT scan shows an intraosseous cyst of the right talus that has communication with the subtalar joint.

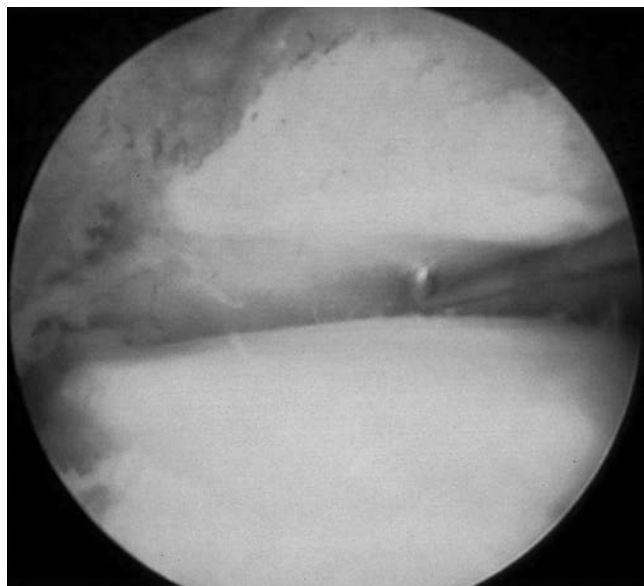


Fig. 18.35 The opening of the cyst (see Fig. 18.36, A and B and text) in the subtalar joint is identified by direct vision and palpation by means of a small probe.

a string to the ceiling of the operating room. The foot is now hanging upside down (Fig. 18.38). Next, the portals for the anterior ankle arthroscopy are made and synovectomy or capsulectomy is performed in the anterior ankle compartment

REHABILITATION

In general, postoperative rehabilitation consists of a compressive bandage and partial weight bearing for 3 to 5 days. The patient

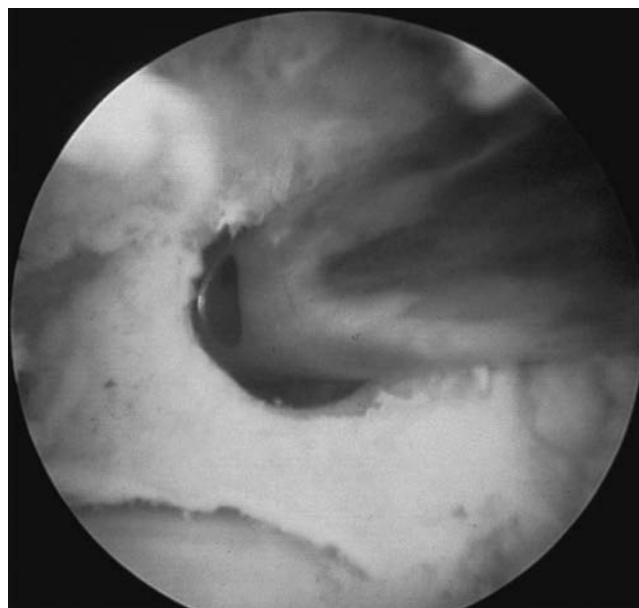


Fig. 18.36 The drill guide is positioned, parallel to the probe, onto the posterior talar process. With a 4.5-mm drill, a hole is drilled into the cystic lesion.

is instructed to actively dorsiflex his or her ankle and foot on awakening and to continue this exercise a few times every hour for the first 2 to 3 days after surgery. In cases where 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 a non-weightbearing lower leg cast. Next, 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.

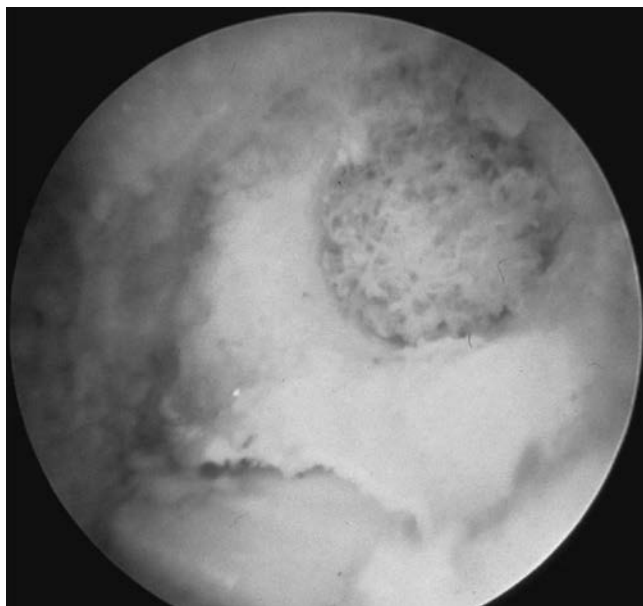


Fig. 18.37 Cancellous bone obtained from the iliac crest is packed into the cystic lesion through a trocar.



Fig. 18.38 Combined anterior and posterior ankle arthroscopy. Posterior ankle examination is carried out with the patient in the prone position. Anterior ankle examination is carried out with the foot hanging upside down.

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Foot and Ankle Endoscopy

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INTRODUCTION

In the last decade, minimal invasive management of multiple foot and ankle pathologies has been proposed, seeking to reduce the postoperative complications that occur in patients classically treated with open surgical management. Arthroscopy and endoscopy has emerged as a powerful minimally invasive tool for intra- and extra-articular pathologies in foot and ankle. As a diagnostic tool, it has been utilized to evaluate articular osteochondral lesions, impingement lesions, syndesmotic instability, and lesser metatarsophalangeal instability, among others. In fracture cases, arthroscopic assistance has been reported to be very useful too, as in pilon, tillaux, talus, and calcaneus fractures, to mention some. For tendinous pathology, increasing indications for diagnostic and treatment alternatives have been reported for the Achilles tendon, posterior tibial, and peroneal tendon, among others. In this review, we will summarize arthroscopic or endoscopic alternatives for foot and ankle pathologies excluding the ankle, subtalar, and metatarsophalangeal joints.

ACHILLES TENDOSCOPY

Achilles endoscopic surgery is a minimally invasive option that has been described to manage different pathologies related to

the Achilles tendon, including non-insertional Achilles tendinopathy, Haglund's deformity, equinus contracture, and even Achilles ruptures.

Tendoscopic surgery minimizes some of the risks present in some open procedures by reducing the complications associated with open surgery, such as scarring, perioperative pain, and wound complications, and it may also provide a faster recovery.¹

Anatomy

The Achilles tendon, or calcaneal tendon, is the thickest and strongest tendon in the human body. It measures approximately 12 to 15 cm in length and is formed by the junction of the aponeurosis of the gastrocnemius (attaches on the distal femur, has two muscle heads, one medial and other lateral, crosses the knee, ankle, and subtalar joints) and soleus muscle (originates posteriorly off the proximal tibia and fibula, and crosses the ankle and subtalar joints). Structurally, the Achilles tendon is formed by fascicles separated by endotenon. Each fascicle is composed of a mature type of fibroblasts denominated tenocytes; these are located in a matrix made of glycoproteins, mucopolysaccharides, elastin, and collagen.

Throughout the tendon trajectory, there is a lateral 90-degree twist, finally inserting itself between 13 and 30 mm distal to the

superior portion of the posterior tuberosity of the calcaneus.² The previous twist explains the fact that the fibers provided by the gastrocnemius muscle insert at the lateral aspect of the posterior process of the calcaneus and the fibers of the soleus muscle insert at the medial aspect. Some of the insertional fibers are in continuity with the plantar aponeurosis.³

The Achilles tendon does not have a sheath, is surrounded by a paratenon that is divided into an outer parietal and inner visceral layer, with a mesotenon layer in the space between both, to provide the blood supply. At the Achilles insertion, the blood supply is limited and is provided by periosteal and osseous vessels. For this reason, it is believed that degenerative changes are often seen in this area and may explain prolonged recovery times for tendinopathic tendons.

Just before its insertion on the calcaneus between the ventral side of the distal part of the Achilles tendon and the calcaneus, the retrocalcaneal bursa is found,¹ which can be inflamed and produce pain. This chronic inflammation can occasionally be detected on plain films because it can disturb Kager's triangle.⁴

Biomechanics

In the gait cycle, the Achilles tendon is placed in maximum tension during the late-stance phase. The gastrocnemius limits the degree of ankle's dorsiflexion while the knee is in extension, whereas the triceps limits dorsiflexion with the knee in flexion. The late-stance phase dorsiflexion beyond 10 degree of dorsiflexion is dependent on the flexibility of the gastrocnemius muscle.⁵

The soleus and gastrocnemius muscles account for almost 90% of energy in plantarflexion, and the Achilles tendon supports the body weight 12.5 times during running activities.^{3,6}

Indications

Chronic achilles tendinopathy has a high prevalence in older athletes. Most of the injuries are caused by overuse during running activities (33%).⁷ Clement and colleagues theorized that the injuries are caused by microtrauma produced by fatigued muscles, eccentric loading, or excess tendon loading.⁸

In 1999, Alfredson and colleagues found there were no significant differences in the mean concentrations of prostaglandin E2 between tendons with the diagnosis of "tendinosis" and normal tendons.⁹ For this reason, the term "tendinitis" is an inaccurate diagnosis, because the inflammatory reaction rarely is found in the tendon. Instead, degenerative changes have been found as intraoperative findings in patients with overuse injuries, such as calcifications; fibrocartilaginous metaplasia; and fatty, hyaline, and myxoid degeneration.⁷ Chronic Achilles tendinopathy can be divided into non-insertional, which can present as a local degeneration of the tendon combined with paratendinopathy, or insertional where tendinopathic changes are found at the insertion, including the retrocalcaneal bursa and the posterior process of the calcaneus.¹⁰

Non-insertional Achilles Tendinopathy

Non-insertional pathology of the Achilles tendon is frequently found in athletes and runners, who present clinically with painful swelling 4 to 6 cm proximal to the insertion in the calcaneus.

It can be divided in three different entities: tendinopathy, paratendinopathy, and a combination of both.⁷

Overuse is commonly associated with this condition, but other factors have been described, such as poor training technique, improper training surface, strength imbalance, compression and friction of the tendon, shoe-related factors, rheumatoid arthritis, and endocrine disorders.

Diagnosis

The Achilles tendon is easily palpable even in obese patients, and local tenderness and swelling should be assessed. If the focal tenderness or swelling moves up and down with passive dorsiflexion and plantarflexion, it suggests that the patient suffers from a tendinopathy. This condition can present three different patterns: local degeneration of the tendon but mechanically intact, diffuse thickening of the tendon, or insufficiency of the tendon with a rupture. In paratendinopathy, the swelling does not move and its pathology is exclusively in the paratenon, clinically and also on magnetic resonance imaging (MRI). Often, pain is more severe on the medial side of the achilles.¹¹ Some investigators have theorized that the degeneration of the soleus tendon and a relatively fixed plantaris tendon is a possible explanation for this medial pain. Medially, the soleus and plantaris tendons are separated by the paratenon and with the simultaneous movement of the knee and ankle both tendons are pulled at the same level and may generate pain.¹

Imaging in Achilles tendinopathy demonstrates intra-substance or intrinsic inflammatory changes of the tendon, with MRI findings including fusiform thickening and linear or diffuse low to intermediate signal intensity on T2-weighted or STIR images. On the sagittal MRI view, an anterior convexity or a focal enlargement of the Achilles tendon can be observed. When paratendinopathy is present, where the inflammation is seen in the paratenon, adhesion can be found between the paratenon and the Achilles tendon.¹

Some other diagnoses should be ruled out when evaluating Achilles tendinopathic patients, like tendinitis of the tibialis posterior or flexor hallucis longus tendon; peroneal tendinopathy; posterior subtalar arthritis; ankle arthritis; tarsal tunnel syndrome; posterior ankle impingement; seronegative arthritis; and systemic diseases, including seropositive arthritis (Rheumatoid arthritis, lupus).⁷

Treatment

Conservative management (nonsteroidal antiinflammatory drugs, activity modifications, shoe modifications, insoles, immobilization, and eccentric stretching)^{1,12} is the first-line of treatment. The use of steroid injections in any presentation is controversial, and injections around or inside the Achilles tendon can promote degeneration and increase the risk for a subsequent rupture of the tendon.¹³ In refractory cases, shock wave therapy could be considered as second-line intervention because of its similar effectiveness to tendon eccentric strengthening.¹⁴ In patients with combined paratendinopathy and tendinopathy, it can be difficult to determine which pathology needs to be treated, because the role

of the focal tendinopathy on clinical pain is not clear.¹⁵ In the majority of the cases, management is focused on treatment of the paratendinopathy, because Alfredson and colleagues in 1999 described as a possible cause of pain the neovascularization around the tendinopathic nodule in patients with a combined pathology.¹⁶

Up to 25% to 45% of patients do not respond to conservative treatment after 6 months, and may require surgical intervention.^{4,13} Open Achilles tendon surgical management for tendinopathy consists in a decompression of the tendon by incision/excision of the degenerative tissue. Releasing the plantaris tendon at the level of the nodule and removing the focal thickened paratenon is the classic procedure in cases of paratendinopathy.¹ Great attention should be paid to the anterior aspect of the tendinopathic area, which presents greater neovascularization changes and neural ingrowth.¹⁷ To obtain optimal results from surgery, some surgeons have tried to achieve all of these goals during surgery. The results of open surgery have been reported to be satisfactory in 50% to 96%¹⁸ of patients, but complications such as prolonged recovery, skin edge necrosis, deep vein thrombosis, superficial wound infection, seroma, hematoma, scarring, and sural nerve irritation have been described.¹⁹ No clear guideline exists relative to how to treat surgically this condition, as we do not even know the pathophysiology of the disease.

Based on these previous factors, an endoscopic option has been developed, trying to achieve the same goals as open surgery but hopefully avoiding its drawbacks. The endoscopic technique has been able to achieve decompression of the tendon by excision of degenerative tissue, excision of thickened paratenon, lysis of adhesions, and stimulation of healing response. One aspect not obtainable with tendoscopy is the removal of degenerative tissue from the substance of the tendon.^{1,20}

Operative Technique

As described by van Dijk and colleagues,²¹ the patient is on the operating table in prone position, with a pneumatic tourniquet placed around the upper thigh. The foot is placed at the end of the table to allow the surgeon to move it in full dorsiflexion and plantarflexion. Before inflating the tourniquet, the surgeon should mark the short saphenous vein on the lateral side of the Achilles tendon.

- **Instruments required:**

A small-diameter short arthroscope (2.7 mm with a 30-degree angle) or a standard 4-mm arthroscope. The first one yields an excellent picture but cannot deliver the same amount of irrigation fluid per time than the standard one. With a 4-mm arthroscope the gravity inflow of the irrigation is usually sufficient, but for the 2.7-mm arthroscope a pump device or a pressurized bag sometimes is used.²² Additionally, a probed endoscopic shaver system is used.

- **Portals:**

The distal portal is first made and is located 2–3 cm distal to the pathologic nodule on the lateral border of the Achilles tendon. Once the skin incision is made, a mosquito clamp is introduced, followed by the cannula in a craniolateral direction. In the same direction, the



Fig. 19.1 Endoscopic achilles tendon portals for non-insertional Achilles disease.

arthroscope is introduced, looking ventrally over the edge of the tendon on the craniolateral side. The cranial portal is located 2–4 cm above the pathologic nodule on the medial border, and is made in the same manner, after localizing the correct site under direct visualization, and the use of a needle (Fig. 19.1).

The Achilles tendon is easy to identify on the healthy part. With the portals previously mentioned, it is possible to visualize and work around the whole surface of the tendon over 10 cm of length approximately. To avoid and minimize the risk of damage to neurovascular structures always keep the arthroscope on the tendon. The plantaris tendon is identified medial to the Achilles tendon.

- **Procedure:**

With the shaver, all fibrotic tissue binding the sheath or retrocalcaneal bursa to the tendon on the deep tendon surface can be removed (Fig. 19.2). Also, Kager's fat pad can be debrided if necessary. In paratendinopathy the plantaris tendon is released from the Achilles tendon, and the thickened paratenon is resected on the anterior aspect of the tendon at the level of the pathologic nodule. The neovascularization, which is accompanied by small nerve fibers, can be found in this area, and it also can be removed. Changing portals can be helpful. At the end of the tendoscopy it must be possible to move the arthroscope over and around the symptomatic area of the tendon. To prevent a postoperative draining sinus, skin incisions must be sutured, and a compressive dressing is applied.

After Care

In our experience, patients can bear weight as tolerated, using a CAM walker boot for 2 weeks. During this period of time the foot



Fig. 19.2 Debridement of Kager's fat pad and retrocalcaneal bursa adhesions on the deep tendon surface.

must be elevated when not walking. After 2 weeks, aerobic exercises and concentric strengthening exercises are initiated as tolerated.^{1,4,20} At the fourth week the patient is able to walk uphill, and at this point jogging or light impact activities can be initiated.²⁰

Results

Steenstra and van Dijk¹ in 2006 published successful results with no complications in 20 patients with non-insertional tendinopathy treated endoscopically. The Foot and Ankle Outcome and SF-36 scores were comparable to a cohort of people without Achilles tendon complaints after 6 years of follow-up. Maquirriain²³ reported in 96% of patients that were treated with endoscopic debridement of peritendinous tissue and percutaneous longitudinal tenotomies a complete resolution of symptoms at 7 years postoperatively. Lui²⁴ also described an Achilles endoscopic debridement associated with a flexor hallucis longus transfer in five patients with Achilles tendinopathy obtaining good results.

In our own experience, we have performed endoscopic debridement and release of the ventral aspect of the Achilles tendon, which is, in the author's opinion, the most important location where to remove diseased tissue, obtaining excellent functional results in 10 of 11 patients (full pain relief at rest observed at a median of 9 weeks, achieving full sports activities at 4 months of average) with a median postoperative VISA-A score of 100 (30–100) points at final follow-up (2 years).²⁰ Similar results have been obtained by Thermann et al.²⁵ in his series of eight patients who underwent an endoscopic debridement of the ventral and dorsal aspect of the tendon. With these results, we suggest that it is not necessary to excise the tendinopathic tissue putting at risk the tendon or consider additional procedures like tendon transfers^{20,25} and it would suffice in order to obtain successful results to ventrally remove the diseased area and any adhesions including the neovascularization normally present.

Insertional Achilles Tendinopathy

Insertional Achilles tendon pathology can present with an enlarged posterosuperior aspect of the os calcis (also called



Fig. 19.3 Lateral x-ray view of the hindfoot. Enlarged posterosuperior aspect of the os calcis (A); calcaneal spurring at the insertion of the Achilles tendon (B).

Haglund's deformity), with or without retrocalcaneal bursitis and insertional tendinopathy. Most commonly, these characteristics are all present with varying degree of severity.^{26,27}

Diagnosis

On physical examination, patients with insertional Achilles tendinopathy have pain and tendon thickening at its calcaneus insertion, and can also present tenderness to palpation on the superolateral aspect of the calcaneus.¹² On clinical inspection, it is possible to see the deformity secondary to the bony prominence at the Achilles insertion.

Imaging the lateral x-ray view of the hindfoot allows the treating physician to evaluate the posterosuperior calcaneal border, the calcaneal spurring at the insertion of the Achilles tendon, and the obliteration of Kager's triangle provoked by the retrocalcaneal bursitis (Fig. 19.3). In addition, the MRI can be useful to rule out intrasubstance tendon degeneration.²⁷

Treatment

The nonoperative management consists of shoe wear modifications, nonsteroidal antiinflammatory drugs, night splinting, heel inserts, rest, and eccentric strengthening. In refractory cases, as well as in non-insertional Achilles tendinopathy, shock wave therapy can be used. However, 50% to 56% of patients may not respond to nonsurgical treatment after 6 months.²⁸

The classic open surgical approach consists in a debridement of the Achilles tendon, retrocalcaneal bursectomy, and resection of the posterosuperior aspect of the calcaneus.²⁹ Depending on

the extension of the Achilles lesion distally and the resulting debridement, in some cases a reinforcement of the insertion is recommended. Some complications have been described with the open technique, including skin breakdown, Achilles avulsion, scar hypersensitivity, altered skin sensation about the heel, inadequate resection, and postoperative stiffness, although they are rare.³⁰ The endoscopic approach first described by van Dijk and colleagues²¹ in 2001 may be used, particularly in younger patients who have mild to no Achilles tendinopathy, and where the main findings are retrocalcaneal bursitis and a Haglund deformity. In some cases, an impingement tendinopathy can be found, with bone edema and focal tendinopathy localized to the point where contact happens between the tendon and the calcaneus.³¹ In these cases, no real Haglund deformity is found; rather, a posterior prominence of the calcaneus may exert more pressure on the tendon and therefore explain the focal tendinopathy. This situation, in the author's opinion, is the best indication for an endoscopic approach.

Operative Technique

As described by van Dijk and colleagues,²¹ the patient can be placed on the operating table in prone or supine position, with a pneumatic tourniquet placed around the upper thigh. In the prone position, the patient's feet are positioned just over the edge of the operating table, while in the supine position, the arthroscopic leg holder is needed to hold the leg around the calf, the end of the table is dropped down, and the other leg is placed out the operative field. In both positions, the surgeon is allowed to move the foot in full dorsiflexion and plantarflexion.

- **Instruments required:**

A small-diameter short arthroscope (2.7 mm with a 30-degree angle) or a standard 4-mm arthroscope. The setting is similar to the one already described for the non-insertional tendinopathy.

- **Portals:**

The lateral portal is made first, at the level of the superior aspect of the calcaneus; the incision should be made immediately adjacent to the Achilles tendon. The retrocalcaneal space is penetrated by a trocar, followed by the arthroscope; the medial portal is located at the same level but on the medial edge of the Achilles tendon. The medial portal is similarly established under direct vision, utilizing a needle (Fig. 19.4).^{4,21}

- **Procedure:**

The inflamed retrocalcaneal bursa is removed through the medial portal, allowing the surgeon to see the posterior calcaneus and the Achilles attachment. With the foot in full dorsiflexion, the impingement site is easily identified. Then, the foot is placed in full plantarflexion and the posterosuperior calcaneal prominence is resected with a synovial resector or arthroscopic burr, taking care not to injure the tendon, which is protected by keeping the closed end of the shaver against the Achilles tendon.^{4,21} Adequacy of the resection is confirmed with lateral fluoroscopy images with the ankle in full dorsiflexion, and confirms the absence of impingement. In fully plantar flexed foot position, the insertion of the Achilles tendon



Fig. 19.4 Endoscopic Achilles tendon portals for insertional Achilles disease.

can be identified, and all the disease area is removed with the shaver. In the tendinopathic areas, as arthroscopically the debridement is very limited, is possible to trephine these areas with a needle, looking for a vascular response.²¹

After Care

The patient should remain nonweight bearing for 2 weeks, depending on local edema and pain. The following 2 weeks the patient is allowed to perform range-of-motion exercises and weight bearing in a CAM walker boot. Return to normal footwear may be initiated as early as 4 weeks.²¹

Results

Only small series can be found in the literature reporting endoscopic treatment for insertional Achilles tendinopathy. In endoscopic calcaneoplasty van Dijk et al.²¹ in their initial series obtained good to excellent results after treating 20 patients who had a painful prominence of the posterosuperior aspect of the calcaneus associated with a retrocalcaneal bursitis. Nineteen of these 20 patients returned to sports after 12 weeks. Ortmann and McBryd³⁰ reported on 28 patients after a similar intervention with an average follow-up of 35 months, good clinical results with an American Orthopaedic Foot & Ankle Society score improvement from 62 to 97. No wound complications or infections were reported, but two patients needed an open reintervention, due to a rupture of the Achilles tendon during the third week postoperatively. Jerosch and colleagues³² in 2007 studied 81 patients with an average follow up of 35.3 months after endoscopic treatment obtaining excellent Ogilvie-Harris score in 41 cases, good in 34, fair in 3, and poor in 3 patients. The poor results underwent revision with an open approach and ossification of the Achilles tendon was found.

Evidence-Based Recommendations

It is important to have a thorough understanding of the surgical anatomy and endoscopic technique to minimize surgical complications and obtain good results. No clear guidelines have been established related to the efficacy of the endoscopic technique in Achilles tendinopathy. Only small series have been reported, and over different populations with varying degrees of tendinopathic compromise, which makes it even harder to give recommendations for its use. We believe that tendonoscopy has a role for Achilles tendinopathy, as it represents a minimally invasive option that can deliver successful results with minimal morbidity. The indication for tendonoscopy in non-insertional Achilles tendinopathy is becoming more accepted, with good results and good outcomes consistently reported with few or no complications. As already said, these studies are limited by its low number of patients and retrospective nature, and therefore on a review of the literature performed in 2014 by Cychosz et al.,³³ a grade C recommendation³⁴ (for intervention) to the Achilles tendoscopy was given. No evidence-based recommendation has been given to tendonoscopy for insertional Achilles tendinopathy, and more information with well-designed studies are needed to further elucidate the role of impingement against the calcaneus. The authors believe that this is a promising area for further research where limited surgical approaches will have an important role in the future.

POSTERIOR TIBIAL TENDOSCOPY

Posterior tibial tendon (PTT) endoscopic surgery is the minimal invasive option described to diagnose and treat PTT disorders, such as tenosynovitis, degenerative and post-traumatic tears, dislocation of the PTT, enthesopathies, and chronic tendinopathy with symptomatic flatfoot deformity.³⁵

Tendonoscopy procedure allows functional after treatment, quick recovery, and offers the advantage of less morbidity minimizing neurovascular damage risk, reduction of the postoperative pain, and easier outpatient treatment.³⁶

Anatomy

The PTT is the most anterior and largest tendon in the deep posterior compartment of the leg and have a synovial sheath that is 7–9 cm in length, starting 6 cm proximal to the tip of the medial malleolus. The PTT is originated on the proximal tibia, fibula, and interosseous membrane; descends into the retromalleolar medial groove; and inserts close to the tuberosity of the navicular.^{3,37} When entering the foot, it flattens and presents an increased amount of fibrocartilage, showing also hypovascularized area in the retromalleolar region.^{38,39} This portion of the tendon rubs back and forth between the malleolus and the flexor digitorum longus.⁴⁰

This tendon has no mesotendon, but it has a vinculum, between the posterior side of the PTT and the sheath,² that runs proximally to end with a free edge at around 4.3 cm above the posteromedial tip of the medial malleolus. It is irrigated by vessels from the posterior tibial artery collaterals.

Biomechanics

The PTT rotates the tibia externally and induces foot supination. In the gait cycle the PTT is stretched during the first rocker to allow subtalar pronation.⁴¹ On the second rocker of gait, the PTT helps to center the talus over the navicular. Finally, during the third rocker, the foot behaves like a second-class lever, balanced by the peroneal tendons and the PTT, with this last one blocking the medial column by locking the calcaneocuboid and talonavicular joints.⁴⁰

The PTT strength is more than twice that of the peroneus brevis (PB), its primary antagonist. Inactivity of the PTT produces midtarsal instability, and the propulsive force of the Achilles acts at the mid-foot instead of at the metatarsal heads. Excessive and persistent pronation of the subtalar joint may render PTT insufficient.

Indications

Symptomatic Adult Flatfoot

The PTT dysfunction ranges from tenosynovitis to a flatfoot deformity. Johnson and Strom⁴² classified PTT dysfunction in three different stages, and later Myerson⁴³ refined it, adding a fourth stage.

Diagnosis

Patients in stage I, as defined by Johnson and Strom,⁴² present with tenosynovitis or tendinitis, with a normal tendon length, without foot deformity associated. Usual findings include medial perimalleolar pain, swelling, and tenderness.⁴⁰

On physical examination, patient can perform a single heel rise, sometimes with slight discomfort over medial ligamentous structures, often resulting in a misleading diagnosis of an ankle sprain, delaying the correct diagnosis and adequate early treatment.^{44,45}

In stage II, the PTT has become permanently elongated and usually presents signs of tendinopathy and a flexible flatfoot deformity.⁴⁶ These patients have a too-many-toes sign positive with forefoot abduction during the physical examination, and when performing a single heel rise, mild to moderate weakness is noted. Plain foot radiographs with weight-bearing lateral and dorsoplantar views may allow for the evaluation of PTT disorder in flatfoot deformity cases.⁴⁰

Trauma

Direct or indirect trauma could develop tendinopathy and partial tears of the PTT.⁴⁰ Partial ruptures frequently occur in middle-aged and low sports active people,⁴⁷ but also some cases have been reported in the literature in athletes, secondary to overuse disorders, though its incidence is underestimated.^{48,49} Masterson et al.⁵⁰ reported that complete PTT tears in young people were most often caused by penetrating trauma.

The retromalleolar region of the PTT is more vulnerable to repetitive microtrauma, and where most ruptures occur, because of the poor repair response of the hypovascular fibrocartilaginous tissue. The longitudinal friction and changes in the gliding resistance of the tendon make PTT more susceptible to suffering longitudinal tears.⁵¹ This rubbing may be affected by an irregularity of the posterior aspect of the tibia after ankle fracture and postfracture adhesions producing initially synovitis and pain.⁴⁰

Dislocation and instability are uncommon but can be found in athletes after suffering injuries with inversion and dorsiflexion of the foot. In these cases, tendonoscopy is useful to confirm it.⁴⁰

Diagnosis

Usually, patients present with posteromedial ankle pain associated to a trauma story or an excessive sport activity increase. On physical examination, it is possible to find swelling around the medial malleolus and tenderness on palpation from the tip of the malleolus to the navicular. Also, weakness with supination of the foot on manual testing can be found.⁴⁰

Plain x-rays of the ankle can reveal post-traumatic changes around the medial malleolus. Ultrasound permits an assessment of the PTT integrity, diagnosing partial or total ruptures of the tendon. Also, increased vascularization on doppler and thickening of the peritendinous tissues may suggest tendinopathy. Even dynamic assessment of the PTT in the retromalleolar groove is useful, permitting diagnosed subluxation or dislocation of the tendon.⁵²

Finally, MRI is the gold standard study for PTT disorders, and it helps to assess other abnormalities in surrounding tissues.⁵³

Systemic Inflammatory and Autoimmune Disorders

Tenosynovitis is the most common extra-articular manifestation of autoimmune disorders and rheumatoid arthritis, and it can eventually lead to a rupture of the tendon.⁵⁴ Michelson and colleagues⁵⁵ found alteration of the PTT in patients with rheumatoid arthritis in 13% to 64%. The criteria used were loss of the longitudinal arch, lack of a palpable PTT, and inability to perform a heel-rise.

Treatment

Initially treatment is conservative, including rest, immobilization, nonsteroidal antiinflammatory drugs, cryotherapy, and local ultrasound, for 3 to 6 months.^{40,45} If nonsurgical treatment fails, then surgical management can be indicated. The purpose of surgical treatment is to achieve a pain-free ankle through debridement, synovectomy, and tendon release. These previous goals can be achieved through an open or endoscopic approach, and most commonly surgical indications have been found in stage I flatfoot patients, post-traumatic tenosynovitis, or secondary to an inflammatory disease.⁴⁰ In patients with stage II flatfoot and persistent symptoms after conservative management, surgical treatment remains controversial. Within the most common options, a medial sliding calcaneal osteotomy, in association with a debridement of the PTT and flexor digitorum transfer is the most common one.⁴⁰ Lui and colleagues⁴⁶ reported the transfer combined to an arthroreisis endoscopic-assisted with a reconstruction of a torn PTT.

When a direct trauma causes a complete rupture of the PTT, surgical management and repair can be indicated.

Operative Technique

PTT tendoscopy was first performed by Wertheimer⁵⁶ in 1994, but it was van Dijk who described the technique in detail,³⁶ with the patient in supine position, and a tourniquet on the affected

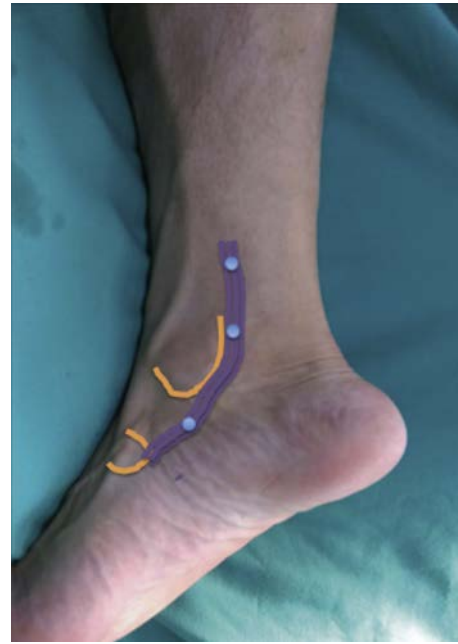


Fig. 19.5 Endoscopic posterior tibial tendon portals.

leg around the upper thigh. The references points are marked on the skin to identify the PPT (asking the patient before the anesthesia to actively invert the foot), the navicular, and the medial malleolus.

- *Instruments required:*

A small-diameter short arthroscope (2.7 mm with an inclination angle of 30 degrees) or a standard 4-mm arthroscope may be used. The setting is similar to the one already described for the Achilles tendoscopy.

- *Portals:*

The distal portal is created first, located halfway between the navicular and medial malleolus following the PTT. Once the sheath is exposed, it is opened perpendicular to the axis of the PTT to avoid enlarging the entry to the tendon with the moving during the procedure. The tendon sheath is penetrated by a trocar, followed by the arthroscope, and filled with saline, inspecting the PTT up to the vinculum.⁵⁴

Under direct visualization, the proximal portal is made around 3 cm proximally to the tip of the medial malleolus. With a blue probe and a shaver through this portal it is possible to inspect the complete tendon sheath (Fig. 19.5).^{36,40,54}

A third portal more proximal, around 7 cm from the medial malleolus, can be used in cases of severe synovitis.⁴⁰

- *Procedure:*

Once the first portal is made, inversion of the foot allows the arthroscope to advance and get a complete overview of the PTT until near 4–7 cm above the tip of the medial malleolus. The tendon sheath can be completely inspected rotating the scope, and the vinculum can be also visualized; if it has an abnormal thickening, it can be debrided or resected if necessary (Fig. 19.6). Adherensiolysis and synovectomy can be performed using a probe to free the tendon and a conventional shaver.^{36,40,54}

Partial ruptures can also be treated and reconstructed with a mini-open approach, while frayed edges or peripheral tears can be resected endoscopically.⁵⁴



Fig. 19.6 Posterior tibial tendonoscopy assessment.

After Care

A compression bandage is used for the first 24 hours and then just an adhesive bandage. Partial weight bearing is allowed for 2 to 3 days. After the third day post-op, weight bearing is allowed as tolerated. Immediately after surgery, active movements like inversion and eversion are encouraged.^{31,40,54}

Results

Posterior tibial tendon tendonoscopy offers advantages over open procedures, namely smaller wounds and reduced risk of infection. Likewise, there is less morbidity and blood loss, quicker recovery, and reduced postoperative pain with early mobilization and functional activity.⁴⁰ van Dijk and colleagues³⁶ reported the outcome in 16 patients with pain over the PTT secondary to different causes (rheumatoid arthritis, post-traumatic), with a focus on the pathological thickening of the vinculum; most of them were free of pain and did not show complications after the tendonoscopy.

Bulstra et al. in 2006 published a study involving a series of 33 patients who underwent PTT tendonoscopy with good results in rheumatoid arthritis and pathologic vincula, but poor results for adherensiolysis, with low complication rate.⁵⁴ At the same time, Chow and colleagues treated endoscopically six patients with symptomatic flatfoot in stage I, with no complications and no progression to stage II.⁴⁴ Similar results were obtained by Khazen and Khazen in 2012.⁴⁵

The most recent publication by Hua et al. in 2015 showed the results of a retrospective review of 15 patients with PTT disorders treated with a posterior arthroscopic approach with no neuromuscular complications.⁴⁶

Evidence-Based Recommendations

There are few quality evidence-based data in the current literature to support the use of PTT tendonoscopy. Only levels IV and V studies are available and reported for tibialis posterior tendonoscopy, with dislocation, tenosynovitis, tendinopathy, and post-traumatic adhesions as the most frequent indications.

Some authors have also reported it as a diagnostic procedure. Because of the low number of studies available for PTT tendonoscopy, on a review of the literature performed in 2014 by Cychoz and colleagues³³ they concluded that it is not possible to make a recommendation for or against this intervention, and assigned a grade I recommendation.³⁴

PERONEAL TENDOSCOPY

The endoscopic approach for peroneal disorders is a technique that has been slowly gaining acceptance, importance, and popularity to be used as a diagnostic procedure and also to treat peroneal tendon pathologies such as tendinopathy, tenosynovitis, peroneal subluxations, and dislocation associated or not associated with ruptures of the peroneal tendons. This minimally invasive approach enjoys the advantages of minimally invasive surgeries, such as small scars, less postoperative pain, and higher patient satisfaction.⁵⁷

Anatomy

The peroneus longus (PL) originates over the proximal two-thirds of the fibula and is completely tendinous 3–4 cm proximal to the tip of the fibula, enters into the fibro-osseous reticular system, and finally inserts at the base of the first metatarsal. The PB originates more distally on the fibular shaft and interosseus membrane, usually has some muscle fibers that extend into the superior peroneal retinaculum, also enters into the fibro-osseous reticular system, and inserts at the base of the fifth metatarsal.⁵⁸

Brandes and Smith⁵⁹ described the zones of peroneal tendon pathology, and later Sammarco and Sammarco⁶⁰ added a fourth zone. Zone A is delimited by the distal fibula anteriorly and the superior peroneal retinaculum posteriorly. At this level, the fibula has an osseous groove that adds stability to the tendons. However, there has been described anatomic variations with the presence or absence of groove deepening structure formed by a periosteal cushion of fibrocartilage that deepens the bony groove. Here the PB is flattened and sits anterior to the PL. Both tendons lie in a common synodal sheath that extends from approximately 4 cm proximal to the tip of the lateral malleolus, to 1 cm distal to it.^{58,61} Zone B corresponds to the location of the inferior peroneal retinaculum. This inferior retinaculum at the level of peroneal tubercle on the lateral wall of the calcaneus. The PL sits inferior to the PB at this location and the tendons are separated and enclosed in two distinct synovial sheaths divided by the peroneal tubercle. Zone C is the groove in the plantar and lateral cuboid where the PL curves. At this level 20% of the individuals present an os peroneus. Zone D corresponds to the level of the tendon insertion for PL and PB.

Finally, both tendons have a 1–2-mm-thick vinculum-like membrane in between, dorsally attached to the dorsal aspect of the fibula.⁵⁷

Biomechanics

The PB provides 63% of total eversion, as well as assisting in ankle plantarflexion. Peroneus longus acts to plantarflex the first ray and evert the foot, and also acts as a secondary plantarflexor

of the ankle, stabilizing the medial column in stance phase. When the PL contracts, the first ray locks at the first tarsometatarsal joint (TMTJ).

The first muscles to contract in response to a sudden ankle inversion stress are the peroneal, and thus are vital to control the dynamic stability of the lateral ankle complex.

Indications

Peroneal Tendinitis and Tenosynovitis

Typically, patients present with posterolateral ankle pain that worsens with activity and improves with rest. On clinical assessment, there is tenderness over the peroneal tendons, and sometimes a palpable mass moves within the tendon.

More commonly tenosynovitis presents in the infra-malleolar portion, and is characterized by thickening and focal tendon degeneration and swelling. Often, associated with splits, tears of the tendon or nodular thickening can be found.

Diagnosis

In zone A the pathology may be limited to impingement caused by excessive or inflamed synovium, and some patients, especially the athletic ones, may suffer from hypertrophy of the PB. Even accessory peroneal muscles can produce symptoms. At zone B it may present tenosynovitis or degenerative tearing of the peroneals in the area of the peroneal tubercle and the inferior extensor retinaculum.⁶⁰ The diagnosis is possible to confirm with ultrasound or MRI, even when the MRI can be misleading in these cases and can erroneously be interpreted as degenerative tendinopathy principally at zone A, although sometimes this condition is assumed to be a MRI artefact.⁶²

Peroneal Subluxation and Dislocation

The principal mechanism is forceful dorsiflexion of the ankle, hindfoot inversion with contraction of the peroneals causing disruption of the superior peroneal retinaculum. The presence of a varus heel and/or a convex retromalleolar groove is a risk factor causing instability.⁶¹

Diagnosis

In general, patients present pain that is localized to the posterior aspect of the fibula. They may report hearing a “snap” or “pop” at the time of the injury or still hear it after the injury with walking, associated to instability sensation when they walk on uneven surfaces. In acute injuries it is possible to identify swelling, tenderness, and posterolateral ankle ecchymosis, with pain produced at the activation of the peroneals muscles.

The ones with chronic injuries usually complain of retromalleolar pain and may refer a snapping sensation along the tip of the fibula or ankle instability. Dorsiflexion and eversion against resistance can reproduce the subluxation.⁶³ Safran and colleagues described a provocative test in which, with the patient's knee flexed, the ankle is actively dorsiflexed and plantarflexed with resisted eversion to assess the dynamic stability of the tendons.⁶⁴

Peroneal Tendon Rupture

There are two possible mechanisms for split lesions of the PB. The first suggests subluxation of the PB, secondary to a laxity

or tearing of the superior peroneal retinaculum, so the PB gets injured with the posterolateral edge of the fibula. The second mechanism theorizes a compression of the PB between the fibula and PL tendon causing a split during an inversion injury.⁶⁵

Diagnosis

The clinical presentation in these patients is similar to that with tendonitis, except the symptoms are usually prolonged, and patients may complain of frequent episodes of weakness. Magnetic resonance imaging is the most used diagnostic method to confirm the diagnosis because it permits visualization of intrasubstance tears and identification of anatomical variants such as the peroneus quartus muscle.⁶⁶

Treatment

The initial treatment is nonsurgical and consists in nonsteroidal antiinflammatory medication, activity modification, rest, and orthoses with lateral forefoot posting in mild cases. In patients with refractory symptoms, immobilization or controlled ankle movement in a walker for 6 weeks may be helpful. The use of corticosteroid injection is controversial, due to the risk of iatrogenic rupture.

If conservative treatment fails, open surgery is indicated and usually consists in an open synovectomy or resection of the distal muscle fibers when symptoms are caused by a hypertrophied PB muscle.

Symptomatic subluxation or dislocation of the peroneals should be repaired surgically. Reconstructive procedures are of three types: (1) rerouting of tendons, involving substituting the calcaneofibular ligament for the incompetent peroneal retinaculum; (2) soft-tissue repair or reconstruction, which could be a direct repair of the superior peroneal retinaculum or using grafts to reconstruct it; and (3) bony procedures including the groove-deepening while preserving the fibro-osseous tunnel to prevent scarring.^{63,66,67}

Management of peroneal tears starts with nonsurgical management as previously discussed for peroneal tenosynovitis. If symptoms persist, surgical treatment is indicated, including debridement and repair of the tendon split.⁶⁶ In one biomechanical study performed by the authors, we suggest that it is safe to leave up to 33% of remaining tendon without risking spontaneous rupture of it, and therefore not needing a tenodesis that is the current indication. For higher-demand patients, we strongly recommend repairing the rupture, either to itself (in case of a split rupture) or to a tendon auto/allograft.⁶⁶

Operative Technique

The peroneal tendoscopy technique was first described in detail by van Dijk,⁶⁸ the patient may be placed in lateral position, with the affected leg slightly elevated, to allow free movement of the ankle joint, and a tourniquet around the upper thigh. Also, patients could be placed prone or supine depending on any concomitant procedure that is planned to be performed.⁶⁹ Anatomic landmarks are palpated and highlighted. The distal part of the fibula, peroneal tubercle, and the fifth metatarsal tuberosity are marked.^{57,68,70}

- **Instruments required:**

A small-diameter short scope (2.7 mm with an inclination angle of 30 degrees) or a standard 4-mm to 4.5-mm scope may be used. The setting is similar to the one already described for the Achilles tendoscopy.

- **Portals:**

The distal portal is made first, around 1.5 to 2.5 cm distal to the apex of the fibula. The tendon sheath is penetrated with blunt trocar, and the scope is introduced. Under direct vision, the proximal portal is created 2 to 3 cm proximal to the posterior edge of the lateral malleolus. Accessory portals can also be performed throughout the complete tendon excursion, according to the pathology that has to be treated.^{57,68,70,71} A plantar lateral portal is developed 1 to 1.5 cm proximal to the tip of the fifth metatarsal tubercle and 1 cm plantar to the tubercle. The plantar medial portal is developed next to the first TMTJ, at the plantar-lateral base of the first metatarsal (Fig. 19.7).⁶⁹

- **Procedure:**

The peroneal tendon sheaths are divided into three zones. Zone 1 extends from the retrofibular groove to the peroneal tubercle, where the PL and PB share a common tendon sheath. Zone 2 tendon sheath runs from the peroneal tubercle to the cuboid tunnel, here the PL and PB have separate sheaths. Zone 3 tendon sheath refers to the PL sheath at the sole (Fig. 19.8).⁶⁹

Once the portals are made, the surgeon has to adopt a triangulation technique positioning the instruments at an angle of nearly 180 degree.⁷¹ The inspection of the tendons is realized with the aim to look for synovitis, tears, or subluxation/dislocation (Fig. 19.9). Also, the superior peroneal retinaculum can be assessed. The two standard portals permit zone 1 examination, while zone 2 needs a separately approach for each tendon, as well as zone 3, with the help of accessory portals.⁶⁹

Depending on the diagnosis, it is possible to realize an endoscopic synovectomy, resection of the hypertrophic PB muscle, and superior peroneal retinaculum reconstruction. In cases of longitudinal tears, resection of the scar tissue or the injured area is carried out. Tendon repair is an option at the retromalleolar area, with the help of an arthroscopic grasper and pusher; if the rupture is too long or too distal, it could be repaired through a mini-open approach.⁶⁹ After the repair, a direct or indirect groove-deepening procedure is performed. In our experience, the indirect groove deepening described by Shawen and Anderson is the most reliable and effective, which involves hollowing the bone beneath the posterior cortex of the fibula by inserting a drill in the apex of the fibula, then collapsing the floor of the fibular groove.⁶⁷

Aftercare

The postoperative management is dictated by the intraoperative diagnosis. In general, a compression bandage is recommended for the first 24 hours, then an adhesive bandage in a removable brace to allow early passive range of motion and preventing adhesions.^{69,67}

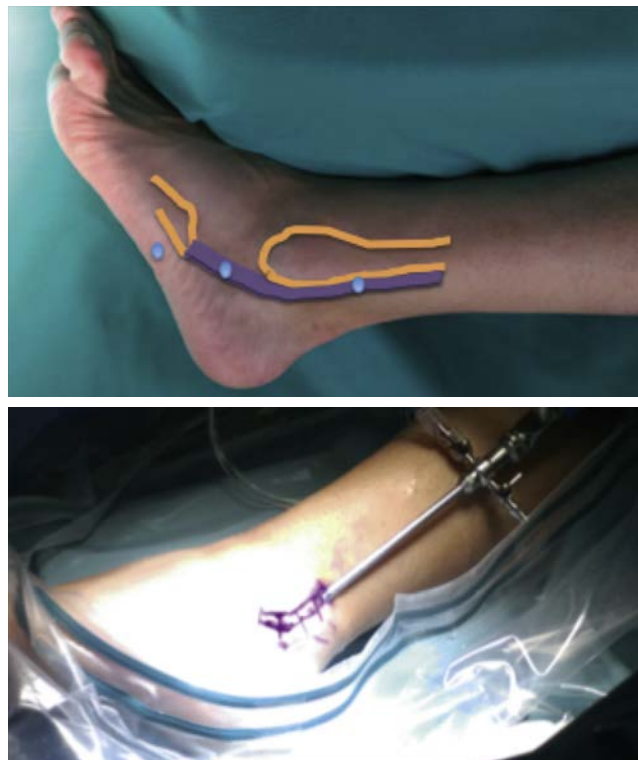


Fig. 19.7 Endoscopic peroneal tendons portals.

Weight bearing is allowed as tolerated, usually after 3 weeks.⁷² At the fourth week, patients can return to nonimpact sports, and impact sports are allowed at 6 to 8 weeks after surgery.⁶⁷

Results

Peroneal tendoscopy allows anatomic evaluation of the tendons and a dynamic assessment. It can be used as a diagnostic procedure and to treat the pathologies previously described. In the literature, the available studies are level IV and V of evidence. Panchbhavi and Trevino in 2003 performed tendoscopy as a diagnostic procedure, finding as a cause of pain a peroneus quartus tendon and a low-lying muscle belly attached to PB, which were treated by a mini-open procedure, after which patient's symptoms resolved.⁷² Scholten and van Dijk treated 23 patients with a minimum follow-up of 2 years, with diverse diagnoses (recurrent peroneal tendon dislocation, tenosynovitis, and longitudinal tears of the PB). None of the patients had complications or recurrence of the symptoms.⁵⁷ Vega et al. treated 52 patients with a follow-up of 1 year, with different indications for tendoscopy, such as peroneal adhesions, tendon rupture, tenosynovitis, and recurrent peroneal tendon subluxation. They reported a complete relief of symptoms in patients with tendon tears in 62.5%, partial relief in 25%, and the final 12.5% had no change. Additionally, excellent results have been reported in patients who were treated with endoscopic groove deepening.⁷⁰

Evidence-Based Recommendations

The levels IV and V studies on peroneal tendoscopy have reported good to excellent outcomes in most patients, with few complications. The indications accepted in the literature for peroneal tendoscopy include subluxation or dislocation

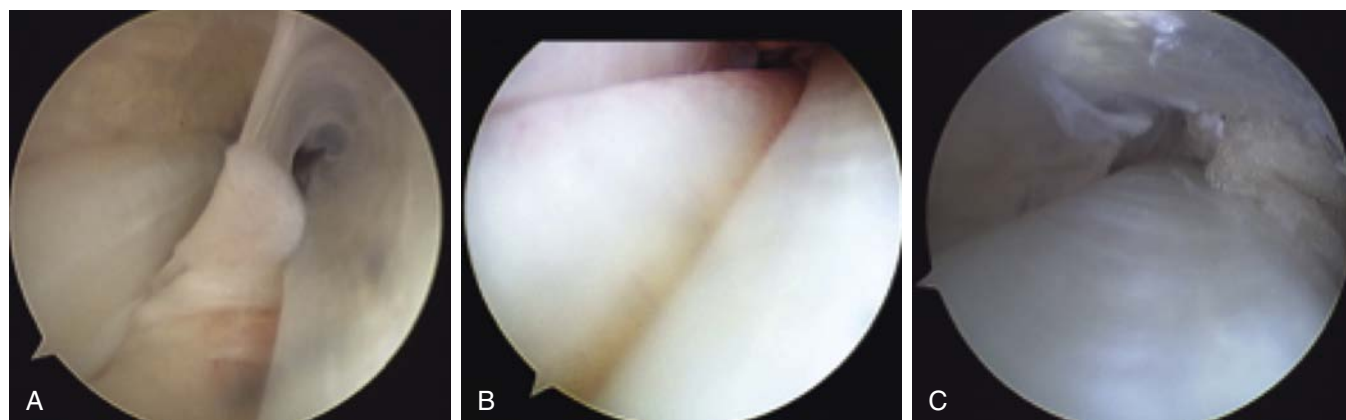


Fig. 19.8 Peroneal tendoscopy (A). Zone 1 (B). Zone 2 (C). Zone 3.



Fig. 19.9 Peroneal Brevis Rupture.

of the peroneal tendons, tenosynovitis, snapping, and partial tears that require debridement. Because of the level of evidence of the studies available for the peroneal tendoscopy, on a review of the literature performed in 2014 by Cychosz and colleagues,³³ they assigned a grade C recommendation for the intervention.³⁴

In our experience, peroneal tendoscopy is a powerful tool to assess and diagnose patients with unclear diagnosis of peroneal pathology. This is particularly true when some other procedures are going to be performed at the same time, such as ankle instability reconstruction, cavus foot surgery, etc., in order to plan incisions the best way.

OTHER TENDOSCOPIES

Flexor Hallucis Longus Tendonoscopy

The flexor hallucis longus (FHL) tendon originates at the inferior two-thirds of the posterior surface of the fibula and

lower part of the interosseous membrane. It extends to the posterior part of the ankle and courses from the fibro-osseous tunnel posterior to the talus, under the roof of the sustentaculum tali. The tendon is bound to the flexor digitorum longus at the Knot of Henry. Distally, FHL passes between the sesamoid bones and inserts into the plantar surface of base of distal phalanx of hallux.^{3,73}

Lui in 2009 described the surgical technique for the FHL tendonoscopy.⁷³ The course of the tendon is divided in three zones:

- **Zone 1:** Behind the ankle joint, from the musculotendinous junction to the entrance of the tunnel underneath the sustentaculum tali. Usually, the FHL at this zone is assessed with a posterior ankle endoscopy, described by van Dijk.⁷⁴
 - **Portals:** The posteromedial portal is located at the intersection point between a line joining the sustentaculum tali and the inferior border of the medial cuneiform and first metatarsal joint with the medial edge of the Achilles tendon. The posterolateral portal is located at the same level in the lateral edge of the Achilles tendon.
 - **Procedure:** The fatty tissue on the Kager's triangle over the FHL tendon is resected with a shaver. Here it is possible to examine the musculotendinous junction of the FHL (Fig. 19.10).
- **Zone 2:** Is located between the tunnel underneath the sustentaculum tali to the Knot of Henry.
 - **Portals:** Once the posteromedial portal is made. Under direct vision, a Wissinger rod is introduced to the tendon sheath through the tunnel. The plantar fascia is penetrated distal to the level of the navicular tubercle for the plantar portal. It may reduce the risk of injury to the medial plantar nerve.
 - **Procedure:** The FHL tendon in zone 2 can be assessed during his trajectory through the tunnel until the Knot of Henry. The portals can be interchanged to do a better examination of the tendon.
- **Zone 3:** It starts at the Henry's Knot and ends at the insertion of the tendon at the base of the distal phalanx of the big toe.



Fig. 19.10 Flexor hallucis longus Zone 1 tendoscopy.

- **Portals:** Using the plantar portal previously described for the assessment of zone 2, the plantar toe portal is created close to the insertion of the FHL at the base of the distal phalanx of hallux.
- **Procedure:** The tendon can be assessed with a 2.7-mm 30-degree arthroscope (Fig. 19.11).

The FHL tendon is susceptible to ruptures, laceration, longitudinal splits, stenosing tenosynovitis, and pigmented villonodular synovitis.⁷⁵

Evidence-Based Recommendations

The evidence-based data in the current literature to support the use of FHL tendoscopy are level of evidence IV and V. Most of the authors reported good to excellent outcomes, with complications rates from 0% to 25%. The indications found in the literature available include stenosing tenosynovitis, tenosynovitis, and also harvesting the FHL for use in the augmentation of the Achilles tendon.⁷⁶ On the review of the literature performed by Cychosz et al.³³ they assigned a grade C recommendation for the endoscopic release of the FHL.³⁴

Tibialis Anterior Tendoscopy

The tibialis anterior (TA) muscle originates in the upper two-thirds of the outside surface of the tibia and inserts into the first metatarsal and the medial cuneiform.³ Tibialis anterior tenosynovitis is not frequent, and the majority of cases alleviate with conservative management. For that reason in the literature, only two studies exist that describe TA tendoscopy: the first one is a case report for the endoscopic technique debridement of the TA tendon, by Maquirriain and colleagues;⁷⁷ the other is the one used for stage II flatfoot patients where TA tendoscopy was used to transfer the medial half of the TA tendon.⁴⁶

- **Portals:** The patient is placed in a supine position. The proximal portal is the first made, and is located 8 cm above the ankle joint line, following the TA tendon. Under direct vision, the distal portal close to the joint line can be made,



Fig. 19.11 Flexor hallucis longus Zone 3 tendoscopy.

as described by Maquirriain et al.⁷⁷ Lui in 2007 described a different distal portal for the TA tendoscopy, located close to the insertion of anterior tibial tendon.⁴⁶

Evidence-Based Recommendations

In the literature the two studies that described the TA tendoscopy are case reports, corresponding to a level V of evidence. With only these studies available in the review of the literature performed by Cychosz and colleagues,³³ they assigned a grade I recommendation, because there is a paucity of evidence-based literature.³⁴

Extensor Tendoscopy

The extensor hallucis longus (EHL) muscle arises from the middle two-thirds of the anterior surface of the fibula, adjacent to the interosseous membrane. It is deep and lateral to the TA tendon and extends distally, reaching the inferior extensor retinaculum. The EHL tendon passes through its own fibrous tunnel and inserts on the dorsum of the base of the distal hallux phalanx. The extensor digitorum longus (EDL) muscle originates from the upper three-fourths of the anterior surface of the fibula, the upper part of the interosseous membrane, and the anterior intermuscular septum. It splits in four tendons over the lower part of the tibia, beneath the superior extensor retinaculum, which are restrained by the inferior extensor retinaculum and finally insert on dorsum of middle and distal phalanges of the lesser toes.^{3,78}

- **Portals:** The patient is placed in a supine position. The proximal portal is made 3–4 cm proximal to the anterior ankle joint line on the lateral edge of the EDL tendon, and the distal portal is located 3–4 cm distal to the ankle joint, also on the lateral edge of the EDL tendon. These portals vary according of the location of the pathology. It is important to be extra careful with the superficial peroneal nerve, which can be easily damaged. To avoid iatrogenic damage, Lui⁷⁸ suggested that the subcutaneous tissue should be bluntly

spread with a hemostat after the skin incision is made until the EDL tendon is seen. Then, the scope can be introduced to assess the EDL and EHL tendons.

Evidence-Based Recommendations

The evidence-based data in the literature to support the extensor tendoscopy are level of evidence V. There were four studies describing the surgical technique for the tendoscopy and used in patients with tenosynovitis, tendinopathy, fibrous adhesions, ganglions, and to assist in the repair of delayed tendon ruptures.^{78,79,80,81} Given the lack of evidence-based literature for extensor tendoscopy, in the review of the literature performed by Cychosz and colleagues³³ they could not make a recommendation on extensor tendoscopy (EHL and EDL), and assigned a grade I recommendation.³⁴

OTHER ARTHROSCOPIES

Metatarsophalangeal Joint of the Lesser Toes

The metatarsophalangeal joint (MTP) of the lesser toe is stabilized by the plantar plate that arises from proximal to the articular surface of the metatarsal head and inserts on the base of the proximal phalanx; collaterals ligaments (medial and lateral), composed of two bands: the phalangeal collateral ligament and the accessory collateral ligament; and the joint capsule and traversing tendons.^{82,83}

Indications

Metatarsophalangeal Synovitis

This pathology is commonly caused by metabolic diseases, such as arthritis, gout; inflammatory diseases, such as rheumatoid arthritis; and abnormal mechanical stress. They present with pain and swelling, in general treated conservatively, but in cases with persistent symptoms, a synovectomy can be indicated.⁸⁴

Metatarsophalangeal Instability

In claw toe deformities, the key deformity is the hyperextension of the MTP joint. The plantar plate is attenuated on the metatarsal neck, leading to dorso-distal subluxation. The synovium ruptures at its proximal attachment resulting in the MTP joint dislocation.^{85,86} When nonsurgical management fails, surgical correction with soft tissues (e.g., flexor to extensor tendon transfer)⁸⁷ and bony procedures (e.g., Weil osteotomy, excision arthroplasty and arthrodesis) have been described, but secondary stiffness is also frequent.

The plantar plate repair should decrease toe stiffness, can be performed open, but has also been described arthroscopically-assisted.^{88,89,90}

Freiberg Disease

Originally described in 1914, Freiberg reports a series of cases with a similar “infarction” pattern of the metatarsal head.⁹¹ Smillie divided the macroscopic progression of Freiberg disease into five stages.⁹² Patients frequently present with pain localized to the involved metatarsal head, and report a feeling that they are walking on something hard.

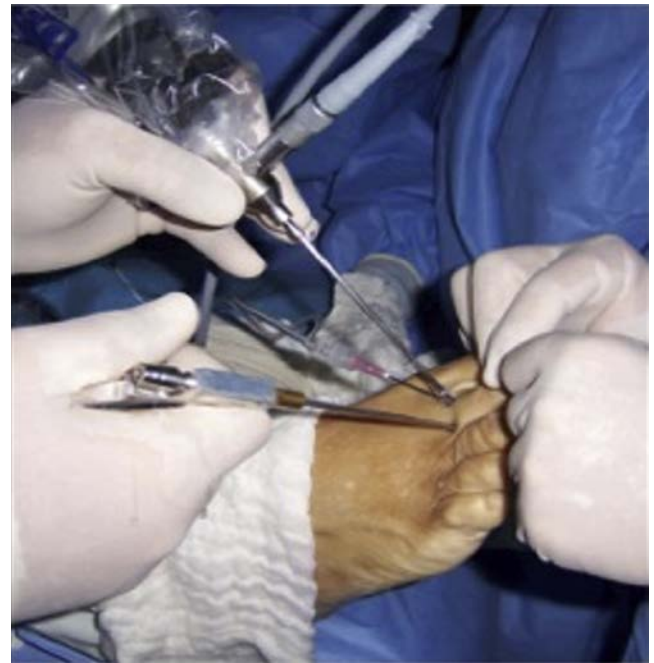


Fig. 19.12 Metatarsophalangeal lesser toe joint arthroscopic portals.

The management of this patient is initially conservative; the goal of the treatment is to alleviate the symptoms and minimize bone deformity. If nonsurgical management fails and symptoms persist or the disease advances, surgical treatment is indicated.⁹³ It varies depending on the stage. Carro and colleagues proposed a pattern of arthroscopic management: the arthroscopic removal of loose body and debridement are recommended in early stages, and an arthroscopic Keller procedure is reserved as salvage procedure in the later stages (stages IV and V).⁹⁴ el-Tayeby described in 1998 an interposition arthroplasty, using the tendon of the extensor digitorum brevis (EDB) to resurface and act as a joint spacer.^{95,96}

Operative Technique

The patient may be placed in a supine position, with a tourniquet around the upper thigh.

- Instruments required:**

A small-diameter short scope (1.9 mm with an inclination angle of 30 degrees) is used. The gravity-driven inflow is always adequate. Instrumented traction is not used routinely.

- Portals :**

The dorsomedial and dorsolateral portals are at the level of MTP joint line medial and lateral to the extensor digitorum longus (EDL) tendon. There is risk to injure the dorsal digital branches of superficial peroneal nerve (Fig. 19.12).^{84,90,96}

- Procedure :**

The portals are interchangeable as visualization and instrumentation portals. The articular cartilage of the proximal phalanx and metatarsal head, collateral ligaments, and dorsal capsule are examined; also the plantar plate can be probed for any tear (Fig. 19.13).

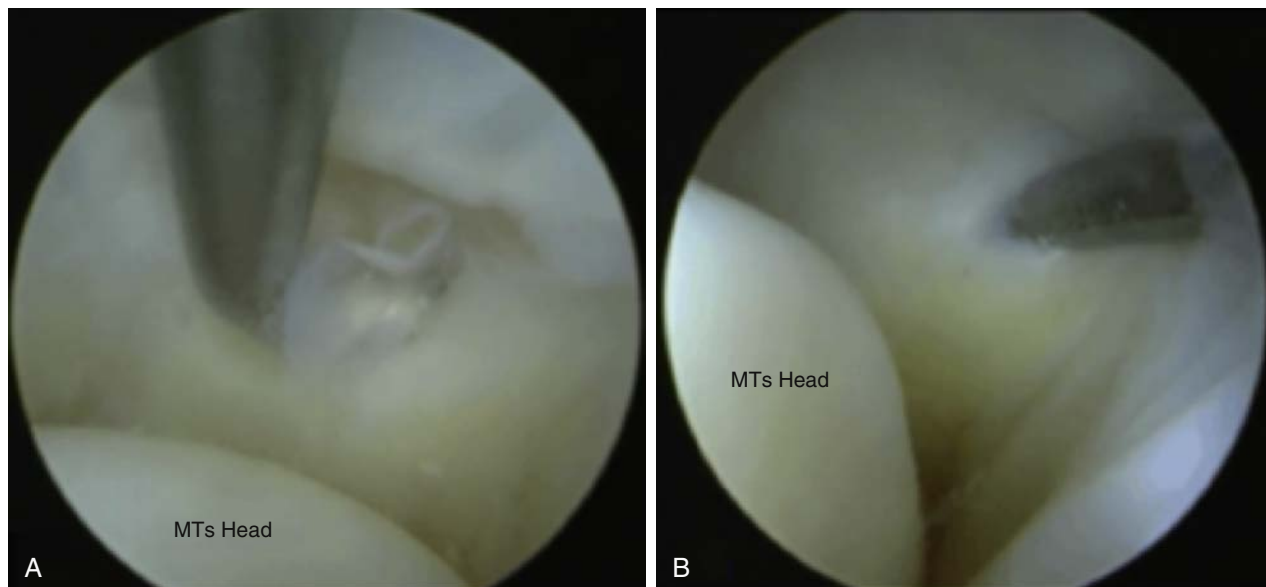


Fig. 19.13 Plantar plate injury (A). Transversal (B) “7” shape.

In MTP synovitis, synovectomy of the dorsal, medial, and lateral gutters can be performed with a shaver. However for the plantar gutter, additional manual traction of the joint is required.⁸⁴

Once MTP joint assessment is performed, instability has to be evaluated. If found, the arthroscopic-assisted plantar plate tenodesis can be performed. First, the dorsal capsule is stripped from the metatarsal neck, the sutures are passed through the lateral and medial part of the plantar plate, and the suture is retrieved through a proximal wound at the diaphysis of the metatarsal. Then, it is secured to the EDL, reducing the MTP joint.^{90,97,98} Using the same portals, the arthroscopic interpositional arthroplasty is performed in cases of Freiberg’s disease. The EDB is identified, cut proximally, and retrieved through the dorsolateral portal. It is rolled into a ball and inserted into the joint, under arthroscopic guidance.⁹⁶

Lisfranc (Tarsometatarsal) Joint

Tarsometatarsal joints can be divided in three columns. The lateral column consists of the fourth and fifth metatarsocuboid joints. The medial column is the first metatarsocuneiform joint, while the middle column is composed of the second and third metatarsocuneiform joints. The stability is afforded through a combination of ligaments and bony architecture (roman arch configuration).⁹⁹

Indications

First Tarsometatarsal Instability

Hypermobility of the medial column can be associated with hallux valgus, transfer metatarsalgia, and arthritis of the second TMTJ. In patients with symptomatic hypermobility of the joint who do not respond to conservative treatment, arthrodesis is indicated. Traditionally, lapidus procedure is performed by open means, but it has some problems, such as prolonged healing, high nonunion rate, and the tendency for dorsal angulation of the first metatarsal. In 2005, Lui et al. described an arthroscopic

lapidus, trying to achieve less bone removal and better control of arthrodesis position, and decrease the malunion rate.^{84,100}

Operative Technique

The patient may be placed in supine position, with a tourniquet around the upper thigh.

- **Instruments required:**

A small-diameter short scope (2.7 mm with an inclination angle of 30 degrees) is used. The setting is similar to the one already described for the Achilles tendoscopy.

- **Portals:**

Two portals are established at the plantar medial and dorso-medial corners of the first TMTJ (Fig. 19.14).

- **Procedure:**

Once the portals are identified, the position can be checked under fluoroscopy if needed. Then, the first TMTJ is assessed, the articular cartilage is denuded exposing the subcondral bone, which is then microfractured.

The intermetatarsal angle is closed up manually and the first metatarsal plantarflexed, then the fixation is performed with cannulated screws.^{84,100}

Post-traumatic Arthritis

After injuries to the TMTJ, post-traumatic arthritis is the most common problem, but not all patients are symptomatic. The initial treatment in symptomatic patients is conservative; when the symptoms persists, arthrodesis of the painful tarsometatarsal joints is the management of choice. Lui in 2007 described six portals to approach the five tarsometatarsal articulations. The choice of the portals depends on which columns are included in the fusion.^{84,101}

Operative Technique

The patient may be placed in a supine position, with a tourniquet around the upper thigh.

- **Instruments required:**



Fig. 19.14 First tarsometatarsal joint arthroscopic portals.

- A small-diameter short scope (2.7 mm with an inclination angle of 30 degrees) is used. The setting is similar to the one already described for the Achilles tendoscopy.
- Medial portal is located at the plantar medial aspect of the first metatarso-cuneiform joint.
- P1-2 portal is located at the junction point between medial cuneiform and first and second metatarsal bones. The proximal P1-2 portal is located at the junction point between the second metatarsal, medial, and intermediate cuneiform bones.
- P2-3 portal is located at the junction point between the second metatarsal and intermediate and lateral cuneiform bones. The distal P2-3 portal is located at the junction between the second and third metatarsal with the lateral cuneiform bone.
- P3-4 portal is located at the junction point between lateral cuneiform and cuboid and third and fourth metatarsal bones.
- P4-5 portal is located between the proximal articular surfaces of the fourth and fifth metatarsal.
- Lateral portal is located at the lateral corner of the fifth metatarsal-cuboid joint (Fig. 19.15).
- *Procedure:*

Once the origin of the pain is identified, the portals used depends on which columns need to be treated. The medial portal and the P1-2 portal permit to assess the first TMTJ. The second TMTJ can be approached using the proximal P1-2 and the P2-3 portals. To evaluate the third TMTJ, the distal P2-3 and the P3-4 portals are necessary. The fourth TMTJ could be assessed using the P3-4 and P4-5 portals. Finally, the fifth TMTJ can



Fig. 19.15 Lisfranc joint arthroscopic portals.

be approached through the P4-5 and lateral portals. The P3-4, P4-5, and lateral portals are interchangeable, because the third, fourth, and fifth TMTJ share a common synovial lining and joint capsule.

The fusion surfaces preparation technique is similar to the already described to the arthroscopic Lapidus. Then, the joints are reduced into the desired position and transfixed with cannulated screws.^{84,101}

Chopart (Transverse Tarsal) Joint

Calcaneocuboid (CC) joint functions as a unit with the talonavicular (TN) joint in the formation of the transverse mid-tarsal joint. It is supported plantarly by the short and long plantar ligaments and the PL. The CC ligaments provide dorsal support. The TN joint is supported primarily by the dorsal TN, deltoid, and spring ligaments.¹⁰²

The Chopart joint helps to allow the foot to transition from a flexible structure when the hindfoot is in valgus during heel strike to a rigid structure when the hindfoot is in varus during toe-off.

Arthroscopy can be used as a diagnostic tool or to treat patients with joint arthritis; these can be post-traumatic, a sequelae of pediatric foot deformities, or secondary to rheumatoid arthritis. The arthroscopic arthrodesis can improve intra-articular visualization, minimize bone removal, and fusion surface preparation. Theoretically, this can reduce the risk for pseudoarthrosis and nonunion.^{84,102}

Operative Technique

The patient may be placed in supine position, with a tourniquet around the upper thigh.

- *Instruments required:*
- A small-diameter short scope (2.7 mm with an inclination angle of 30 degrees) is used. The setting is similar to the one already described for the Achilles tendoscopy.



Fig. 19.16 Calcaneo-cuboid joint arthroscopic portals.

There are four mid-tarsal portals. The lateral portal is located at the plantar lateral corner of the CC joint; the structures at risk are the peroneal tendons and the sural nerve. The dorsolateral portal requires a fluoroscopic guidance, and is located over the space between the TN and CC joints; the lateral branch of the superficial peroneal nerve and the lateral terminal branch of the deep peroneal nerves are at risk (Fig. 19.16).

The medial portal is located at the medial side of the TN joint, dorsal to the PTT. Finally, the dorsomedial portal is located at the midpoint between the medial and dorsolateral portals; during the creation of this portal, the intermediate cutaneous branch of superficial peroneal nerve and EHL tendon are at risk (Fig. 19.17).

- **Procedure:**

The lateral and medial portals permit the assessment of the CC and TN joints, respectively. The most important portal of the midtarsal arthroscopy is the dorsolateral, because through it is possible to reach the medial aspect of the CC joint, lateral and plantar aspects of the TN joint, even the anterior subtalar joint, the junction between the talus, calcaneus, navicular, and cuboid bones.



Fig. 19.17 Talonavicular joint arthroscopic portals.

It can be used for arthroscopic resection of calcaneonavicular coalition, symptomatic nonunion of the anterior calcaneus process, and to fuse the CC and TN joints. The fusion surface preparation, joint reduction, and fixation technique are similar to arthroscopic Lapidus procedure.^{84,102}

CONCLUSIONS

Endoscopic procedures in foot and ankle have been proven extremely useful and with increasing indications. For the less common indications, as the ones described in this chapter, we recommend to be enthusiastic without forgetting the goal of arthroscopy, which is intended to be a minimally invasive procedure.

For this reason, the less experienced surgeon may conclude that instead of using arthroscopic vision to perform a cheilectomy in a hallux rigidus, it could be easier to use a minimally invasive technique. The same principle could be true for Haglund deformity, midfoot arthroscopy, and some other procedures described in literature with just few cases.

So even though we encourage everyone to use endoscopic assistance for diagnosis, assistance in fracture treatment, arthrodesis, tendoscopies, etc., we think that there are limits depending on personal experience.

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Lesser Toe Disorders

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Metatarsalgia in the athlete can be a debilitating disorder that can lead to loss of function. Forefoot disorders encompass lesser toe abnormalities such as claw toes, hammertoes, mallet toes, and hard and soft corns. More proximally, problems can include intractable plantar keratosis (IPK), bunionettes, neuromas, and metatarsophalangeal (MTP) joint capsulitis and instability.

For the athlete, repetitive activities can lead to repeated stress reactions in soft tissues, bones, and joints. Abrasions and repeated trauma over bony prominences can lead to callus formation and bursitis.

Ideally, avoiding the development of problems through the use of good footwear, proper training practices, and education should be the goal. Many of these problems may develop despite prophylactic care and thus require the intervention of the orthopedic surgeon either conservatively or surgically. Often, nonsurgical treatment is successful, leading to a rapid return to athletic activity.

The complaint of forefoot pain can be frustrating for the athlete and the physician. The pain must be differentiated in order to make a correct diagnosis. The accompanying algorithm (Fig. 20.1) may prove useful in determining the specific forefoot diagnosis when a patient complains of metatarsalgia. Most important is the exact location of pain. In addition, the physician should ask the following questions: Which specific

activities increase symptoms? Which activities alleviate discomfort? Is the pain dorsal or plantar, medial or lateral? Is there an associated neuritis symptom with the pain? Are enlarged exostoses or prominences associated with pain, swelling, or inflammation?

When a patient complains of metatarsalgia, the initial concern on physical examination is the presence of an associated callosity. This can be seen laterally over the fifth metatarsal head with a bunionette formation. It can be localized to the plantar metatarsal region with an IPK. A callosity may develop over the dorsal distal interphalangeal (DIP) joint (a mallet toe) or the dorsal proximal interphalangeal (PIP) joint (hammertoe). On occasion a patient may complain of a callosity both overlying the PIP joint and beneath the associated metatarsal head. With a concomitant contracture of this toe, the diagnosis of a claw toe is made based on these clinical findings.

Development of a callus between two toes (a soft corn) or over the lateral aspect of the fifth toe (a hard corn) can be extremely painful.

When a patient complains of metatarsalgia, but there is no callosity present, the patient should be carefully examined for nerve-related symptoms. When such a scenario is present (along with other specific symptoms), the diagnosis of an interdigital neuroma can be made. When neuritic symptoms are not present, but symptomatic pain is still localized to the forefoot,

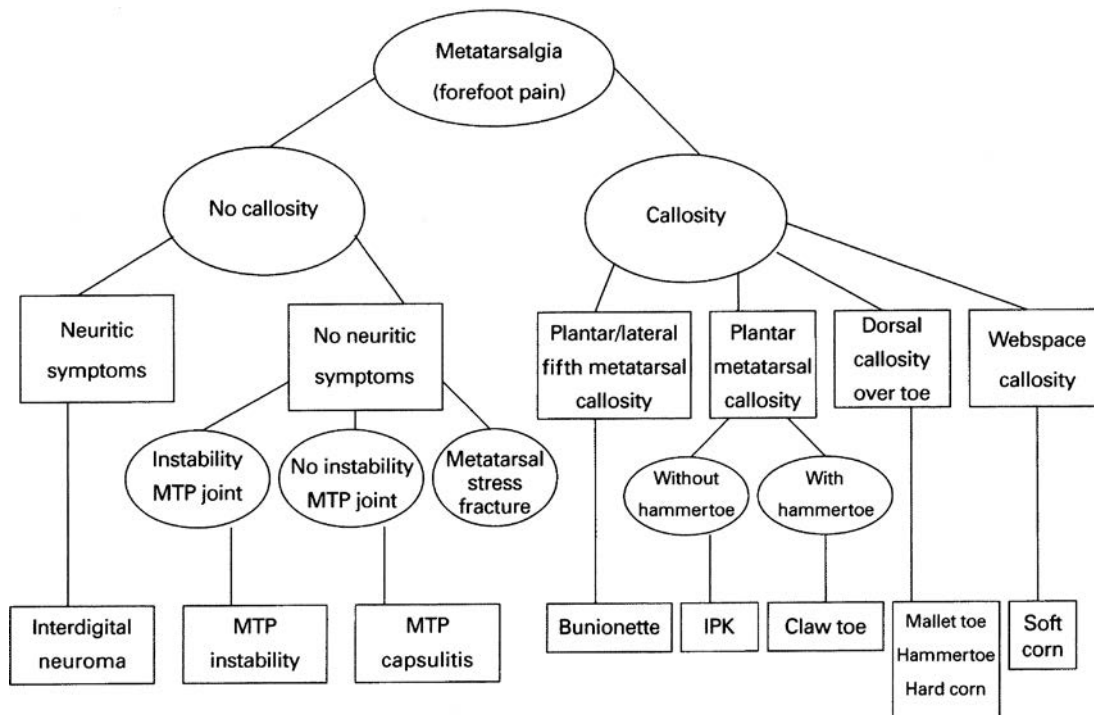


Fig. 20.1 Algorithm. *IPK*, Intractable plantar keratosis; *MTP*, metatarsophalangeal. (From Coughlin, MJ. Common causes of pain in the forefoot in adults. *J Bone Joint Surg Br* 2000;82:781-790)

metatarsophalangeal joint capsulitis and/or instability should be considered. The presence of a positive drawer sign (dorsal plantar instability) or malalignment of the involved toe at the metatarsophalangeal joint aids in confirming the diagnosis of second toe instability. Although this algorithm is not all-inclusive, and much more goes into the specific diagnostic process than this flow sheet allows, it does offer a method of approaching the athlete with metatarsalgia. Sometimes symptoms overlap, frequently symptoms are vague—and repeated evaluation and clinical and radiographic examination are necessary to confirm a diagnosis. The cooperation of patients in defining their symptomatic complaints, and in defining their problem through varying their athletic activity, is highly important. Likewise, patient cooperation in modifying activities when conservative management is attempted is a critical factor in any successful treatment. When surgery is performed, patient cooperation in allowing adequate healing to occur before resuming athletic activity is instrumental not only in the recovery process but also in the avoidance of other associated problems or complications.

PEARL: LOCATION OF FOOT PAIN

When evaluating forefoot pain, it can often be difficult to localize the exact location of a patient's pain in order to make a correct diagnosis. Often, the physical examination is inconclusive, especially when the patient presents to clinic when asymptomatic. This is especially common in athletes whose complaints are often activity related. To identify the location of pain, instruct the patient to repeat the offending activity, recreating the patient's symptoms. The patient is then instructed to mark the spot with a Sharpie marker. This "spot" will stay with the patient for the next clinical examination.



Fig. 20.2 Bunionette with enlarged bursa. (From Mann RA, Coughlin MJ. Keratotic disorders of the plantar skin. In: *Surgery of the Foot and Ankle*. St Louis, CV Mosby; 1993:443.)

BUNIONETTES

The development of inflammation, an enlarged bursa, or a callus over a prominent fifth metatarsal head may lead a physician to diagnose a bunionette (Fig. 20.2). Just as bunions can present with differing magnitude and different characteristics, so too can a bunionette.¹ A bunionette may appear radiographically as an enlarged fifth metatarsal head (type I). A flare in the metaphysis may cause bowing of the fifth metatarsal (type II), leading to symptoms, or a widened 4–5 intermetatarsal angle (type III) characteristic of a splayfoot may lead to pain and callus formation (Fig. 20.3).

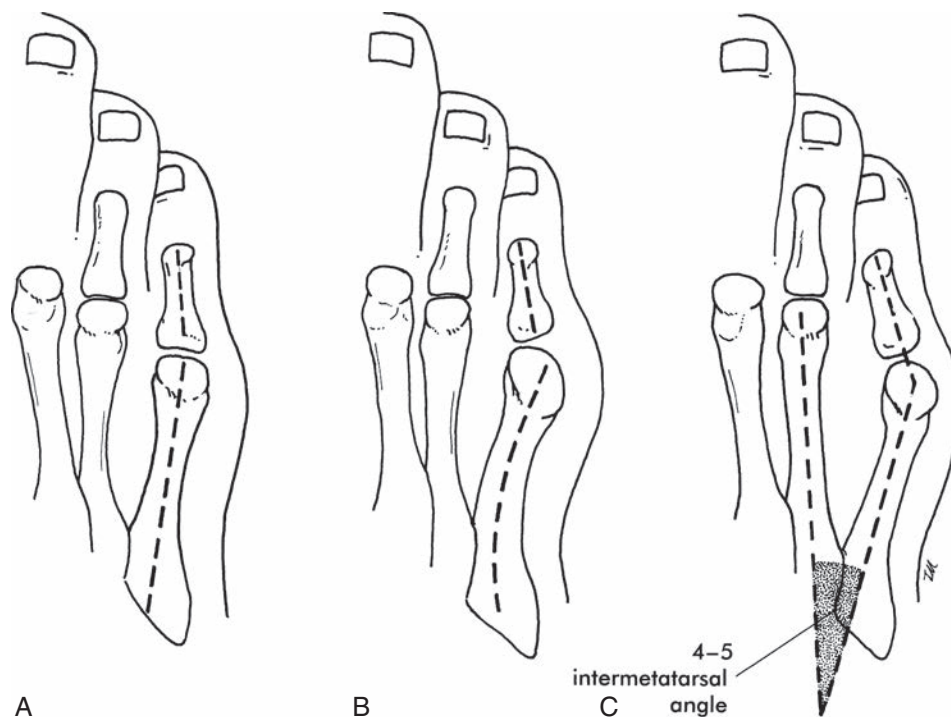


Fig. 20.3 (A) Bunionette with enlarged fifth metatarsal head. (B) Bunionette with bowing of the metaphysis. (C) Bunionette with enlarged 4–5 intermetatarsal angle.

Initially an athlete may complain of pain directly lateral over the fifth metatarsal head, but the examiner should be aware of plantar symptoms as well. Neuritic symptoms involving the fifth toe may occur due to pressure over the lateral digital nerve to the fifth toe. Complaints of pain, inflammation, blistering, ulceration, or infection may be noted by the athlete.

On physical examination, significant callus formation may be observed on the lateral, plantar, or lateral-plantar position overlying the fifth metatarsal head. Any pronation of the longitudinal arch should be noted, as should any restriction in hindfoot motion.

Radiographic evaluation may demonstrate an enlarged metatarsal head, outflaring of the fifth metatarsal metaphysis, or widening of the 4–5 intermetatarsal angle. Widening of the 4–5 intermetatarsal angle is the most common. Abduction of the fifth toe in relation to the fifth metatarsal head may also be demonstrated.

Conservative Treatment

Early treatment involves attempting to relieve pressure on the underlying bony prominence. Stretching of shoes or obtaining shoes with a soft upper that is more forgiving will relieve overlying pressure. Seams or stitching directly over the bunionette should be avoided. Moleskin applied to a blister may promote healing and protect the area while athletes continue their activities. Altering running and/or training activity may also diminish symptoms. Nonimpact activities such as stationary cycles or swimming can be integrated into the training program. A reduction in total miles per day/week may be required. Trimming the callus may significantly relieve symptoms. Physicians may teach their patients how to pare the callus appropriately. The callus is shaved in thin layers with the scalpel parallel to the toe. A pumice stone may also be used to pare down the callus. A pumice stone is safer and often more acceptable to patients for home use than using a scalpel.

When athletic activity is significantly impaired after conservative efforts, surgical intervention may be considered (see [Case Study 20.1](#)). The type of osteotomy selected is dependent upon the location of the callosity, as specific osteotomies of the fifth metatarsal can be used to direct the metatarsal head in different directions. Surgical intervention in treating forefoot callosities should be tailored to the patient. Extensive soft-tissue stripping, unsecured osteotomies, and multiple metatarsal osteotomies should all be avoided in athletes. Although a surgical procedure may relieve the painful callosity, athletic performance of the patient may be diminished, and thus the procedure would be considered unsuccessful. The two surgical procedures presented here fulfill the requirements of less surgical exposure, employ internal fixation, and appear better suited to athletes.

CASE STUDY 20.1

A 30-year-old skier developed pain and swelling over the plantar lateral aspect of the fifth metatarsal head. An increased callosity was observed over the plantar lateral aspect of the bunionette. A painful inflamed bursa developed during the middle of ski season that was partially relieved by grinding down the inner aspect of the ski boot overlying the bunionette. Likewise, the area overlying the fifth metatarsal head was relieved in the athlete's everyday footwear by stretching the leather surface.

On physical examination, a normal neurologic and vascular examination was noted. Prominence of the fifth metatarsal head was characterized by a callosity both on the plantar and lateral aspects. Radiographic evaluation demonstrated an enlarged fifth metatarsal lateral condyle ([Fig. 20.4A](#)).

Conservative care, stretching of shoes, and padding were all recommended.

At the end of ski season, the patient requested surgical treatment due to continued symptoms. An oblique osteotomy was performed and fixed with screws. At 8 weeks following surgery, the osteotomy was healed and the patient began advancing gradually over a 2-month period. [Fig. 20.4B](#) shows the correction obtained. The patient skied the following season without symptoms.



Fig. 20.4 Case study 1. (A) Bunionette preoperative x-rays. (B) Follow-up x-rays demonstrating correction.

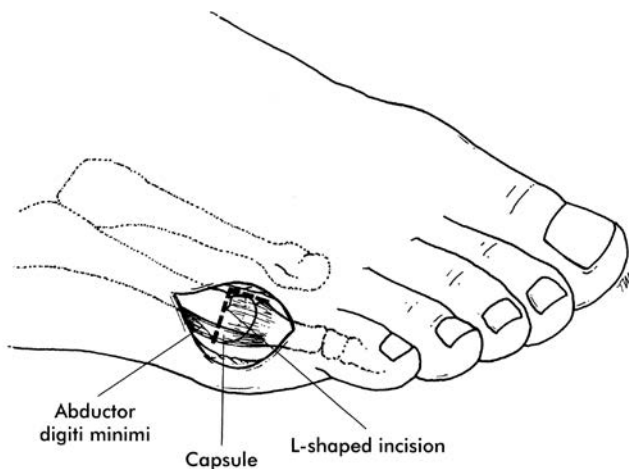


Fig. 20.5 An L-shaped capsular incision is used to approach the bunionette with dorsal and proximal capsular incisions.

Surgical Treatment

1. The foot is prepped and draped in the routine sterile fashion. An Esmarch bandage is used to exsanguinate the foot. The ankle is carefully padded and the Esmarch is used as a tourniquet.
2. A longitudinal lateral incision is centered directly over the bunionette, extending from the midproximal phalanx to 1 cm above the metatarsal head. Care is taken to protect the neurovascular bundles.
3. The metatarsophalangeal joint capsule is detached on the dorsal and proximal aspect and retracted plantarly, exposing the prominent lateral condyle (Fig. 20.5).
4. A sagittal saw is used to resect the lateral condyle in line with the diaphyseal shaft of the fifth metatarsal. (At this point, a decision must be made regarding the type of osteotomy to be performed. For a pure lateral callus, a chevron

osteotomy is performed. For a combined plantar lateral callus, a distal oblique osteotomy is performed.)

- 5A. *Chevron osteotomy*^{2,3}: A lateral to medial drill hole is placed in the center of the fifth metatarsal head, marking the apex of the chevron osteotomy. A 60-degree angled osteotomy based proximally is directed in a lateral to medial plane. The metatarsal head is translated medially and fixed with a percutaneous 0.045 K-wire (Fig. 20.6).
- 5B. *Distal oblique osteotomy*^{4,5}: After exposing the metatarsal head and metaphysis, an oblique osteotomy is performed from a distal lateral to proximal medial direction. The metatarsal head is displaced medially and slightly proximally and is allowed to “raise up” approximately 3 mm to decrease plantar pressure beneath the fifth metatarsal head. The osteotomy is fixed with a percutaneous 0.045 K-wire (Fig. 20.7).
- 5C. *Midshaft osteotomy*⁶: After a distal lateral eminence resection is performed, in the case of an increased 4–5 intermetatarsal angle, a diaphyseal midshaft osteotomy may be necessary to achieve correction. A subperiosteal dissection is performed at the midshaft of the fifth metatarsal with care to protect the distal capsular tissue and attachments. Care must also be taken to prevent overdissection of the metatarsal by dissecting no more than 50% of the circumference of the metatarsal shaft. This preserves as much medial-based blood supply as possible. The osteotomy may be performed, but prior to completing the oblique osteotomy, which generally runs from plantar-proximal to dorsal-distal, fixation is considered. Two screws are generally sufficient for fixation, and the more proximal of the two screws is drilled and placed. After completing the osteotomy, with a depth gauge placed in the hole, this drill hole acts as the center of rotation of the osteotomy and allows easier placement of the screw for definitive fixation after correction has been achieved.

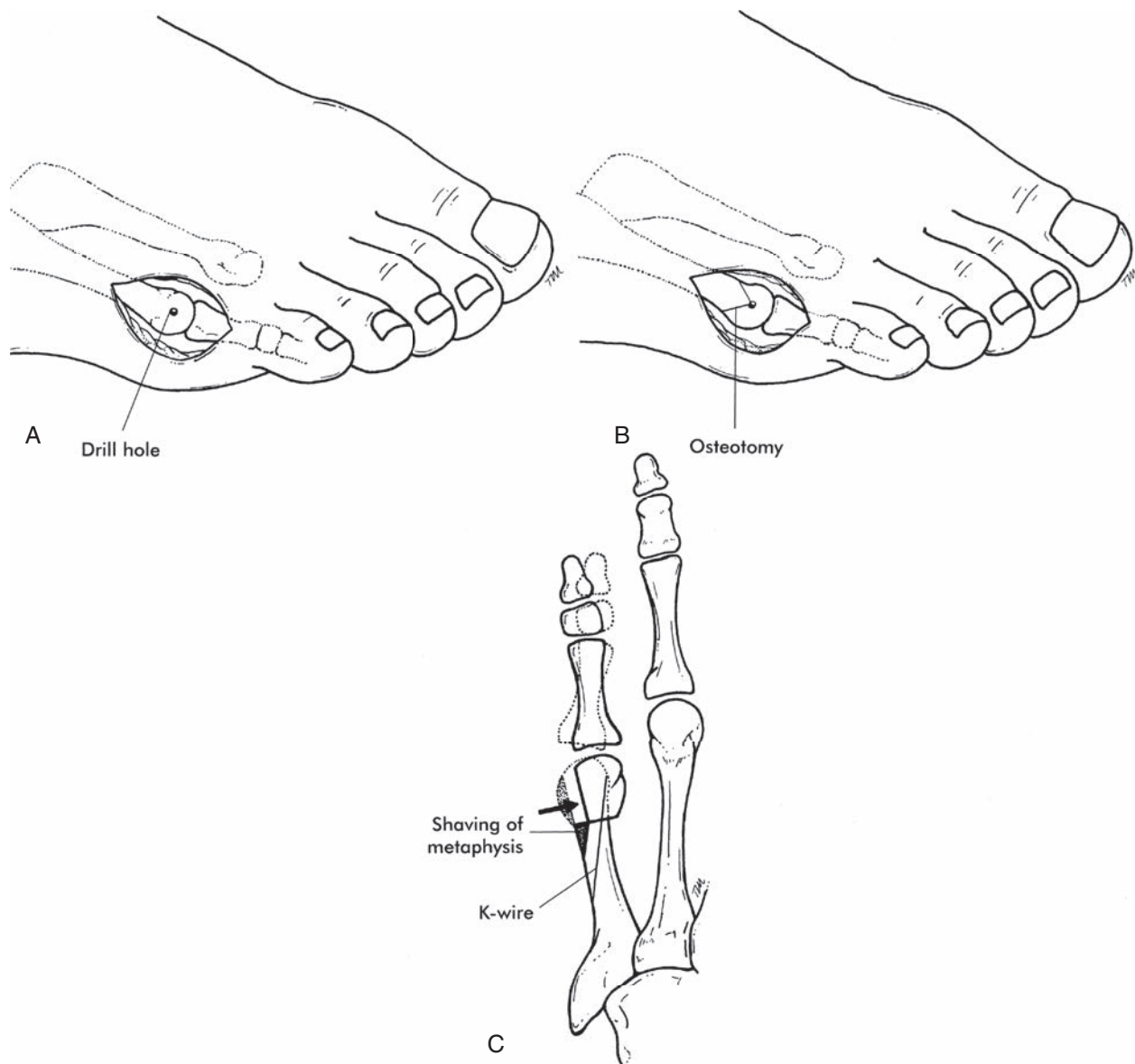


Fig. 20.6 (A) A drill hole is placed in the center of the metatarsal head and drilled in a lateral to medial direction. (B) A chevron-shaped osteotomy is based proximally with the apex at the drill hole. (C) Medial translation of the metatarsal head with K-wire fixation and shaving of the metaphyseal flare (*shaded area* denotes shaved bone in metaphysis).

6. Any remaining prominent metaphysis is shaved with the sagittal saw. A drill hole is placed in the dorsal proximal metaphysis, and the capsule is anchored with an interrupted suture. Remaining interrupted sutures are placed to reinforce the capsular repair (Fig. 20.8).
7. The skin is closed in a routine fashion. A gauze and tape dressing is applied and changed on a weekly basis. The patient is allowed to ambulate in a stiff postoperative shoe. Athletic activity is increased as swelling and pain diminish. Patients are allowed to heel weight bear as soon as pain and swelling permit. Radiographic confirmation of healing should be present before aggressive activity such as jogging, running, or jumping is considered. In general, a patient can return to nonimpact activities at 2 months and progress as tolerated thereafter.

INTRACTABLE PLANTAR KERATOSES

The development of a keratosis beneath one or more of the metatarsal heads is referred to as an IPK. A callus may be a localized discrete lesion or a diffuse keratotic buildup (Fig. 20.9). Callus formation in athletes is not uncommon and, if asymptomatic, rarely requires medical intervention. With significant buildup, painful symptoms may occur requiring evaluation and treatment.

A diffuse callus may be due to repetitive abrasion associated with athletic activity. It also may be associated with a long second metatarsal or long second and third metatarsal. A discrete callus may occur beneath a single metatarsal head.⁷ It is typically associated with an enlarged plantar metatarsal condyle. It is important to distinguish this from a wart (Fig. 20.10). Although warts

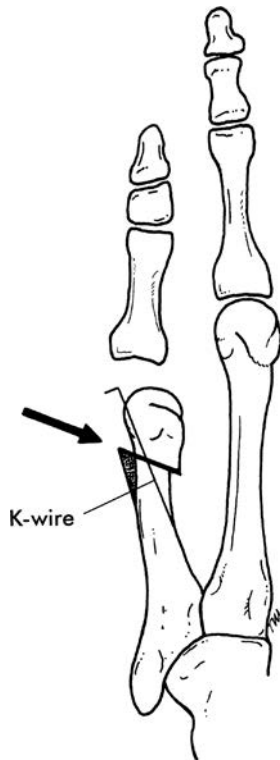


Fig. 20.7 Distal oblique osteotomy with K-wire fixation (shaded area denotes shaved bone in metaphysis).



Fig. 20.8 L-shaped capsular closure. The dorsal proximal corner may be fixed with a drill hole in the metaphysis to anchor the repair.

(plantar verrucae) typically are not found beneath a metatarsal head, on occasion they can occur in this region and thus must be differentiated from an IPK. Trimming of a wart will uncover end arterioles in the lesion characterized by punctuate hemorrhages. Evaluation of the athlete with an IPK involves determining the significance of the symptoms, length of duration, and association, if any, with specific athletic activity. A patient with minimal symptoms requires no treatment.

Radiographic evaluation entails weight-bearing films with markers to determine the exact location of the IPK (a long metatarsal may be associated with an IPK; likewise, a marker may be located directly beneath the plantar condyle of a metatarsal head).

PEARL: PRESSURE POINTS

A Harris mat is invaluable in identifying abnormal pressure points on the plantar surface of a patient's foot. The patient is instructed to walk in a normal manner, stepping on the Harris mat. The test is repeated with the contralateral foot. Abnormal pressures are illustrated as unusually dark regions and will aid in making the correct diagnosis and appropriate treatment.

Conservative Treatment

Conservative treatment revolves around paring the IPK and padding it to relieve the pressure (Fig. 20.11). A patient can be instructed to trim the lesion every 7 to 10 days, and this will significantly relieve discomfort. Placement of a metatarsal pad just proximal to the IPK can transfer pressure to the metatarsal diaphysis and relieve symptoms (see Case Study 20.2). Custom or prefabricated orthotic devices also can be a significant help in relieving symptoms. Athletes may consider altering their workout, or change sporting activities, or change duration or intensity of the workout.

CASE STUDY 20.2

A 50-year-old tennis player developed a painful callus beneath the second and third metatarsals. It was a diffusely thickened callus that began to limit his sports activities. On initial evaluation the diffuse callus was trimmed, and the patient instructed in how to care conservatively for the IPK. A pumice stone was used to pare the callus. The patient also obtained disposable scalpels to shave his thickened callosity. When he returned for further follow-up, radiographs demonstrated a long second and third metatarsal in relationship to the adjacent metatarsals. A soft pad was placed in his shoe just proximal to the callosity. Between shaving the callosity and padding it, symptoms were completely relieved, and he returned to full sports activities. Later, a soft orthotic device was fabricated to relieve pressure beneath the second and third metatarsals.

When all methods of conservative treatment have been exhausted, surgical intervention may be considered. Caution is advised in considering any metatarsal osteotomy in a high-level athlete. The possibility of delayed union, nonunion, or malunion can significantly impair athletic activity. The development of a transfer lesion beneath another metatarsal head is not uncommon. Multiple metatarsal osteotomies are to be discouraged.

Surgical Treatment; Partial Condylectomy⁸

1. The foot is prepped and draped in a routine sterile fashion. An Esmarch bandage is used to exsanguinate the foot. It is carefully padded at the ankle and used as a tourniquet.
2. A longitudinal incision is centered over the metatarsal head with a "hockey stick" extension distal into the adjacent interspace if needed. (The extensor tendon may be temporarily released to aid exposure and is repaired at the conclusion of the procedure.)
3. The MTP joint capsule is released and the toe is flexed to 90 degrees at the MTP joint.
4. A McGlamry elevator is used to release the plantar plate from the plantar metatarsal head in order to prevent damage during the resection.
5. An osteotome is used to remove 25% of the plantar condyle. Care is taken to avoid fracture to the metatarsal head (Fig. 20.12). The condyle is removed.
6. A 0.045 K-wire introduced at the MTP joint is driven distally out the tip of the toe. With the MTP joint reduced, the pin is driven in a retrograde fashion stabilizing the joint.

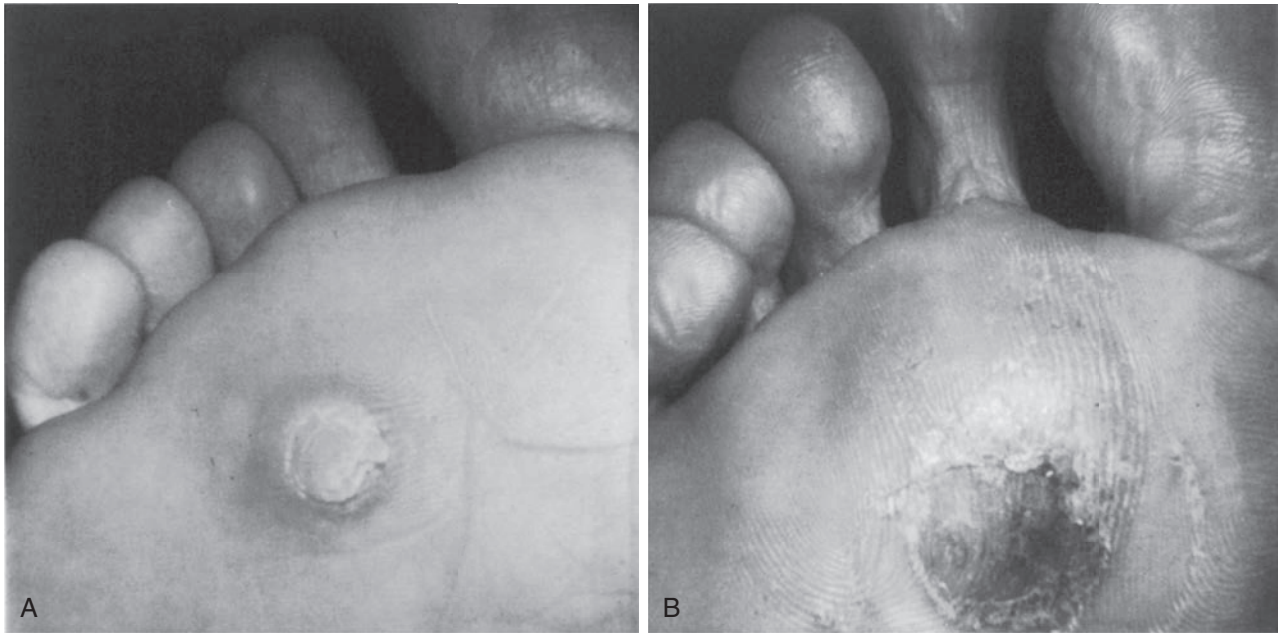


Fig. 20.9 (A) Discrete callus in a tennis player with an enlarged fibular condyle. (B) Diffuse callus in a runner. (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:86.)

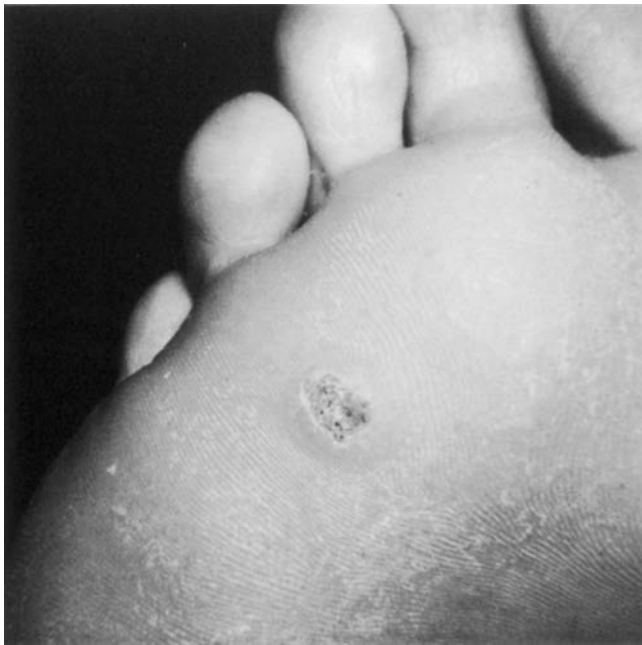


Fig. 20.10 A wart is characterized by punctate hemorrhages, which are obvious when the callus is trimmed. (From Mann RA, Coughlin MJ *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:86.)

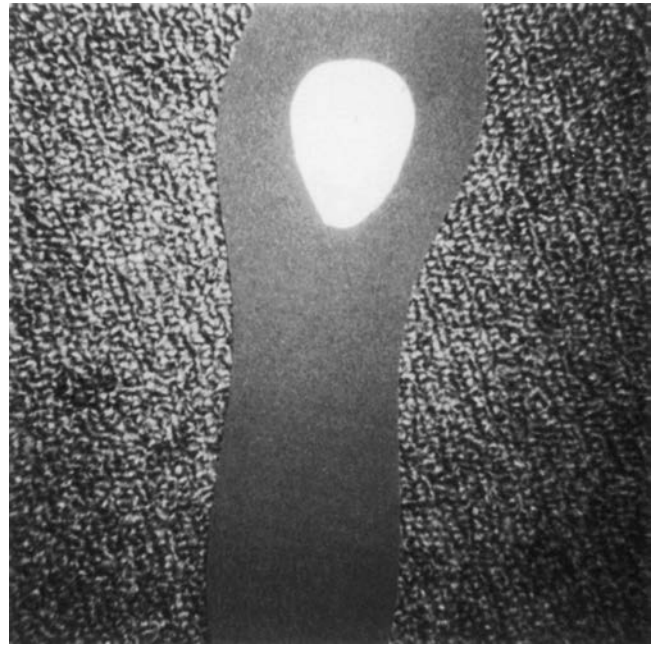


Fig. 20.11 Padding an intractable plantar keratosis often is successful treatment.

7. The extensor tendon (if released) is repaired. The skin is closed in a routine fashion.
 8. A gauze and tape dressing is applied and changed on a weekly basis. The patient is allowed to heel weight bear in a wooden-soled shoe.
 9. At 3 weeks the K-wire is removed.
- Athletic activity is permitted as swelling and pain decrease. The toe is protected for 6 weeks following surgery with taping

immobilization. In general, a patient can return to nonimpact activities at 1 month, limited impact activities such as jogging at 6 weeks, and may progress as tolerated.

Surgical Treatment: Metatarsal Osteotomy

1. The foot is prepped and draped in a routine sterile fashion. An Esmarch bandage is used to exsanguinate the foot. It is carefully padded at the ankle and used as a tourniquet.

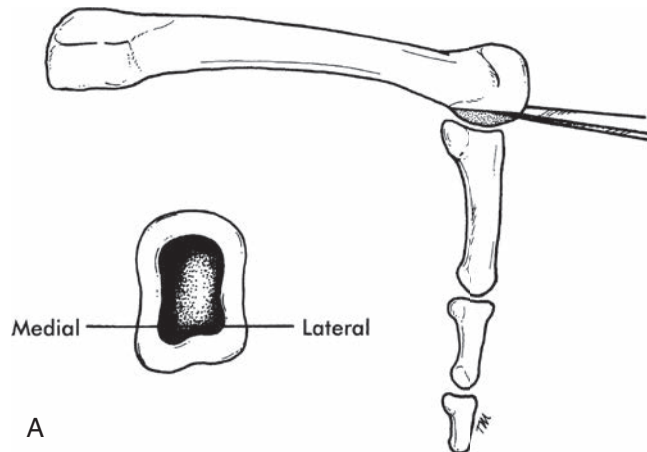


Fig. 20.12 (A) Plantar condylectomy for a discrete intractable plantar keratosis. (B) Intraoperative view of plantar condylectomy (one-fourth to one-third of the plantar metatarsal head is excised).

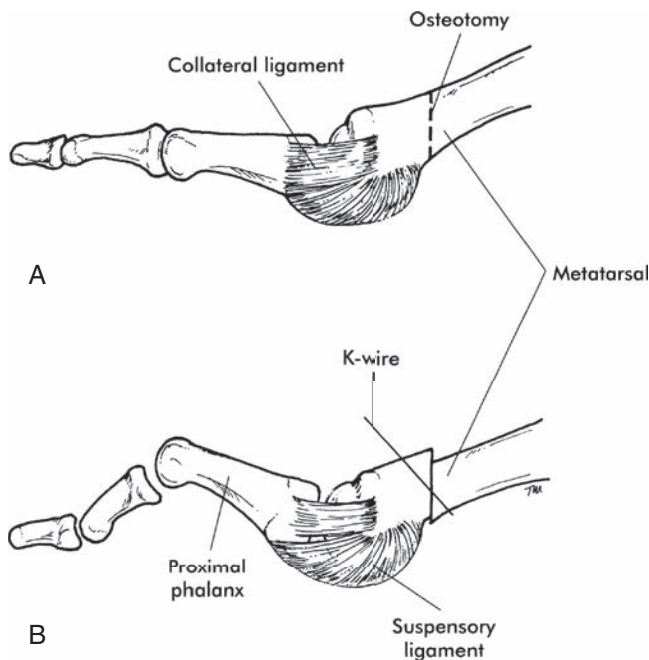


Fig. 20.13 (A) Distal oblique osteotomy (dotted line shows proposed osteotomy site). (B) Following displacement and internal fixation with K-wire.



Fig. 20.14 Distal chevron osteotomy with internal fixation. (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:93.)

2. A dorsal longitudinal incision is centered over the involved metatarsal.
- 3A. If a *distal oblique osteotomy*⁹ is performed (Fig. 20.13), the cut is directed in a vertical direction. The metatarsal head is displaced upward 3 mm¹⁰ and fixed with a 0.045 K-wire.
- 3B. If a *vertical chevron osteotomy*² is performed (Fig. 20.14), the V-shaped osteotomy is directed in a vertical direction.

(This has more coronal stability than a transverse osteotomy.) The metatarsal head is displaced upward 3 mm and fixed with a 0.045 K-wire.

- 3C. If a *proximal transverse osteotomy*¹¹ is performed (Fig. 20.15), a dorsally based closing wedge osteotomy is performed. The further proximal the osteotomy is located, the more sagittal plane elevation is achieved with wedge



Fig. 20.15 Proximal closing wedge osteotomy with screw fixation. (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:91.)

removal. (Care must be taken not to overcorrect at the osteotomy site in order to prevent transfer metatarsalgia.) The wedge may be removed with a sagittal saw or with a small rongeur. Internal fixation is recommended. A plate-and-screw construct, screw-only construct, or K-wire is used.

4. The wound is closed in a routine fashion. A gauze and tape dressing is applied and changed on a weekly basis. The patient is allowed to heel weight bear in a wooden-soled shoe.
5. Sutures are removed 3 weeks following surgery. Percutaneous K-wires are removed 3 to 4 weeks following surgery. The forefoot is then strapped with tape and gauze until symptoms resolve.
6. Radiographic union is important before aggressive athletic activity can begin.

Athletic activity is permitted as swelling and pain diminish. In general, a patient can return to nonimpact activities at 2 months, limited impact activities such as jogging at 3 months, and progress as tolerated.

In general, with a partial plantar condylectomy, satisfactory results are attained for relieving the symptoms of a discrete well-localized IPK.⁸ Likewise, a distal osteotomy^{9,11} may be efficacious for a similar lesion. A diffuse callus in the athlete is probably best padded and shaved, as a more extensive procedure involving a diaphyseal osteotomy¹² may require prolonged healing time and place the athlete at greater risk for delayed healing, malunion, and transfer metatarsalgia. A proximal closing wedge osteotomy⁹ may be used to elevate a symptomatic long second or third metatarsal.

INTERDIGITAL NEUROMAS

An interdigital neuroma may be a source of ill-defined forefoot pain. Most commonly located in the third intermetatarsal space (IMS), a neuroma is rarely isolated to the first, second, or fourth

interspace. Rarely (less than 3% of cases) do two neuromas occur in the same foot simultaneously.¹³

Typically an athlete initially describes ill-defined forefoot pain, often exacerbated with running or sports activities, which is relieved by rest or removal of shoes.

Although ill-defined forefoot discomfort is common, the treating physician needs to help patients define the exact area of pain. With time and education, athletes may be able to pinpoint the exact area of pain from the dorsal and plantar aspect of the forefoot. Neuritic symptoms or numbness in either the second or third common digital nerve distribution may be observed. Intermetatarsal space neuromas are located in the third intermetatarsal space about 85% of the time and in the other intermetatarsal spaces less commonly. It is important to appreciate that more generalized neuritic symptoms in the toes may have other causes. Neuritis may cause irritation of all of the intermetatarsal spaces. Alternatively, tarsal tunnel syndrome or compression of Baxter's nerve proximally at the ankle can cause generalized neuritis or, in the case of compression of Baxter's nerve, may preferentially cause symptoms in the distribution of the lateral plantar nerve.

On physical examination, care is taken to observe for signs of peripheral neuropathy, vascular insufficiency, or neuritic symptoms at the ankle. Peripheral neuropathy is characterized by a loss of cutaneous, positional, and vibratory sensation. The Semmes-Weinstein 5.07 monofilament is the classic test for protective sensation. Vascular insufficiency is characterized by a loss of distal hair, lack of pulses, dependent rubor, varicose veins, atrophic skin, and delayed capillary refill. The toes are examined for fixed deformity. Any callus or IPK is noted, and the adjacent MTP joints are evaluated for pain or instability (see section on Metatarsophalangeal Instability). MTP capsular instability symptoms closely mimic those of an interdigital neuroma, especially in the second intermetatarsal space.¹⁴ This is thought to occur because the second intermetatarsal space nerve is near the plantar-lateral second MTP joint capsule, which is commonly inflamed in both second MTP instability as well as second MTP joint capsulitis. In all three diagnoses, palpation of the involved interspace usually elicits pain. Grasping and compression of the transverse arch at the level of the metatarsal heads may elicit a click (Mulder's sign),¹⁵ which occurs when the neuroma subluxates below the metatarsal head and transverse metatarsal ligament (TML).

PEARL: DIAGNOSIS OF MTP INSTABILITY VERSUS INTERDIGITAL NEUROMA

When a patient has difficulty isolating the location of pain, a 1% lidocaine injection may be used to determine the site of pain.¹⁶ During serial office visits 1 week apart, the physician may inject the second IMS, then the third IMS, then the second MTP joint, then the third MTP joint. Fluoroscopy and injectable contrast may be used to verify an intraarticular injection. It is important to use small volumes (1–3mL) to prevent extravasation of anesthetic agent to adjacent structures. The patient is asked to repeat the activity that causes the most discomfort while anesthetized and keep a "pain diary" carefully describing how much of their symptoms improved and for how long. Within 1 or 2 hours the anesthetic wears off. When temporary relief is achieved with the injection, followed by recurrent symptoms, an anatomic diagnosis can be confirmed.¹⁴

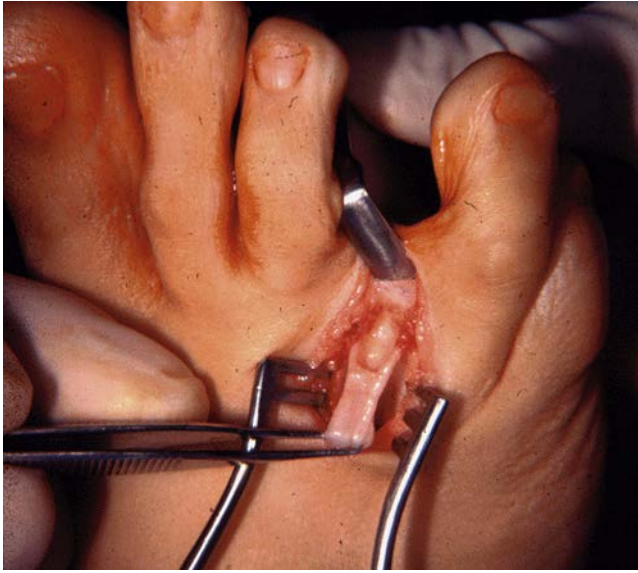


Fig. 20.16 Dorsal incision demonstrates a large interdigital neuroma. The transverse metatarsal ligament has been sectioned. (From Coughlin, MJ. Soft tissue afflictions. In: Chapman M, ed. *Operative Orthopaedics*. Philadelphia: JB Lippincott;1993:2289.)

Conservative Treatment

Early conservative treatment may alleviate symptoms in the athlete. With intermittent symptoms exacerbated by intense athletic activity, or sports of significant duration, a change in the type of activity or its duration may completely relieve symptoms.

Placing a small metatarsal pad just proximal to the symptomatic interspace may relieve symptoms. Change in athletic shoes may also alleviate pain.

When conservative methods including the modification of sports activities have not relieved symptoms, surgical intervention may be considered.¹⁷⁻¹⁹

Surgical Treatment: Excision of Interdigital Neuroma

1. The foot is prepped and draped in the usual sterile fashion. An Esmarch bandage is used to exsanguinate the foot. The ankle is carefully padded and the Esmarch is used as a tourniquet.
2. A 3-cm dorsal incision is centered in the involved interspace.
3. The dissection is carried down to the TML (Fig. 20.16).
4. A 2-3 prong Weitlaner retractor is used to distract the adjacent metatarsals and place the TML under tension.
5. The TML is sectioned to expose the neuroma and common digital nerve.
6. The digital nerves distal to the bifurcation are severed at the level of distal webspace. The proximal stumps are tensioned. A nerve freer is used to dissect longitudinally to isolate the common digital nerve. Special attention is directed at freeing the plantar and capsular branches. With tension on the proximal nerve, a scalpel is used to transect the nerve as proximal as possible in the interspace.



Fig. 20.17 Strapping of the foot is continued for 6 weeks postoperatively to promote healing of the transverse metatarsal ligament.

7. The interspace is inspected for any other nerve tissue that may be a cause of pain. It is important to sever any adjacent capsular nerve branches that prevent proximal migration of the nerve stump. The retractor is removed, and the surgical wound is irrigated and closed in a routine fashion.
8. A gauze and tape dressing is applied and changed on a weekly basis, and the patient is allowed to ambulate in a postoperative shoe.
9. Suture removal is carried out 3 weeks following surgery, and a circumferential gauze and tape strapping is continued for 3 more weeks to allow adequate healing of the TML if it was sectioned (Fig. 20.17).

Aggressive walking can be commenced 4 weeks following surgery, with increased activity as pain and swelling permit.

Patients can present with concurrent interdigital neuroma and MTP joint capsular instability. Isolated treatment of one of these conditions is unlikely to resolve the patient's symptoms. Simultaneous interdigital neuroma resection and stabilization of the MTP joint result in better outcomes than isolated procedures.¹⁴ Techniques to address capsular instability are addressed later in this chapter.

HARD CORNS AND SOFT CORNS

A hard corn (Fig. 20.18) develops over the lateral aspect of the fifth toe usually due to pressure of the shoe against an underlying exostosis or condyle on the fifth toe. Patients may complain of pain associated with a hypertrophic callus on the lateral aspect of the fifth toe. A soft corn (Fig. 20.19) develops between the toes due to pressure between two adjacent bony prominences. Patient may complain of exquisite pain; maceration sometimes occurs that resembles a mycotic infection. Desiccation of the lesion may then help to distinguish it from an infection.²⁰



Fig. 20.18 Hard corn with keratotic buildup. (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:50.)



Fig. 20.19 A soft corn is demonstrated in the fourth web space, mimicking a mycotic infection. (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:51.)

On physical examination, the obvious callosity occurs overlying a bony prominence. Radiographic evaluation may help to define the location of the lesion (Fig. 20.20).

Conservative Treatment

Padding of the hard corn (Fig. 20.21) may alleviate discomfort. Stretching of shoes overlying the lesion may decrease symptoms. Shaving of the callosity on a frequent basis may diminish the painful symptoms.

With a soft corn, padding of one or both toes with either a foam spacer (Fig. 20.22) or tubular foam gauze often eliminates



Fig. 20.20 Radiograph demonstrating the location of a soft corn (arrows). (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:51.)

compression between the two toes. Shaving of the callosity also may be indicated (see Case Study 20.3). When conservative measures have failed, surgical resection of the involved condyle may eliminate the prominence and alleviate the symptoms.

CASE STUDY 20.3

A 40-year-old jogger developed exquisite pain beneath the fourth and fifth toes. He recognized maceration in the fourth web space. It was unclear whether this was a fungus infection or a soft corn.

On his initial evaluation, radiographs demonstrated impingement between the PIP joint of the fourth toe and DIP joint of the fifth toe.

Initial treatment included using rubbing alcohol applied with a cotton-tipped applicator three times a day to desiccate the area. Then, lamb's wool was placed between the toes to pad and alleviate pressure between the two prominent condyles. Later, a foam spacer was placed between the toes, and the patient was allowed to resume all jogging activity. No surgery was performed.

Surgical Treatment for Hard Corns²¹

1. The foot is prepped and draped in the usual sterile fashion. Often a digital anesthetic block is used, although a foot block may also be considered.
2. A dorsolateral longitudinal incision is centered over the prominent lateral condyle.
3. With sharp dissection, the capsular fibers are peeled off of the condyle.
4. A rongeur is used to remove the prominent condyle, with care taken to leave enough articular surface to retain joint stability (Fig. 20.23).
5. The sharp bony edges are beveled with a rongeur.
6. The capsule is closed with two or three interrupted absorbable sutures.

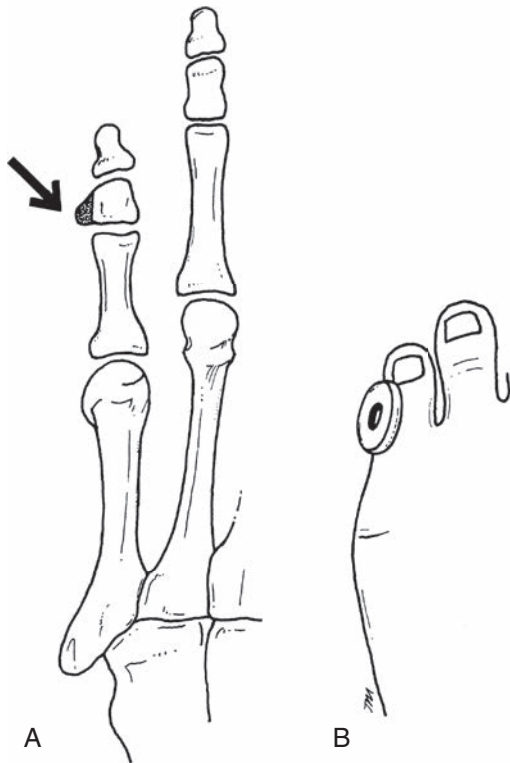


Fig. 20.21 (A) An underlying exostosis (arrow) combined with restrictive footwear leads to a hard corn. (B) A pad may be used to relieve pressure.



Fig. 20.22 A pad is used to relieve pressure in the web space. (From Mann RA, Coughlin MJ. *Video Textbook of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:51.)

7. A percutaneous flexor tenotomy is performed at the MTP joint.
8. The skin is closed with a running skin closure. A gauze and tape dressing is applied and changed on a weekly basis. The patient is allowed to ambulate in a postoperative shoe.

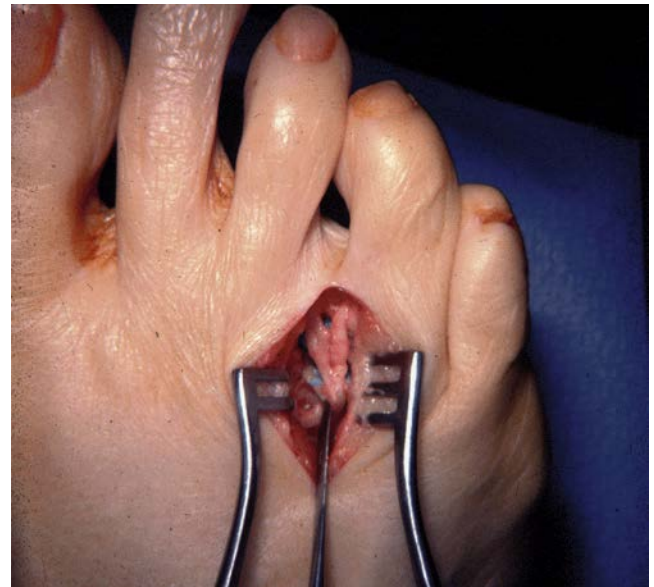


Fig. 20.23 A dorsal incision is used for the condylectomy as the treatment for a hard corn. (From Coughlin MJ. *Soft tissue afflictions*. In: Chapman M, ed. *Operative orthopaedics*. Philadelphia: JB Lippincott; 1993:2223.)

9. Sutures are removed 3 weeks after surgery. The toe is then taped to the adjacent toe for 3 more weeks to promote stability and avoid injury.

After suture removal, an increase in sports activity can begin. Walking and cycling may be started when sutures are removed. Running may begin after swelling has diminished enough to allow shoes to fit comfortably, typically 6 weeks postoperatively.

Surgical Treatment for Soft Corns

1. The foot is prepped and draped in the usual sterile fashion. Often a digital anesthetic block is used, although a foot block may also be considered.
2. A decision is made whether to treat both lesions on adjacent toes or to treat only one. (With a significant lesion on one toe and a minor lesion on the corresponding toe, a surgical repair of the larger lesion usually will successfully eliminate the entire problem.)
3. A dorsolateral longitudinal incision is centered over the prominent lateral condyle. This avoids an incision in the affected webs pace.
4. With sharp dissection, the capsular fibers are peeled off of the condyle.
5. A rongeur is used to remove the prominent condyle, with care taken to leave enough articular surface to retain joint stability (Fig. 20.24).
6. The sharp edges are beveled with a rongeur.
7. If a fixed contracture of the toes exists, a percutaneous tenotomy of the flexor tendon is performed.
8. The capsule is closed with an interrupted absorbable suture.
9. The skin is closed with a running skin closure. A gauze and tape dressing is applied and changed on a weekly basis. The patient is allowed to ambulate in a wooden-soled postoperative shoe.

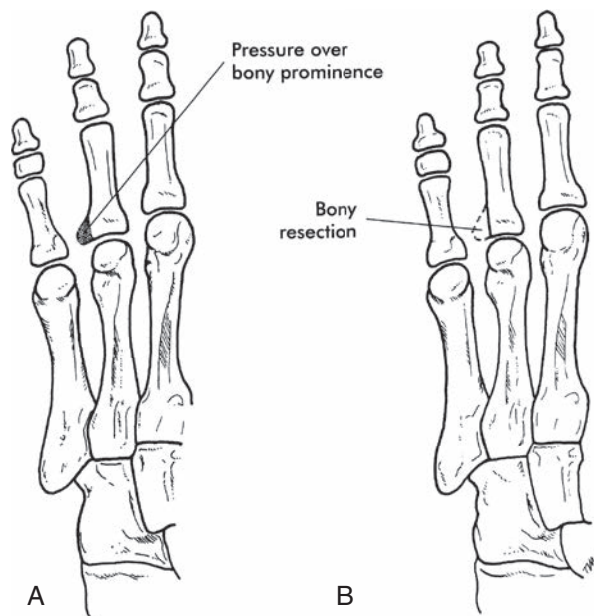


Fig. 20.24 (A) A soft corn may develop over the base of the proximal phalanx. (B) Resection of the bony prominence.



Fig. 20.25 Hammertoe deformity. (From Coughlin MJ. Operative repair of the fixed hammertoe deformity. *Foot and Ankle Int.* 2000;21:94–104, Fig. 1.)

10. Sutures are removed 3 weeks after surgery.
11. A small gauze spacer is used between the toes for another 3 weeks until the surgical incisions have softened. After suture removal, walking and cycling may be started. Running may begin after swelling has diminished enough to allow shoes to fit comfortably, typically 6 weeks postoperatively.

HAMMERTOES, MALLET TOES, CLAW TOES

Deformities of the lesser toes include both flexible and fixed deformities. Typically callus formation occurs over bony prominences, and at times during athletic activity these areas may become inflamed and painful. A hammertoe deformity (Fig. 2.25) is characterized by a flexion contracture at the PIP joint.

Early on, it may present as a flexible deformity that in time may become fixed. With a mallet toe (Fig. 20.26), there is a flexion contracture at the DIP joint. Early on, it may present as a flexible deformity due to tightness of the flexor digitorum longus (FDL) tendon. With time it may become a fixed deformity. A callus may develop dorsally over the DIP joint due to pressure or abrasion from impacting against the toe box. A callus may also develop at the tip of the toe due to pressure against the insole of the shoe.

With a claw toe deformity, typically a flexion contracture develops at the PIP joint with hyperextension at the MTP joint. A callosity may develop over the PIP joint; with a long-standing contracture, and IPK may develop beneath the metatarsal head. Early on, a flexible contracture may be passively correctable, although with time a fixed contracture may develop.

Subjectively a patient typically complains of pain over a prominent callus on the involved toe; occasionally a painful callus will develop at the tip of the toe.

On physical examination, the flexibility or rigidity of the deformity may determine the particular surgical repair, should it be necessary. The presence of multiple toe deformities, contractures at adjacent joints, and neurologic deficits must be appreciated during physical examination. With all of these lesser toe deformities, an athlete may complain of blistering, callus formation, swelling, or pain due to a dynamic or static deformity. Occasionally an infection may develop in the overlying tissue.

Conservative Treatment

Conservative care includes relieving pressure over the painful area.²² The use of roomy footwear will often relieve discomfort in the athlete. Padding often allows return to sports activity. Shaving of painful callosities may temporarily improve keratotic buildup. Often conservative care will allow an athlete to continue activity, although decreasing the duration or intensity of the workout or changing to a different sporting activity may be necessary on a temporary or permanent basis. When conservative measures do not allow acceptable athletic activity, surgical intervention may be considered.

Surgical Treatment: Hammertoe Repair²³

1. The foot is prepped and draped in the usual sterile fashion. Usually a digital nerve block is used as an anesthetic.
2. A dorsal elliptical skin incision is centered over the PIP joint. The incision is carried down to bone with excision of an ellipse of skin, extensor tendon, and capsule exposing the condyles of the proximal phalanx.
3. The collateral ligaments of the PIP joints are severed, enabling the condyles to be delivered.
4. A bone-cutting forceps is used to osteotomize the proximal phalanx in the supracondylar region (Fig. 20.27). The sharp edges are beveled with a rongeur.
5. The articular surface of the middle phalanx is exposed, and a rongeur is used to remove the articular surface.
6. A 0.045 K-wire is introduced at the PIP joint and driven distally, exiting the tip of the toe. Then with the toe reduced to the desired position, the K-wire is driven in a retrograde fashion, stabilizing the hammertoe repair. The pin is bent at

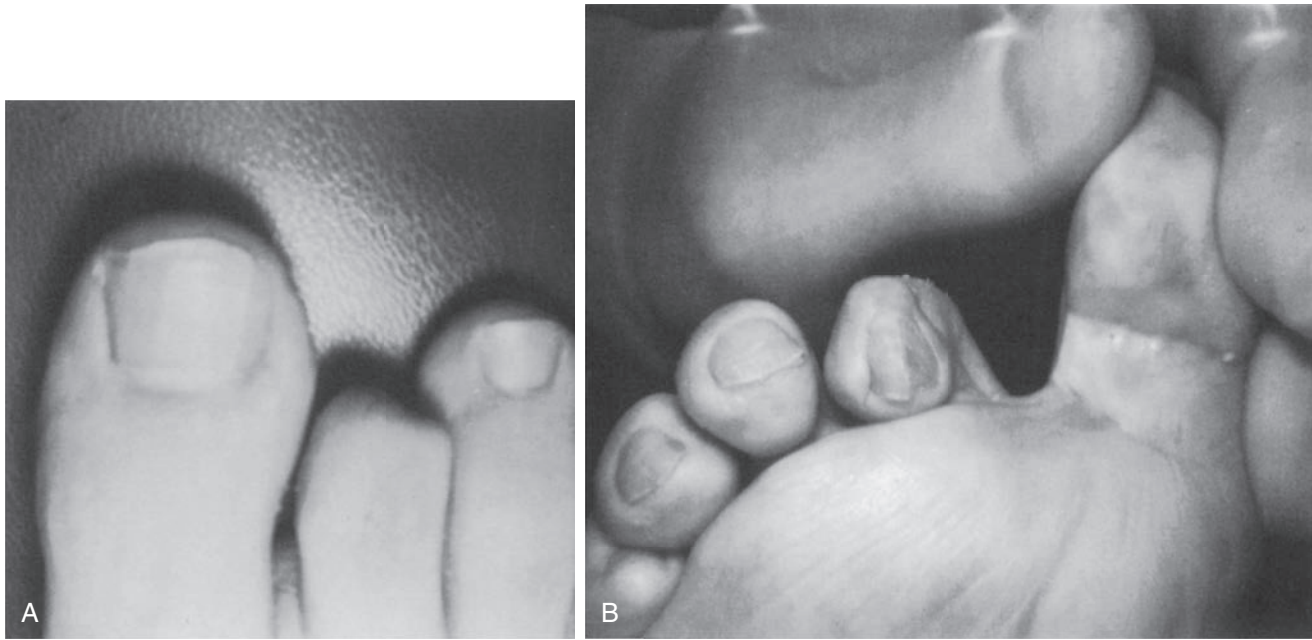


Fig. 20.26 Mallet toe deformity. (A) Dorsal and (B) plantar views. (From Mann RA, Coughlin MJ. *Video Text-book of Foot and Ankle Surgery*. St Louis: Medical Video Productions; 1991:48.)

the tip of the toe to prevent proximal migration. An intramedullary implant may be considered to eliminate the need for K-wire fixation.

7. A gauze and tape dressing is applied and changed on a weekly basis. The patient is permitted to ambulate in a stiff soled postoperative shoe. Sutures and K-wire are removed 4 weeks after surgery.
8. The patient is then instructed to tape the toe to an adjacent toe for an additional 4 weeks to protect it from injury.

After the K-wire is removed, increased walking activity is performed. Cycling may be allowed. Running or jogging is usually avoided until swelling has diminished (6 to 8 weeks). Whether an arthrodesis occurs at the PIP joint is not of significant concern. Fibrous ankylosis with a small amount of motion is equally acceptable.

Surgical Treatment: Early Flexible Mallet Toe Repair

1. The foot is prepped and draped in the usual sterile fashion. A digital block is used for anesthesia. (With an early mallet toe deformity, the toe can be passively corrected to neutral with pressure.)
2. A #11 scalpel blade is introduced on the plantar aspect of the DIP joint and the FDL tendon is released.
3. The incision is closed with an interrupted skin suture.
4. A gauze and tape dressing is applied, and the patient is allowed to ambulate in a postoperative shoe.
5. The sutures are removed 10 days after surgery.

Athletic activity can be resumed rapidly following this procedure with little downtime. In general, a patient can begin to advance activities at 2 weeks. Typically, full activities can be resumed by 4 weeks.

When a fixed mallet toe is corrected, a similar procedure is performed as is carried out for a hammertoe deformity. In this case the procedure is carried out at the DIP joint (Fig. 20.28).²⁴

CLAW TOE

A claw toe deformity may be flexible, semirigid, or fixed. Frequently it involves all of the toes on a foot. Although the etiology frequently is idiopathic, the treating physician should inspect the patient for other causes such as spasticity, muscular dystrophy, spinal abnormality, and previous trauma (old fractured tibia, old compartment syndrome, etc.).

Many cases may be effectively treated with roomy footwear, padding, and pedicures; however, on occasion an athlete is so symptomatic that surgery is contemplated. Although claw toes frequently involve multiple toes, they have similarities with different stages in the development and treatment of hammertoe deformity.

Early on, flexible claw toes (although multiple in nature) resemble flexible hammertoes. A flexor tendon transfer of the second, third, and fourth toes may achieve adequate realignment by releasing the contracted FDL tendon and depressing the proximal phalanx through the tendon transfer. Rarely is a flexor tendon transfer performed on the fifth toe. A flexor tenotomy is occasionally performed, although often the fifth toe is asymptomatic.

As a claw toe becomes fixed, a patient may develop symptoms of a hammertoe with callus formation overlying the PIP joint. Because of the fixed dorsiflexion contracture at the MTP joint, the toe buckles, depressing the metatarsal head. A plantar callus (IPK) may develop due to increased pressure beneath the metatarsal head. The treating physician needs to remember that the IPK is usually due to the contracted toe rather than to a prominent metatarsal condyle. Correction of the toe deformity often is associated with diminution or resolution of the plantar callosity.

The fixed claw toe resembles a fixed hammertoe, although the claw toe also has contracture at the MTP joint. A PIP joint contracture is surgically repaired (Fig. 20.29) with a condylectomy of the proximal phalanx (see the section on Fixed Hammertoe Repair). Once the PIP joint contracture has been corrected, attention must be directed to the MTP joint contracture.

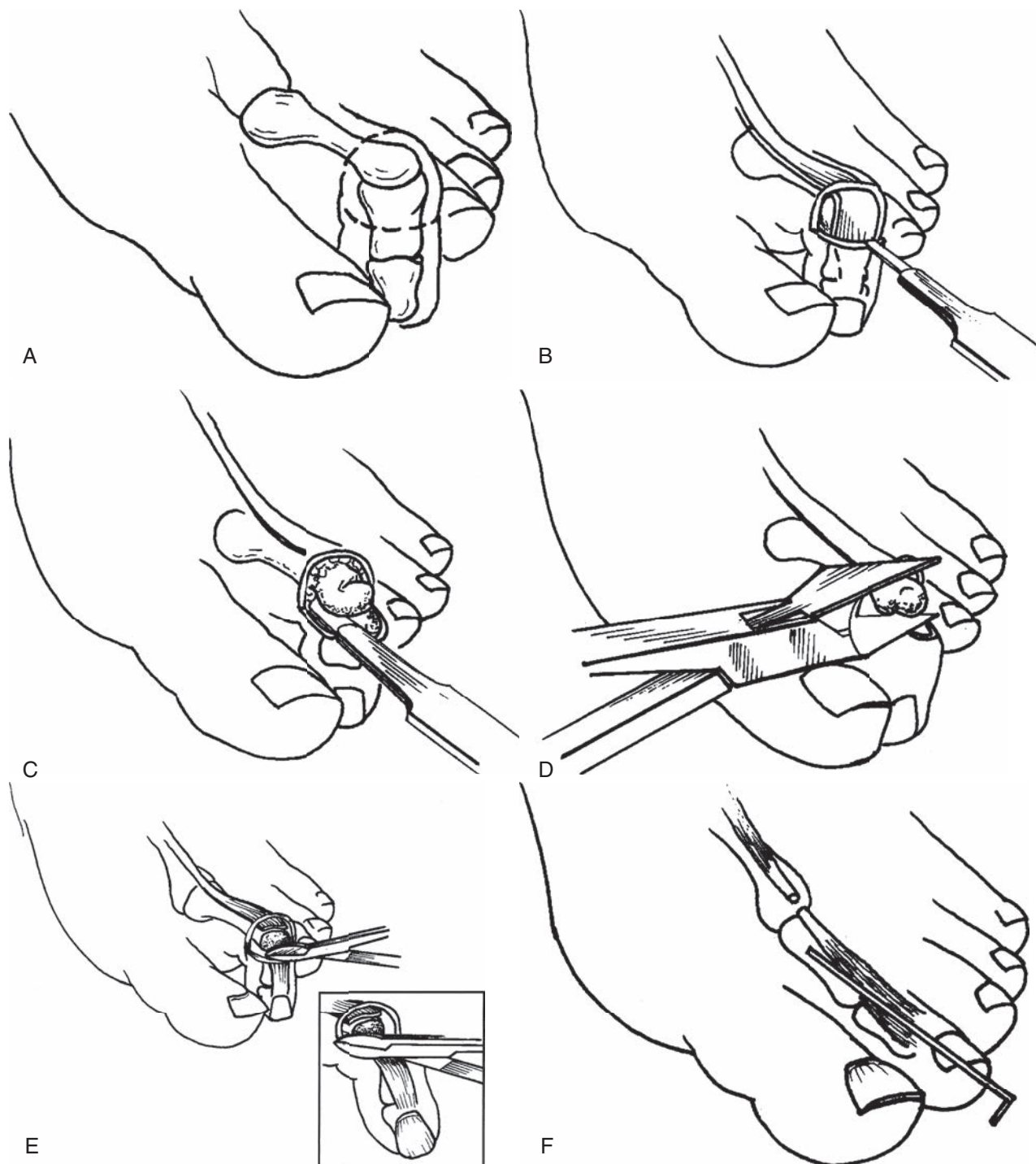


Fig. 20.27 Hammertoe repair. (A) An elliptical incision is made at the proximal interphalangeal joint, (B) the extensor tendon is excised, (C) collateral ligaments are released, and (D) the distal aspect of the proximal phalanx is removed, followed by (E) the proximal aspect of the middle phalanx. Finally, (F) a K-wire or intramedullary device is used to fix the toe. (From Coughlin MJ. Operative repair of the fixed hammertoe deformity. *Foot and Ankle Int.* 2000;21:94-104, Fig. 2.)

Surgical Treatment: Metatarsophalangeal Soft-Tissue Arthroplasty^{24,25}

1. The foot is prepped and draped in the usual sterile fashion. An Esmarch bandage is used to exsanguinate the foot. The ankle is carefully padded and the Esmarch is used as a tourniquet.
2. An oblique or longitudinal incision is centered over the MTP joint.
3. The long extensor tendon is split longitudinally and Z-lengthened.
4. The medial, dorsal, and lateral capsule is completely released to allow reduction of the MTP joint. (This requires a significant release in a plantar direction of both collateral ligaments.) When a toe still does not reduce completely following an MTP release, there may be adhesions between the plantar

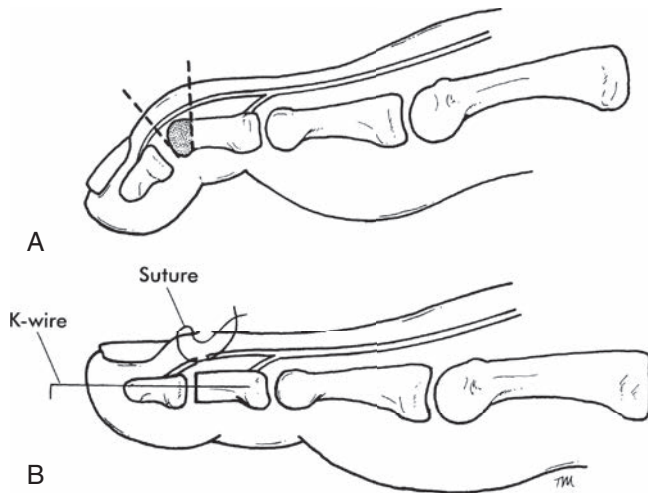


Fig. 20.28 mallet toe repair. (A) Proposed resection. (B) K-wire fixation following condylectomy.

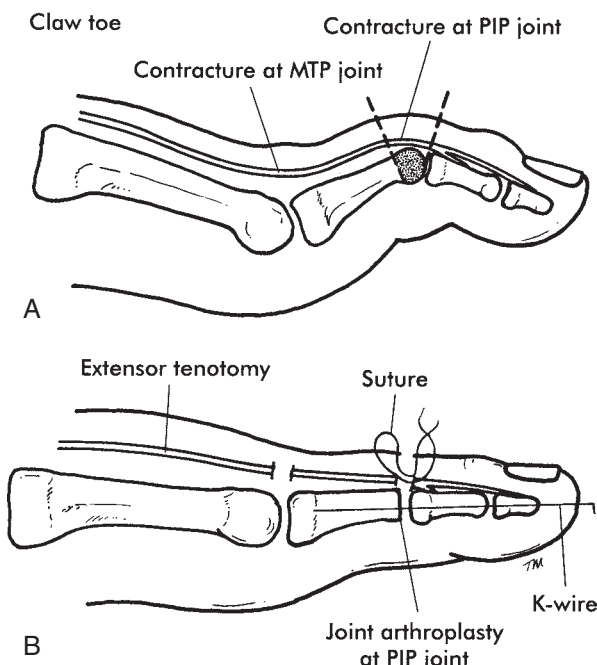


Fig. 20.29 Claw Toe Deformity. (A) Prior to and (B) following metatarsophalangeal (MTP) joint release, extensor tenotomy, and proximal interphalangeal (PIP) joint arthroplasty.

capsule and the plantar metatarsal head. These usually can be released with a curved Freer or McGlamry elevator. The toe should be then easily reducible in a dorsal plantar plane.

5. An 0.045 K-wire is used to stabilize the repair. The pin is introduced at the MTP joint and driven in a distal direction exiting the tip of the toe. (When combined with hammertoe repair, it is introduced at the PIP joint and driven proximally, exiting the base of the proximal phalanx. It is then driven distally, exiting the tip of the toe.)
6. The pin is then driven in a retrograde fashion, stabilizing the MTP joint. The pin is bent at the tip of the toe to prevent proximal migration.
7. The extensor tendon is repaired in a lengthened fashion, and the skin is closed in a routine fashion. A gauze and

tape dressing is applied and changed on a weekly basis.

The patient is allowed to ambulate in a postoperative shoe.

8. The K-wire and sutures are removed 3 to 4 weeks after surgery.

The toe is taped in a corrected position for 4 to 6 weeks. After removal of the K-wire, increased activity is permitted. Walking, cycling, and swimming are allowed. Jogging and running are prevented until adequate healing has occurred and swelling has subsided (6 to 8 weeks).

METATARSAOPHALANGEAL (MTP) JOINT INSTABILITY

Instability of the metatarsophalangeal joint can be an extremely difficult diagnosis to make, especially early on when there is a lack of clinical deformity. The second MTP joint is the most frequent location of instability due to the longer length of the second ray. In a report on athletes with second MTP instability,²⁶ Coughlin reported 100% of the patients to have an elongated second metatarsal in relationship to adjoining metatarsals. Most likely due to the stress of repeated and prolonged athletic activity, pain without deformity develops in the forefoot. The mechanism of instability is generally described as rupture or attenuation of collateral ligaments and plantar plate of the MTP joint.²⁷

Typically an athlete initially describes ill-defined forefoot pain often exacerbated by running and sports activities and relieved by rest. Sometimes pain increases with intensity and/or duration of sports activities.

On physical examination, the treating physician needs to initially isolate the exact point of tenderness. With palpation, tenderness is typically elicited over the plantar, medial, or lateral MTP capsule. Usually pain is not so pronounced in the third or second intermetatarsal spaces. It may initially be difficult to differentiate second MTP pain from a second IMS neuroma. A critical differentiating finding, however, is that there are no neuritis symptoms in the second or third toes and no numbness associated with capsulitis or instability of the second MTP joint. It is worth pointing out, however, that neuritic symptoms may be present due to inflammation of the nerve due to nearby MTP joint capsulitis.

Capsulitis or inflammation of an MTP joint can be associated with MTP joint instability, but it can also be caused by a systemic or localized arthritis. Freiberg's infarction is one possible cause of second MTP joint capsulitis or inflammation caused by avascular necrosis of the metatarsal head bone (Fig. 20.30). Degenerative or posttraumatic osteochondral lesions are another possible cause of MTP joint degeneration, which may originate in the second MTP joint or the adjacent first MTP joint causing generalized inflammation (Fig. 20.31). Systemic inflammatory conditions often involve other MTP joints. Whereas without a preexisting inflammatory arthropathy, only the second MTP joint is usually involved. A drawer sign²⁸ (Fig. 20.32) is typically the diagnostic test most helpful in defining capsulitis and/or instability of the MTP joint. By grasping the involved toe between the fingers and stressing the MTP joint in a dorsal plantar direction, exquisite pain can be elicited, likely due to



Fig. 20.30 Freiberg's infarction. Anterior-posterior radiograph demonstrating a Freiberg's infarction of the second metatarsal head.

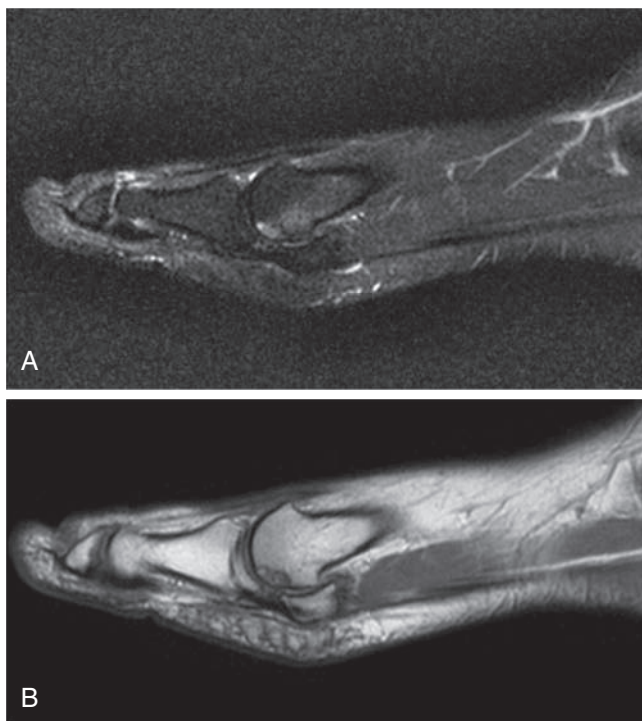


Fig. 20.31 (A) T2 and (B) T1 sagittal images of an osteochondral defect. Osteochondral lesion of the first metatarsal head causing pain in the plantar metatarsophalangeal joint.

stress on the attenuated plantar capsule or collateral ligaments. (This finding is absent in an isolated interdigital neuroma.)

With time, the diagnosis becomes obvious as the toe deviates^{29,30} (Fig. 20.33). Initially the toe deviates medially and with time dorsally, developing into a crossover second toe deformity. This development can be acute, although typically in athletes it occurs insidiously over several months.

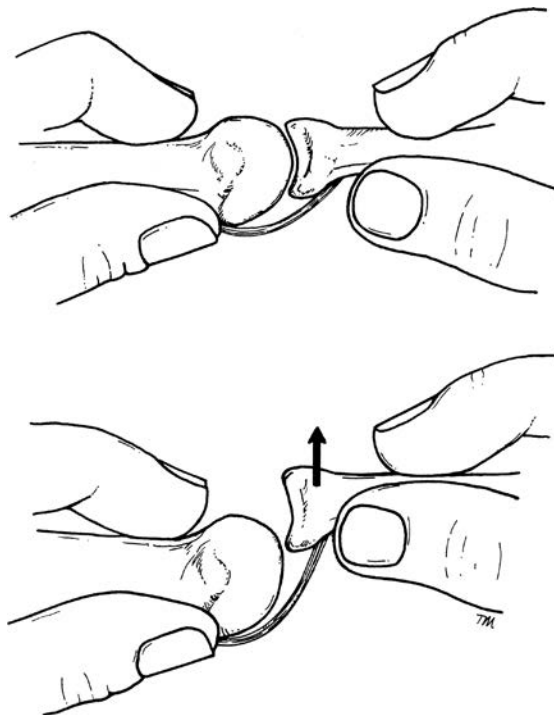


Fig. 20.32 A drawer sign is used to detect dorsal plantar instability.

Radiographic evaluation involves routine anterior-posterior (AP) and lateral radiographs to determine if there is widening of the joint space (effusion), narrowing (arthritis), or malalignment in relationship to the adjoining MTP joints (Fig. 20.34). Magnetic resonance imaging (MRI) can demonstrate a plantar plate tear; however, these tears can be small and visualization may be limited by the width of the cuts of the MRI (Fig. 20.35).

Conservative Treatment

Early conservative treatment relies on early diagnosis by the treating physician. Before deformity has developed at the second MTP joint, early MTP instability is best treated with taping the involved toe, padding, and a change in athletic activity. Taping requires stabilizing the toe to prevent dorsal plantar flexion excursion. Taping to an adjacent toe may be effective. A sling-type taping technique also may be effective (Fig. 20.36). An athlete may need to tape the involved toe for several months, although some athletes find it necessary to tape the toe only during sports activities. A metatarsal pad placed just proximal to the metatarsal head may alleviate pressure and relieve symptoms on the involved MTP capsule. Restructuring workouts and modifying athletic activity can be helpful in relieving pain.

With unsuccessful resolution of discomfort, or insistence on a higher level of athletic activity, surgical intervention may be considered.

Surgical Treatment: Metatarsophalangeal Instability

For patients that fail to respond to conservative treatment, several surgical techniques exist. Selection of the appropriate technique

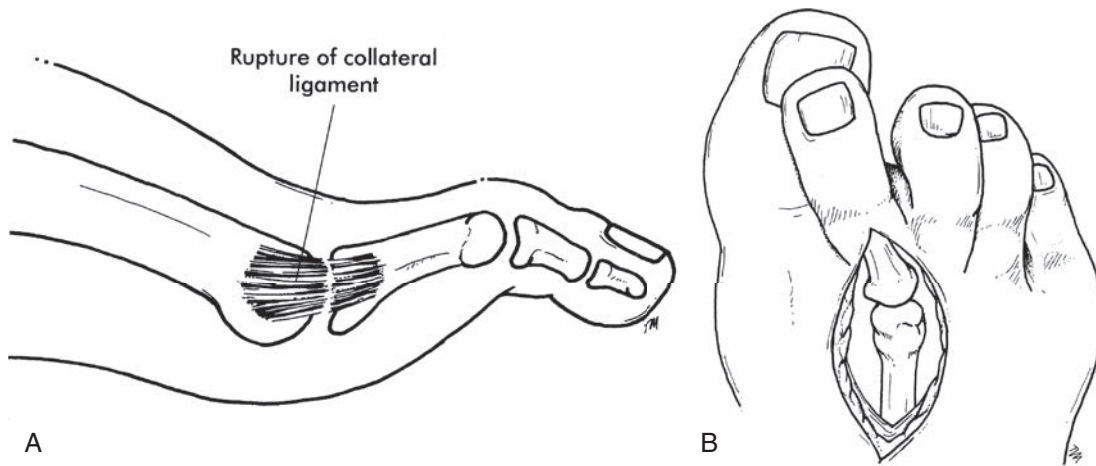


Fig. 20.33 (A) Instability of the second metatarsophalangeal joint with a crossover second toe may occur due to degeneration of the lateral collateral ligament. (B) Malalignment as demonstrated with a crossover second toe.

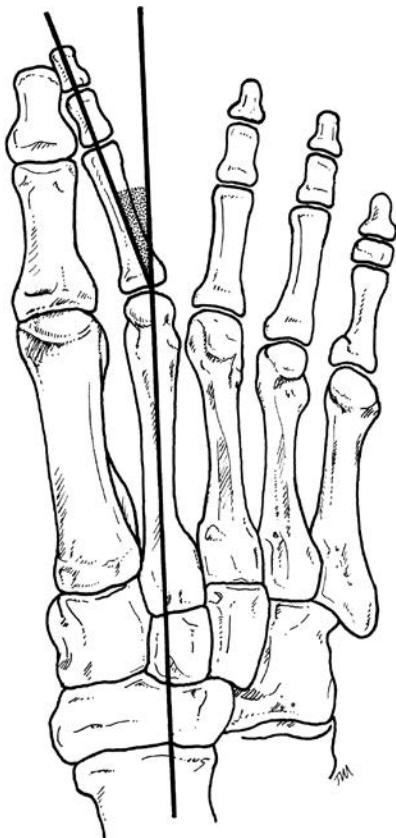


Fig. 20.34 Axial malalignment may be demonstrated on radiographic examination.

depends on clinical assessment of the deformity. Synovitis and mild deviation of the MTP joint is classified as a mild deformity and can be treated with capsular reefing. Dorsomedial deviation at the MTP joint or overlapping of the adjacent toe is considered moderate deformity and can be treated with capsular reefing and direct repair of the plantar plate.

Surgical Treatment: Weil Osteotomy

The Weil osteotomy is a shortening osteotomy that primary benefits patients with painful instability associated with a long

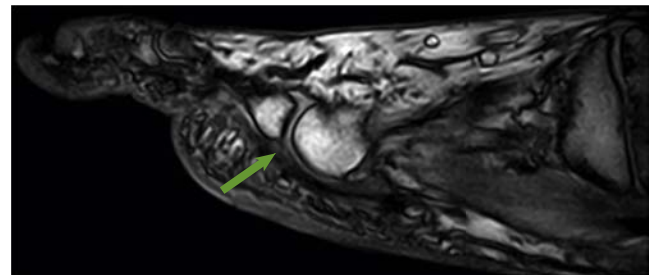


Fig. 20.35 Sagittal MRI cut demonstrating a plantar plate tear (arrow). (Courtesy Lowell Weil Jr, DPM)

lesser toe. This osteotomy should be avoided in patients with a plantarflexed metatarsal as it does not dorsally displace the distal fragment.³¹ The relative stability of this osteotomy with weight bearing and the use of internal fixation make this an ideal osteotomy for the active patient.

1. The foot is cleansed and draped in the routine fashion. The foot is exsanguinated with an Esmarch bandage. The ankle is carefully padded, and the Esmarch is used as an ankle tourniquet.
2. A 3-cm incision is made in the intermetatarsal space just proximal to the webspace. This allows access to adjacent metatarsals should more than one require attention.
3. The metatarsal head is exposed through a lateral capsular incision under the extensor tendon. The toe is plantarflexed exposing the metatarsal head.
4. A narrow oscillating saw is used to make the osteotomy parallel to the weight-bearing surface of the foot. The osteotomy originates in the dorsal quarter of the MTP joint (Fig. 20.37).
5. The distal fragment is displaced proximally until the metatarsal head is at the level of line drawn from the MTP of the first and fourth rays. The fragment is fixed with two threaded K-wires.
6. The overhanging bone is rongueured smooth.
7. The capsule is reefed if necessary, as described above. The capsule is repaired with absorbable sutures.
8. The patients are placed in surgical shoes and allowed to weight bear with crutches.

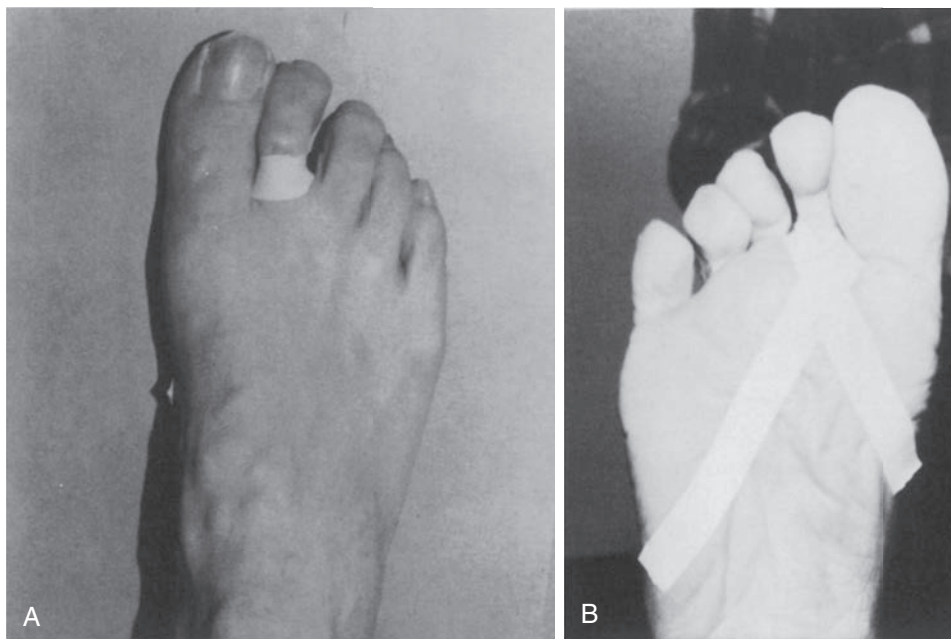


Fig. 20.36 Technique of taping a toe. (From Coughlin MJ. Crossover second toe deformity. *Foot Ankle* 1987;8:29-39.)

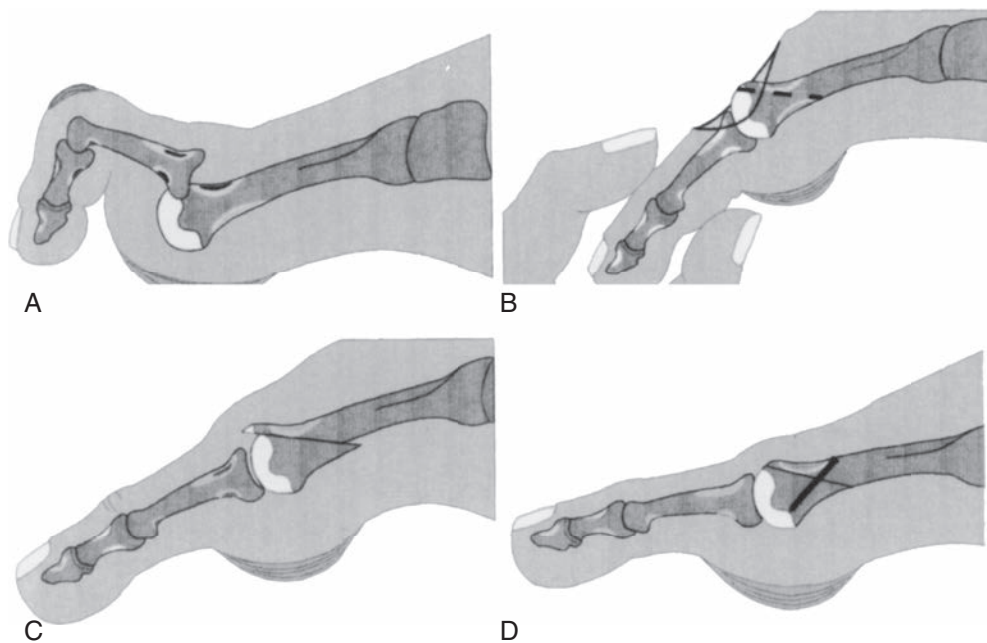


Fig. 20.37 The Weil osteotomy (A) Demonstrates MTP instability; (B) the toe plantarflexed to the dorsal MTP joint can be accessed for the osteotomy, which is aligned parallel to the plantar aspect of the foot; (C) the metatarsal head is slid proximally to the desired level and (D) fixed with a small screw from dorsal to plantar (From Trnka H. Comparison of the results of the Weil and Helal osteotomies for the treatment of metatarsalgia secondary to dislocation of the lesser metatarsophalangeal joints. *Foot and Ankle* 1999;20:72-79.)

Surgical Treatment: Plantar Plate Repair

The plantar plate can be repaired from either a dorsal or a plantar approach. Several techniques have been described, the outcomes of each have not been directly compared and the long-term outcomes of the repairs are limited. At least one study with 1-year follow-up demonstrated 80% good to excellent results utilizing a dorsal approach to the repair.³²

The following is a technique for a direct repair through a dorsal approach.

1. The foot is prepped and draped in the routine sterile fashion. The foot is exsanguinated with an Esmarch bandage. The ankle is carefully padded, and the Esmarch is used as an ankle tourniquet
2. A 3-cm dorsal midline incision is centered over the MTP joint. If hyperextension of the toe is present, the extensor

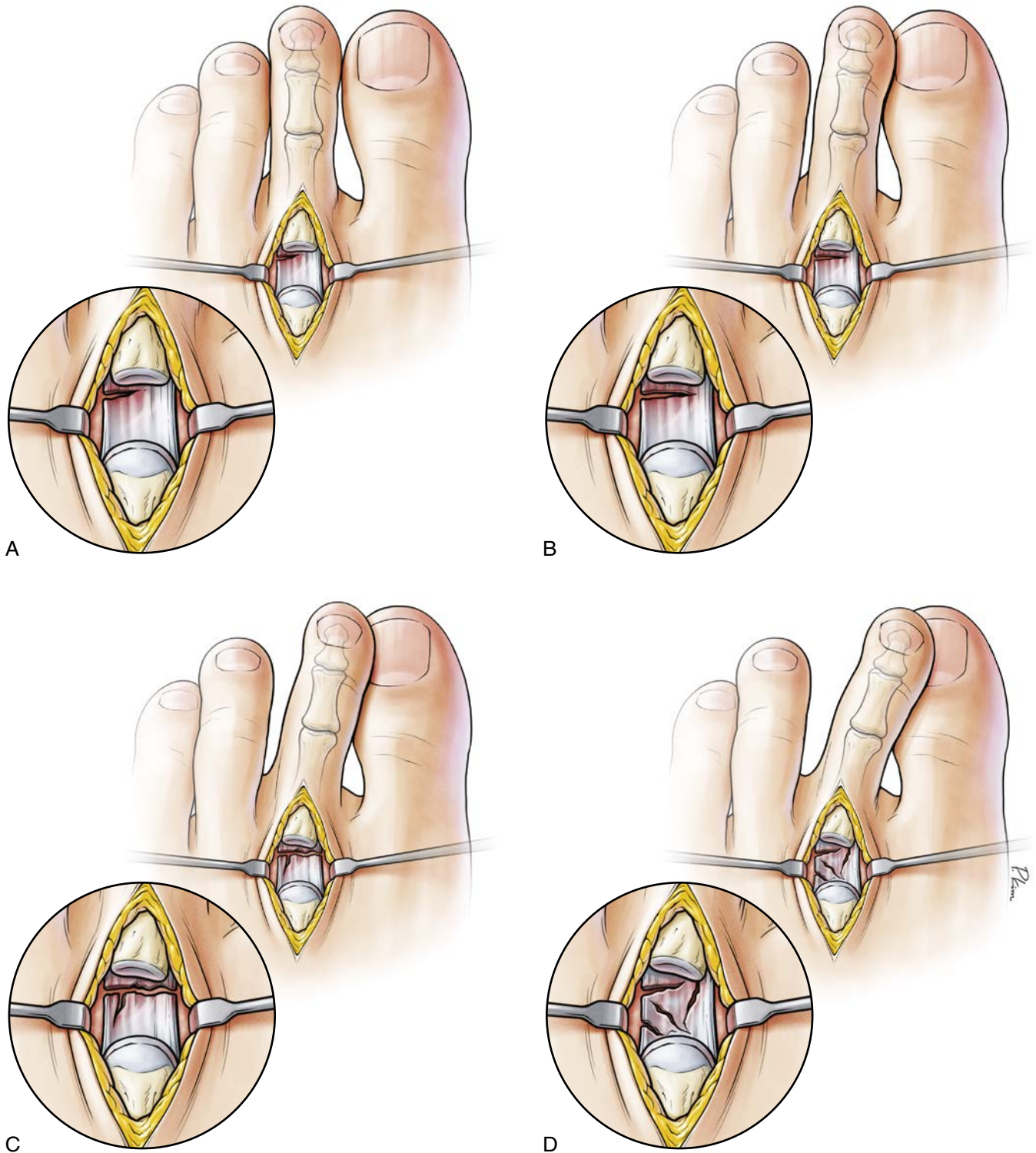


Fig. 20.38 plantar plate types. Plantar plate tears: (A) grade 1, (B) grade 2, (C) grade 3, and (D) grade 4.

digitorum longus tendon may be lengthened and later repaired at the conclusion of the procedure.

3. The dorsal MTP capsule is released. The medial and lateral collateral ligaments are released off the proximal phalanx. Care is taken to preserve the collateral ligament on the metatarsal head in order to preserve blood supply to the metatarsal head.

4. A Weil metatarsal osteotomy is performed (see previous section) parallel to the plantar aspect of the foot, and the metatarsal head is translated proximally 8 to 10 mm. It is fixed temporarily in a proximal position with a vertical Kirschner wire. The remaining 2–3 mm of the dorsal metaphyseal bone is removed to improve visualization of and gain access to the plantar plate.

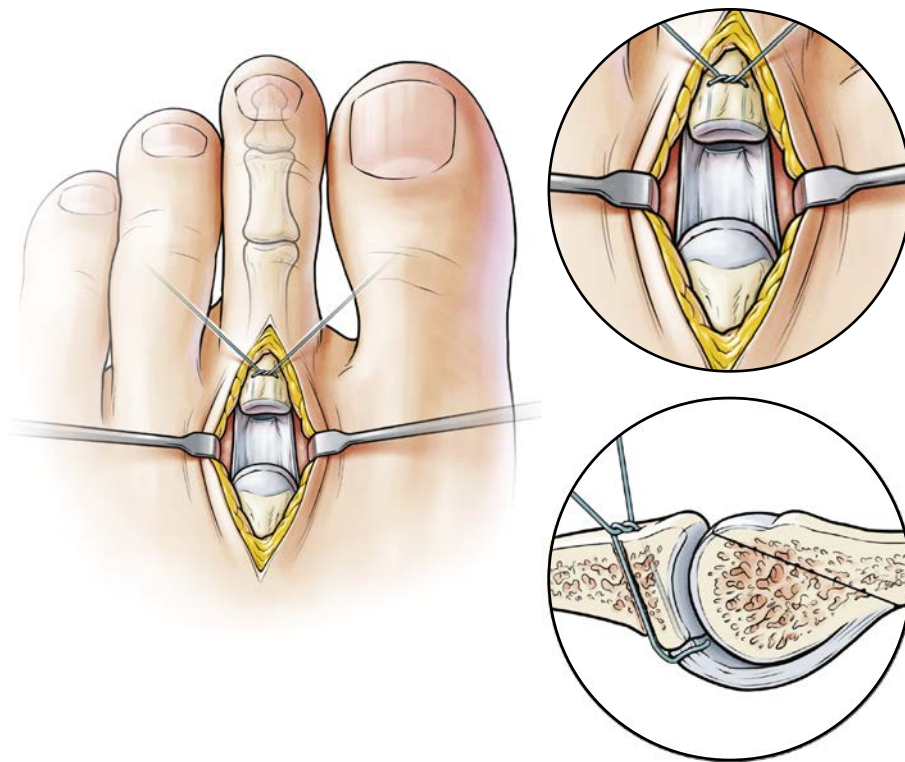


Fig. 20.39 Bone tunnels. Figure demonstrating suture passage through the plantar plate in a horizontal mattress fashion followed by passage through the proximal phalanx bone tunnels.

5. A second K-wire is placed in the proximal phalanx metaphysis. A joint distractor is positioned over the two K-wires and used to open the joint and directly inspect the plantar plate for pathology. The tear type guides the suturing technique (Fig. 20.38).
6. A distal transverse tear is repaired with one or two horizontal mattress sutures. A longitudinal tear is often repaired using a suture passer. Commercial devices can aid in this technique.
7. Following suture placement in the plantar plate, two vertical holes are drilled in the base of the proximal phalanx. The sutures are then passed through the phalangeal drill holes (Fig. 20.39).
8. The Weil osteotomy is fixed in a corrected position. In the event that the patient has a long second metatarsal, the surgeon may consider fixing the Weil osteotomy in a shortened position to better match the other lesser toes in order to take stress off the second MTP joint with dorsiflexion.
9. After fixation of the Weil osteotomy, the sutures are tied over the bone bridge of the proximal phalanx with the toe held in roughly 30 degrees of plantarflexion. The drawer test will generally demonstrate improved stability immediately after fixation.
10. The wound is closed in a routine fashion. A gauze and tape dressing is applied and changed on a weekly basis. The patient is allowed to ambulate in a wooden-soled shoe.
11. Sutures are removed 3 weeks after surgery. If a K-wire has been placed, it is removed at this time.

For those returning to athletic activity, the patients are allowed to begin forefoot weight-bearing activities at 6 weeks

postoperatively. A graphite insole with a soft orthotic is placed in the shoe to prevent MTP dorsiflexion. It is recommended that this insert be used for 6 months postoperatively.

CONCLUSION

When correctly diagnosed and treated, forefoot disorders should not limit athletic endeavors. While many of these conditions are treated nonoperatively, the orthopedic surgeon often is the most appropriate clinician to identify the problem and direct treatment. When operative treatment is required, the patient should be able to resume activities at their previous level of competition.

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Great-Toe Disorders

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INTRODUCTION

Injuries to the hallux metatarsophalangeal (MTP) joint are not uncommon, particularly in the running athlete, and may result in chronic pain and deformity. Causes of hallux injuries range from soft-tissue disruption to overuse and degeneration. Trainers and physicians may fail to recognize the potential dysfunction of these injuries, thus providing inadequate care and protection from further injury. Long-term sequelae of even isolated soft-tissue injury include flexor hallucis longus (FHL) tendon tears, hallux valgus or varus, cock-up deformity with or without interphalangeal (IP) joint contracture, and degenerative joint disease (DJD), i.e., hallux rigidus.

Clanton and Ford¹ found that foot injuries rank third behind ankle and knee injuries as the most common time-loss injury among university athletes. Of these foot injuries, a large proportion were sprains of the forefoot and, more specifically, the hallux MTP joint. Hunt and colleagues² demonstrated that the incidence of turf toe injuries was 0.062 per 1000 athlete exposures, but were 14 times more likely to occur during games than practice and more common in running backs and quarterbacks. In the author's practice, we have seen a number of professional athletes with a broad range of injuries to the great toe and base this chapter on our experiences.

Great-toe injuries can lead to significant functional disability, especially when not recognized early. In the short term, these injuries can result in difficulties with push-off strength and running. Long-term sequelae typically involve pain due to progressive degeneration. Physicians involved in the treatment of foot and ankle injuries, especially those caring for athletes, must become familiar with the spectrum of injuries about the hallux MTP joint, the biomechanical implications, the conservative and operative treatments for these injuries, and the late sequelae encountered in these athletes.

ANATOMY

The provider responsible for the care of athletes with great-toe injuries must have a keen knowledge of the anatomy of the

hallux MTP joint. In the simplest of terms, the motion of the joint consists of rolling, sliding, and compression. More specifically, the morphology of this joint allows for plantarflexion and dorsiflexion but very limited abduction and adduction. The fact that there is more than one center of motion contradicts the theory of a simple, hinged joint. Instead, the joint is a dynamic acetabulum or "hammock" as described by Kelikian.³ The joint articulation provides little of the overall stability because of the shallow, glenoid-like cavity of the proximal phalanx. Most of the stability comes instead from the capsular-ligamentous-sesamoid complex, which is described in detail later.

There are two sets of ligaments that contribute to the stability of the metatarsal (MT) head as it articulates with the proximal phalanx: the medial and lateral collateral ligaments and the metatarsosesamoid suspensory ligaments.⁴ The fan-shaped medial collateral ligament is composed of the medial MTP ligament and the medial metatarsosesamoid ligament (Fig. 21.1). The lateral collateral ligament is structured in a similar fashion.⁴

In addition to the collateral ligaments, the strong, fibrous plantar plate (see Fig. 21.1) also affords structural support. The capsular ligamentous complex of the hallux MTP joint, distal to the sesamoids, is actually a confluence of structures including the plantar plate, collateral ligaments, the flexor hallucis brevis (FHB), the adductor hallucis, and abductor hallucis tendons. This plantar plate is attached firmly to the base of the proximal phalanx and only loosely attached at the MT neck through the capsule.⁵

The split tendon of the FHB runs along the plantar aspect of the hallux and envelops the sesamoids before inserting at the base of the proximal phalanx as the capsular-ligamentous complex (Fig. 21.1). The two sesamoids are united by a thick, interseamoid ligament, all of which help to maintain the course of the FHL tendon. Adding to the stability of the hallux MTP joint are three other intrinsic muscles of the great toe. The extensor hallucis brevis (EHB) originates at the fascia overlying the sinus tarsi and runs obliquely to attach into the extensor mechanism on the dorsum of the MTP joint. It functions primarily as an extensor of the hallux MTP joint. On the plantar

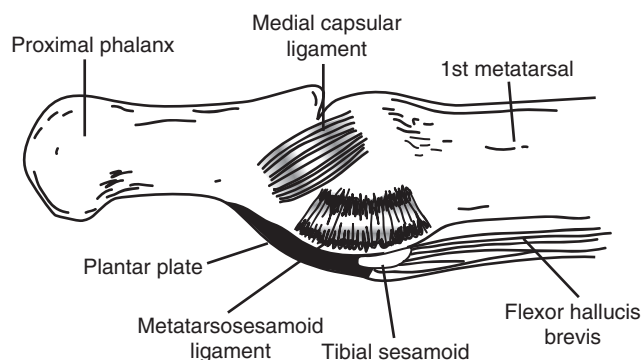


Fig. 21.1 Medial diagrammatic representation of first metatarsophalangeal joint. (From Adelaar RS, ed: *Disorders of the great toe*, Rosemont, IL: American Academy of Orthopaedic Surgeons; 1997.)

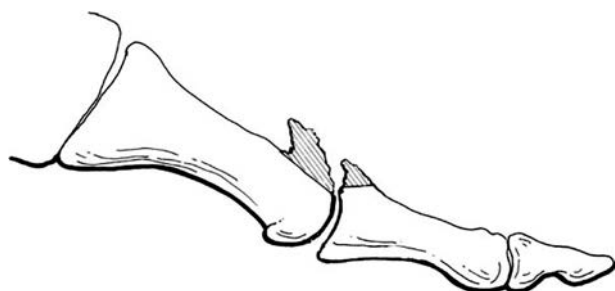


Fig. 21.2 Twenty percent to 30% of the metatarsal head is removed, as well as the exostosis. (From Coughlin MJ, Mann RA, eds: *Surgery of the foot and ankle*, 7th ed, St Louis: Mosby-Year Book; 1999.)

aspect, the abductor and adductor hallucis tendons insert on the medial and lateral aspects of the hallux MTP joint, respectively. These tendons blend into the capsular-ligamentous complex, as well as the sesamoids, to provide additional structural support (Fig. 21.2).⁶

- Not a simple, hinged joint.
- Most of the stability comes instead from the capsular-ligamentous-sesamoid complex.
- Capsular ligamentous complex: plantar plate, collateral ligaments, FHB, adductor hallucis, and abductor hallucis tendons.
- Collateral ligaments have phalangeal and sesamoid insertions.
- Split tendon of the FHB runs along the plantar aspect of the hallux and envelops the sesamoids before inserting at the base of the proximal phalanx.

BIOMECHANICS

The hallux MTP joint lies in an intricate balance of opposing tendons and ligaments. The anatomy outlined previously, especially with regard to the plantar plate, is important when considering the biomechanical demands placed on the first MTP joint. During normal gait, the great toe typically supports twice the load of each of the lesser toes and accommodates forces reaching 40% to 60% of body weight.⁶ During athletic activity, including jogging and running, the peak forces may approach two to three times body weight, and the forces increase to eight-fold when a running jump is performed.^{7,8}

The range of motion (ROM) in the normal foot has been studied extensively; it is noted to be highly variable and to decrease with aging. In the resting position, the first MTP joint is in a mean resting position of 16 degrees of dorsiflexion. The passive arc of motion was noted by Joseph to be from 3 to 43 degrees of plantarflexion and from 40 to 100 degrees of dorsiflexion.⁹ The mean passive MTP joint dorsiflexion during push-off was 84 degrees. One study found that at least 60 degrees of dorsiflexion is considered normal in barefoot walking on a level surface.¹⁰ Athletes may accommodate up to 50% reduction in MTP joint motion resulting from acute injury to the plantar plate or hallux rigidus by various gait adjustments such as foot/leg external rotation, shortened stride, and increased ankle, knee, or hip motion.⁵ In addition, a stiff-soled shoe is capable of decreasing MTP joint dorsiflexion to 25 to 30 degrees without significantly affecting gait.¹⁰

The effects on the push-off power of the great toe following sesamoidectomy have been studied in vitro by Aper et al.¹¹ They confirmed the importance of this seemingly insignificant bone to the function of the toe, particularly in the athlete, in whom even a small loss of power will affect overall performance. The study noted that the isolated excision of the tibial sesamoid equated to an 11% loss of flexor power, there was 19% loss for a fibular sesamoidectomy, and 32% when both are excised.¹¹

- Great toe supports twice the weight of each lesser toe.
- Hallux dorsiflexion during gait/running is 60 to 84 degrees.
- Up to 50% reduction in ROM can be accommodated through gait adjustments such as foot/leg external rotation, shortened stride, and increased ankle, knee, or hip motion.
- Sesamoidectomy: tibial excision results in 11% loss of flexor power, fibular 19% loss, and 32% when both are excised.

SPECIFIC ENTITIES OF THE GREAT TOE

Hallux Rigidus

Hallux rigidus is defined as a localized degeneration of the hallux MTP joint. It was first described as hallux flexus in 1887 by Davies-Colley.¹² In his first description of this condition, he discussed a plantarflexed posture of phalanx relative to MT head. The actual term “hallux rigidus” was coined by Cotterill in 1888 and remains the most common term used today.¹³ Numerous papers have theorized the etiology and pathophysiology of hallux rigidus. One such theory is that of metatarsus elevatus, a term describing the dorsiflexed posture of the first ray in relationship to the foot and the subsequent plantarflexed posture of the hallux. This has been discussed by many authors, but the most current data indicate that the elevated posture of the first MT improves after dorsal decompression of the hallux MTP joint.^{14–17} Overuse and repetitive dorsiflexion forces, such as those occurring in a runner or kicker, may lead to chondral lesions and other occult injuries¹⁸ or to osteochondritis dissecans.^{3,19,20} It also may result as a sequelae to a turf-toe injury. Anatomic abnormalities that may lead to hallux rigidus include the flat or pronated foot,^{16,21,22} a long first MT or hallux,²² and a flat MT head.²³ At this time the true potential etiologies for the development of hallux rigidus remain in question.

Clinical grading from mild to severe (or I, II, and III) has been proposed by many authors. Grading depends on the severity of disease and is based on ROM, pain or crepitus with motion, the size of the dorsal osteophyte on the MT head, the presence of sesamoid involvement, and the radiographic alignment of the hallux (on anterior-posterior [AP] and lateral views). A radiographic classification scheme was created by Hattrup and Johnson in 1986.²⁴ Their grade I is considered mild; the joint space is maintained and there is minimal spurring. Grade II is moderate disease in which the joint space is narrow, bony proliferation is present on the MT head and phalanx, and there is subchondral sclerosis and/or cyst formation. Grade III is the severe type, with significant joint space narrowing and extensive bony proliferation that involves the entire periphery and includes loose bodies, a dorsal ossicle, or subchondral cyst formation.²⁴ Easley et al.³³ described the prognostic importance of midrange crepitance, and thereafter a grading scheme was proposed by Coughlin and Shurnas,²⁵ combining objective and subjective clinical data with radiographic findings (Grades 0 to IV). Treatment recommendations are made on the basis of grade severity.

Symptoms with which the typical athlete may present include pain that is worse with push-off and more severe after increased activity (i.e., twice-a-day practice regimens), as well as swelling. Although there may be bilateral radiographic involvement, the patient almost always presents with unilateral symptoms. Swelling and the bony prominence itself may interfere with athletic footwear (especially in soccer and football, sports in which athletes prefer tightly fitting footwear). A dysesthesia in the dorsomedial cutaneous nerve can result from tight footwear's impinging on the bony prominence. Occasionally transfer lesions and metatarsalgia may develop, secondary to the lack of hallux dorsiflexion, which results in increased pressure on the lateral forefoot.

In treating hallux rigidus in the athlete, one must consider the sporting activity and position played (i.e., a lineman who requires little hallux MTP dorsiflexion vs. a running back or wide receiver), footwear requirement, and ROM of the entire foot and ankle. Even more minor or early-presenting cases can be problematic because some athletes create more forceful dorsiflexion, which can limit the function of the runner and incapacitate the dancer. As one seeks to improve the overall motion of the effected joint, it is paramount that the patient understands that if a bad joint is provided more motion, it may degenerate more quickly and lead to worsened symptoms.

Nonoperative treatment options include the use of nonsteroidal antiinflammatory drugs (NSAIDs) and footwear modifications. Shoes of adequate size and a more full-fitted toe box or increased depth are helpful and can be modified further with a balloon patch over bony prominences. Turf-toe inserts (Springlite, Otto Bock, Minneapolis, MN) that limit dorsiflexion and subsequent dorsal impingement are potentially useful but may limit performance in the elite runner. Rigid rocker soles function in the same manner as semirigid inserts and, although helpful in the general population, are not popular with the athlete because of the increased weight and excessive stiffness. Orthotic devices can unload the hallux MTP joint, but one must remember

to increase the shoe size to accommodate for it. Taping techniques can limit dorsiflexion and provide pain relief. Application is the same as that for turf-toe; however, skin problems such as blistering can occur. Steroid injections must be given judiciously and perhaps only for unique/special game situations. Repeated injections may accelerate the degenerative process.²⁶

Surgery in the management of hallux rigidus is feasible, and there are many options. The decision to proceed with surgery requires a lengthy discussion with not only the athlete but the trainer and possibly the athlete's agent. It must be emphasized to all parties that this is an arthritic process, there is no "cure," and there is the potential for a lengthy rehabilitation with incomplete resolution of the symptoms. The physician must determine the following: What is causing the problem? Is it the bony prominence over the MT head and secondary footwear irritation? Is there limited ROM? Are there biomechanical implications such as poor push-off? Does the athlete suffer from transfer pain issues and other compensatory problems? Lastly, and most concerning, is there the presence of global pain and diffuse arthritis, especially in sesamoid-MT articulation?

The most commonly performed surgical procedure in the management of hallux rigidus is a cheilectomy. This procedure can be defined in general as an excision of an irregular osseous rim that interferes with motion of a joint. In this particular instance it is the removal of the dorsal osteophyte of the MT head. As noted previously, the athlete should be counseled that the underlying condition is DJD and that full symptom relief is not realistic. A cheilectomy may prolong the athletic life of the individual but probably does not slow the rate of joint degeneration. As a general rule, the dorsal ridge does not recur, but progressive narrowing of the joint is expected to occur.

Indications for a cheilectomy include a lateral radiograph showing that reasonable space exists in the plantar one-half of the MTP joint. There should be an absence of pain or crepitus with midrange motion and no sesamoid-MT pain or disease. This procedure allows for complete relief of dorsal impingement. It increases dorsiflexion by decreasing the bulk of the joint and subsequently relieving dorsal impingement pain. It also eliminates the source of painful shoe pressure. The true advantage of the cheilectomy is that "no bridges are burned," and even in unsuccessful cases a salvage procedure is still technically possible.

The technique has been described and popularized by Mann and Clanton.²⁷ Their preference is a dorsal longitudinal incision centered over the hallux MTP joint. The joint capsule is incised on either side of the extensor hallucis longus (EHL) tendon and a complete synovectomy is performed. The joint is plantarflexed to permit inspection of the sesamoid articulation. Hamilton²⁸ recommends mobilizing the sesamoids by blunt dissection, for they often are anchored by adhesions and limit dorsiflexion even after removal of impinging osteophytes. The amount of bone to be removed from the MT head is dictated by the size of the dorsal exostosis and the degree of articular cartilage destruction. If degeneration of articular cartilage is not significant and the main problem is the dorsal exostosis, then 20% to 30% of the dorsal aspect of the MT head is removed along with the exostosis (Fig. 21.2).

It is reasonable to be relatively aggressive with this resection, removing up to one-third of the dorsal head to achieve improved motion. The cheilectomy should include removal of all osteophytes and a rounding of the MT head. The cheilectomy should achieve a minimum of 70 to 80 degrees of dorsiflexion, because approximately one-half of this will be lost in the postoperative period as a result of scar formation. It is Mann's recommendation that if insufficient dorsiflexion is achieved after cheilectomy, then a proximal phalangeal osteotomy (Moberg) should be performed as described later.

We have modified the cheilectomy technique through a medial approach. This allows for plantar debridement and release of plantar capsule and adhesions, thus improving dorsiflexion. In addition, the incision avoids the EHL tendon and the potential for tenodesis secondary to scar formation while still providing access to lateral osteophytes. We recommend a two-cut technique to avoid excessive resection of the MT head (Fig. 21.3). The first cut of the saw includes the dorsal exostosis and is made flush with the dorsal diaphysis. The subsequent cut removes the amount of articular surface necessary to achieve the desired dorsiflexion while eliminating the risk of excessive head removal that may jeopardize later arthrodesis.

Hamilton²⁸ describes a "radical cheilectomy," similar to the cheilectomy of Mann, but also removing the dorsal portion of the base of the proximal phalanx, matching the resection performed on the MT head. This modification serves as an option for dancers with end-stage disease and is similar to the Valenti^{29,30} procedure described later in this chapter.

A cheilectomy affords a fairly rapid postoperative course and return to activity. The patient is allowed to weight bear immediately, typically in a rigid-soled healing sandal. ROM can be initiated by a therapist or trainer as soon as pain allows but not so aggressively as to create wound dehiscence. Sutures generally are removed at 10 days, at which time active and passive

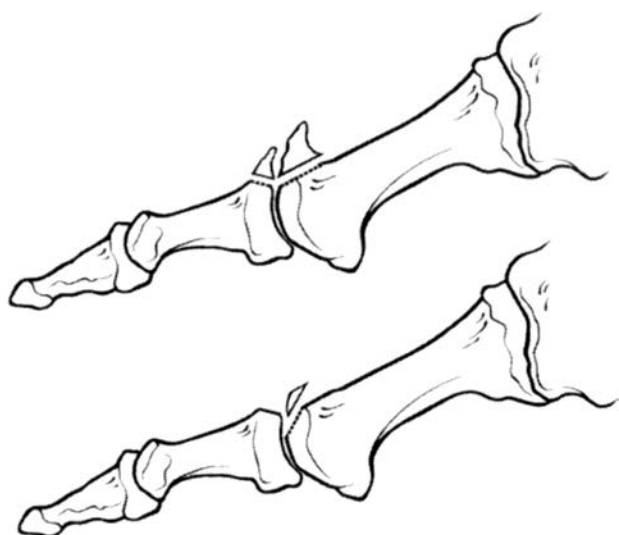


Fig. 21.3 The first cut of the saw includes the dorsal exostosis and is made flush with the dorsal diaphysis. The subsequent cut removes the amount of articular surface necessary to achieve the desired dorsiflexion while eliminating the risk of excessive head removal that may jeopardize later arthrodesis. (From Adelaar RS, ed: *Disorders of the great toe*, Rosemont, IL: American Academy of Orthopaedic Surgeons; 1997.)

ROM should be conducted at least three to four times per day. Close monitoring is required to ensure that the motion within the hallux MTP joint is at a functional level, a minimum of 40 degrees of dorsiflexion. No significant athletic activities generally are allowed for 6 to 8 weeks following a cheilectomy, giving the joint time to "mature" following surgery. Athletes can continue to train by bicycling, swimming, running in water, and engaging in other activities that avoid significant impact against the MTP joint. The patient should appreciate that swelling may continue for many months but that maximal motion usually is achieved by 3 months.

A number of authors have reported their results of cheilectomy. Mann and Clanton²⁷ found that 22 of 31 patients had complete relief, 6 of 31 achieved considerable relief, and ROM increased an average of 20 degrees in 23 of 31 feet. Hatstrup and Johnson³¹ reported that 53.4% were satisfactory and 27.6% unsatisfactory. Their failure rate increased from 15% with grade I radiographic changes to 37.5% with grade III changes. They concluded that cheilectomy is the procedure of choice in patients with hallux rigidus and grade I changes. Graves³² experience showed little improvement in motion and stated that satisfaction with cheilectomy was more likely if the patient and the physician had reasonable expectations regarding outcome. He recommended careful patient selection. Myerson agreed that the procedure improves pain, not motion. Easley et al.³³ reported on 57 patients (75 feet) with greater than 3-year follow-up (average 63 months). Their cheilectomy was performed via a medial approach by a single surgeon. American Orthopaedic Foot and Ankle Society (AOFAS) scores were 45 preoperative, 85 postoperative, and 90% satisfied. The average dorsiflexion improved from 19 degrees preoperative to 39 degrees postoperative. The majority of patients had worsening of radiographic arthritis, but this did not correlate with symptoms. Three patients eventually required an arthrodesis.

Phalangeal osteotomy has been advocated as a useful surgical adjunct to a cheilectomy. This technique was first proposed by Bonney and Macnab in 1952.¹⁴ Kessel and Bonney²⁰ described its use in 10 adolescents in 1958. Moberg is the name most commonly associated with the procedure, after his case series reported in 1979.³⁴ The procedure involves a dorsal closing wedge osteotomy of the proximal third of the proximal phalanx. It relies on the principle that the arc of motion of the hallux MTP joint is translated to plantar aspect of head, thereby increasing functional motion. Basically, it creates pseudodorsiflexion, which in turn places less stress on the hallux with push-off. Adequate plantarflexion of the joint is a prerequisite. Thomas and Smith³⁵ also found that the procedure appeared to provide dorsal joint space decompression, as well, further relieving stress from the arthritic joint (Fig. 21.4).

The indications for performing a Moberg osteotomy on the proximal phalanx includes grade I or II hallux rigidus, adolescent hallux rigidus, and the running athlete, perhaps regardless of grade. Most authors now recommend combining the procedure with a dorsal cheilectomy.^{33,35,36}

The technique can be performed through a medial or dorsal incision, extending distally from the incision used for the cheilectomy of the hallux MTP joint. It is important to protect the

dorsomedial and plantar medial cutaneous nerves to limit paresthesia and the potential for neuritis or neuroma. Longitudinal reflection of soft tissues at the proximal third of the phalanx is performed, maintaining capsular insertion. The FHL and EHL tendons are protected as a dorsal closing wedge osteotomy is performed with a microsagittal saw approximately 3 to 5 mm distal to the MTP joint. In the adolescent, it is necessary to avoid the physis. Intraoperative fluoroscopy can be useful in confirming proper position of the osteotomy. The plantar cortex is maintained to allow for a “greenstick” effect with manual closure of the osteotomy. Generally, 2 to 6 mm of dorsal cortex should be removed, with the actual amount determined by the degree of joint stiffness and amount of plantarflexion of the hallux available. The goal is to obtain 20 to 30 degrees of dorsiflexion relative to the first MT axis. The osteotomy should be stabilized with a suture, K-wire, screw, or staple. If combined with a cheilectomy, stable, internal fixation is mandatory to allow for the initiation of early motion (Fig. 21.5, A and B).

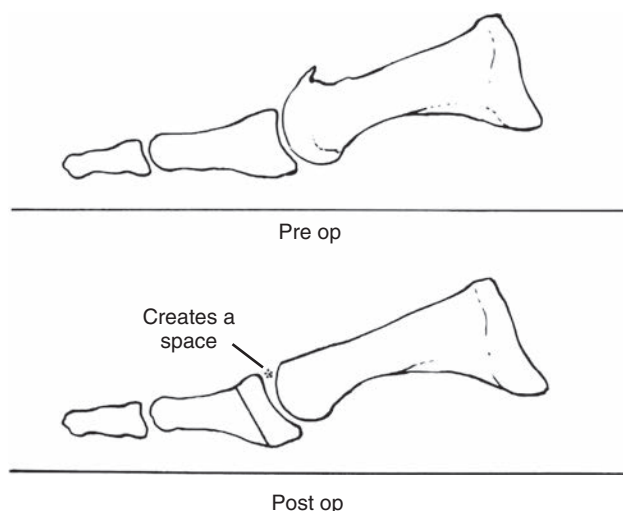


Fig. 21.4 Space created by dorsiflexion osteotomy of the proximal phalanx. (From Thomas PJ, Smith RWL: *Foot Ankle Int.* 1999;20:4.)

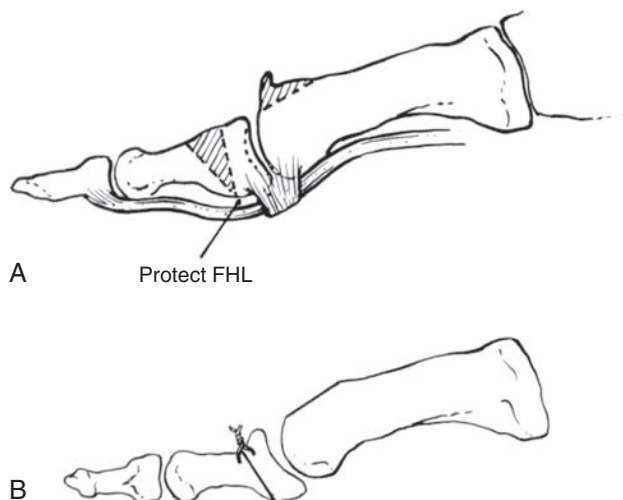


Fig. 21.5 (A) Dorsal cheilectomy and dorsiflexion osteotomy of the proximal phalanx. (B) The amount of correction after fixation. (From Thomas PJ, Smith RWL: *Foot Ankle Int.* 1999;20:4.)

The postoperative care is similar to that described for an isolated cheilectomy. Immediate full weight bearing is permitted in a hard-soled sandal, with passive dorsiflexion exercises begun at 1 to 2 weeks. In ranging the joint it is important to hold the entire toe as single unit. Plantarflexion exercises are delayed until 3 to 4 weeks postoperative. When a pin is present, removal is performed at 4 to 6 weeks, followed by transition to accommodative shoes.

Published results of the proximal phalanx osteotomy include Moberg's review of older individuals at short follow-up. Eight patients were noted to have satisfactory results. Citron and Neil³⁷ evaluated 10 feet in eight patients with 22-year follow-up (minimum 10 years) and identified five symptom free, others with progression of DJD, and one requiring arthrodesis. The average postoperative motion was 43 degrees, 22 degrees being dorsiflexion, with late loss of plantarflexion noted. Asymptomatic compensatory hallux IP flexion contracture often was present. They felt that this osteotomy represented an especially good option in the adolescent. Thomas and Smith³⁵ performed the osteotomy with a dorsal cheilectomy in 27 feet, 20 patients. At a follow-up average of 5.2 years, there was a 100% union rate, the average dorsiflexion increased 7 degrees, and 96% of patients were satisfied or satisfied with reservation.

Complications of the Moberg osteotomy include nonunion or malunion, a problem avoided by using internal fixation and “greensticking” the plantar cortex to avoid gross instability. Injury to the FHL and EHL tendons can occur, as can neuritis or neuroma, although the latter typically is transient. The possibility of progressive arthritis of the hallux MTP joint is an outcome that must be discussed with the patient preoperatively. Decreased push-off power can occur and may be of concern in the athlete or dancer.

Salvage for advanced degeneration or for a failed cheilectomy or osteotomy includes either arthrodesis or arthroplasty. Arthrodesis is best avoided in the “sprinting” athlete or dancer. If an arthrodesis must be performed, the toe tip should be at least 10 mm off the ground. Failure to meet this requirement will place significant stress on the distal hallux and IP joint. Slight shortening of the hallux also is of benefit, further lessening the potential of the athlete's having to “vault” over the hallux during running activity.

Resection arthroplasty, like that of a Keller, is reserved for the older individual. Capsular interposition is a modification of this procedure devised by Hamilton.^{38,39} In this procedure the proximal 5 to 10 mm of proximal phalanx is resected, followed by transection of the EHB tendon and dorsal capsule. This dorsal soft-tissue complex then is advanced to the plantar complex. Some authors release the FHB tendon from the base of the phalanx and suture this to the dorsal capsule. Temporary pin fixation is not necessary (Fig. 21.6, A and B). Our own experience with the procedure has noted good relief of pain from dorsal impingement and joint degeneration but a concerning loss of push-off strength. Similarly, the Valenti^{29,30} procedure is a salvage technique in which an angled resection on both sides of the joint is performed, preserving the plantar complex and overall length. The result is a “hinge” effect at the level of the joint (Fig. 21.7).

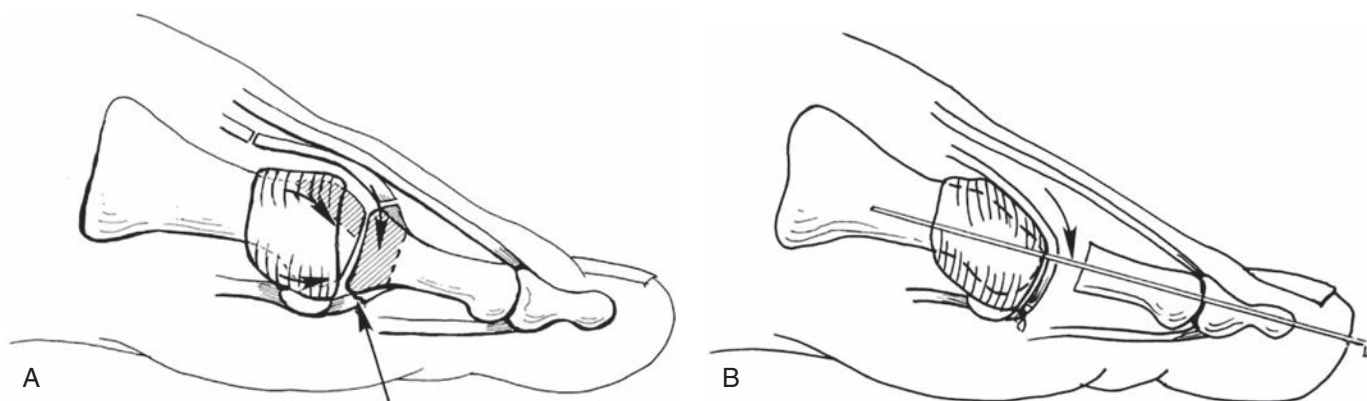


Fig. 21.6 (A) Interposition arthroplasty as described by Hamilton. (B) Pin fixation is not necessary. (From Hamilton WG, Hubbard CE: *Foot Ankle Clin.* 2000;5:663.)



Fig. 21.7 Resection of the dorsal metatarsal head as well as dorsal proximal phalanx. (From Coughlin MJ, Mann RA, eds: *Surgery of the foot and ankle*, 7th ed, St Louis: Mosby-Year Book; 1999.)

Soft-tissue interpositional arthroplasty of the hallux MTP joint has been performed in those individuals failing a cheilectomy but needing to maintain hallux MTP motion. Conical resection on both sides of the joint is followed by insertion of a semitendinosus allograft rolled into an “anchovy.” We have used this technique on a number of young athletic patients, including two professional football players, with good short-term results. Coughlin and Shurnas⁴⁰ recently reported on their experience with this technique in seven patients with excellent results. This case series demonstrates that this is a good surgical option in patients who otherwise would be treated with MTP arthrodesis. Our concern, however, is the amount of bone removed with this procedure and future implications in regard to arthrodesis. For this reason we have evolved to a modification of a technique described by Berlet et al., which employs a “parachute” dermal allograft coverage of the MT head and a mini-Keller/Valenti resection of the base of the proximal phalanx preserving the plantar plate.⁴¹

Implant arthroplasty has been advocated by some authors; the options described include a silastic double-stem hinge, titanium hemiarthroplasty, or total toe replacement. These implants are unlikely to hold up in the running athlete, and the surgeon is faced with a difficult revision should failure occur. It remains our recommendation to avoid this procedure in the athlete, career or recreational.

Because of the suboptimal results with difficult reconstructions following the above implant arthroplasty techniques, a small hydrogel spacer has been recommended for hallux rigidus grades II, III, or IV.^{42–45} This implant offers a more simple conversion to a hallux MTP fusion if it fails than others that require extensive resections. Mid-term results are promising;

however, in our experience it has not provided the expected outcomes such that we recommend it for the athlete.

Arthroscopic intervention for disorders of the hallux MTP joint has received some attention over recent years. It has been shown to be more of a diagnostic modality than a therapeutic one but may be a reasonable option for the removal of small dorsal osteophytes or loose bodies. It also may be used for debridement of an osteochondral defect on the MT head but is not indicated in advanced hallux rigidus. Van Dijk et al.⁴⁶ performed a prospective study with 24 athletes and found that it was not favorable for hallux rigidus because of scar fibrosis.

- X-rays: AP and lateral weight-bearing foot.
- Multiple etiologies, occult trauma or overuse most common.
- Large dorsal soft-tissue and/or bony mass.
- Dysesthesia in the dorsomedial cutaneous nerve can result from tight footwear impinging on the bony prominence.
- Nonoperative treatment: NSAIDs, large toe box shoes/balloon expansion, turf-toe plate, rocker bottom shoes.
- Surgical treatment: Cheilectomy if plantar joint space intact, Moberg phalanx osteotomy for running athletes, resection arthroplasty in elderly patients, and interpositional arthroplasty for complete joint destruction.

Sesamoid disorders

There are many etiologies for sesamoid pain. The general term “sesamoiditis” is best considered a term for a symptom rather than a diagnosis. This term implies pain in the sesamoid region with negative radiographs and an equivocal magnetic resonance imaging (MRI). It represents a diagnosis of exclusion in which soft-tissue ailments such as bursitis or flexor tendinitis are considered and often is associated with a history of overuse or trauma.^{47,48}

Fracture of the sesamoid, acute or stress, typically involves the tibial hallux sesamoid because of its larger size and greater propensity for weight-bearing forces. The classic radiographic appearance is a transverse fracture line, usually at the midwaist. It also can occur as the result of an MTP dislocation (Jahss Type II).⁶

Degenerative etiologies for pain in the sesamoid include chondromalacia, osteophytes, impingement, or plantar prominence. These particular problems may occur in an isolated fashion or in association with gout.

Osteochondrosis has an unknown etiology but often is found as a late sequela to a crush injury or stress fracture. Avascular necrosis (AVN) also has been described, most often affecting the fibular hallux sesamoid. Painful fragmentation and cyst formation with flattening of the sesamoid can be seen in either AVN or osteochondrosis, with radiographic changes following symptoms by 6 to 12 months.

Plantar prominence of a hallux sesamoid can occur with bursitis or with an intractable plantar keratosis (IPK). Osteomyelitis of the sesamoid can be the result of direct extension from a neuropathic ulcer or puncture wound but is unusual in the athlete.⁴⁹ Tumors of the sesamoid seldom occur but are considered more likely in the fibular side than the tibial.⁶

Diagnostic evaluation begins with a complete history of the problem. The typical patient will relate pain localized to the plantar hallux MTP joint with weight bearing, worsened with sports and stair climbing, and often with no precipitating event. The clinical examination identifies the specific location of pain and tenderness. Plantar medial signs relate to disorders of the tibial sesamoid, whereas direct plantar tenderness is indicative of fibular sesamoid pathology. In addition, the presence of swelling, warmth, and erythema should be documented. Joint motion and stability are assessed, noting restriction of motion secondary to pain or associated hallux rigidus. Vertical instability may follow a turf-toe or hyperextension injury. Sesamoid compression that produces pain and grind is consistent with metatarsosesamoid arthritis.

It is mandatory that the radiographic evaluation of sesamoid disorders include standing AP and lateral foot views and axial or tangential sesamoid views. These views are adequate in assessing for focal arthrosis, plantar osteophytes, or bony prominences. The tangential sesamoid view is helpful for identifying fractures of tibial sesamoid. It is helpful to always place a marker (B-B) on the skin overlying the site of tenderness. This simple maneuver helps to differentiate which sesamoid is symptomatic, or may help correlate a sesamoid location if there is a flexor tendon problem.

A question that often arises is the differentiation of a fracture versus a bipartite sesamoid. A fracture has sharp, irregular borders on both sides of the separation, whereas a bipartite has smooth, cortical edges and a relatively total size larger than that of a single sesamoid. Contralateral AP radiographs may be useful in this differentiation, as there is a reported 90% incidence of bilateral occurrence.⁵⁰

Further diagnostic studies useful in the evaluation of sesamoid disorders include MRI, which helps to localize pathology while differentiating between bone and soft-tissue abnormality. It further assesses sesamoid viability, joint degeneration, and tendon continuity. A readily available tool that is sensitive yet inexpensive is the bone scan. Although there is a reported high rate of false positives, a three-phase study with pinhole images helps to identify the problematic sesamoid. Computed tomography (CT) imaging can be performed to delineate the degree of metatarsosesamoid arthrosis or to assess fracture healing.

The nonoperative treatment of sesamoid disorders is general and begins with the principle of rest, ice, compression, elevation (RICE). Athletic activity and the training regimen are modified.

Analgesics and antiinflammatory medication are useful adjuncts. A boot or cast is applied for the first week in more severe injuries. The cast can include a toe spica extension with the joint in mild plantarflexion, removing stress from the plantarly positioned sesamoids. Weight bearing is permitted as tolerated. Taping of the hallux, as one would for a turf-toe, provides compression and limits movement. This is found to be most helpful in milder injuries. As the patient or player returns to athletic or recreational activity, orthoses and footwear modifications are mandatory. Off-the-shelf products, such as a Springlite turf-toe plate (Otto Bock, Minneapolis, MN) made of carbon fiber, in full or forefoot lengths, are useful in limiting dorsiflexion stresses. Custom-made devices can be fabricated with a Morton's extension to limit hallux MTP motion. A dancer's pad, MT pad, or arch support placed just proximal to the symptomatic sesamoid will assist in unloading weight-bearing pressures. Furthermore, the shoe itself can be stiffened with a plate incorporated into the sole. The patient should maintain low heel heights to minimize weight-bearing pressures. Turf shoes are modified by removing the cleat under the area of pain. Cortisone and/or anesthetic injections are not advised in any injury. An anesthetic injection alone may be used for localized pain in single-nerve distribution, but we would not completely anesthetize the toe or joint to enable an athlete to return to play.

Surgeries for disorders of the sesamoid are directed to the pathology identified. The first problem to consider is the IPK, attributable to the tibial hallux sesamoid. There are instances in which the plantar aspect of the sesamoid will develop a bony prominence, or osteophyte, and an overlying distinct callus will arise. This may occur in the presence of fat atrophy, and there may be an associated bursal component. Failure to improve with an orthosis to relieve pressure from this area may necessitate surgical decompression. The recommendation is for a plantar shaving of the tibial sesamoid via a plantar-medial approach. The periosteum overlying the sesamoid is reflected and the plantar 50% of the sesamoid is resected with a microsagittal saw. The FHL tendon is protected and the joint itself is not entered. The overlying soft tissues then are repaired so that the FHB tendon has been maintained in continuity, thus avoiding the risk of instability. The patient is allowed to weight bear to tolerance in the immediate postoperative period in a protective hard-sole boot or postoperative shoe. Return to regular footwear and activity is expected over the following 6 to 8 weeks as pain and swelling subside.

Fractures of the sesamoid can occur as acute events or can be stress induced. Acute fractures occur as a result of direct trauma, such as a forceful impact to the forefoot region. Because of its larger size and greater propensity for weight bearing, the tibial sesamoid is more likely to be involved.⁵⁰ These fractures generally heal with limitation of weight-bearing forces by use of such appliances as a cast (with a toe spica extension), boot, or postoperative shoe. There have been anecdotal reports of internally fixing these midwaist fractures with small, dual-pitched screws,⁵¹ but this is technically demanding and may not provide significant benefit over traditional treatment methods.

Stress fractures of the tibial hallux sesamoid have been noted to occur in athletes involved in repetitive-impact exercises, such as

long-distance running or aerobics. The diagnosis usually is made months after the onset of discomfort. By then the fracture likely has progressed to an established nonunion. Failure to improve the situation with orthoses designed to relieve pressure and limit excessive dorsiflexion through the joint may necessitate surgical intervention. Bone grafting of these tibial sesamoid nonunions has been performed successfully in an effort to avoid excision and the subsequent risk of losing push-off strength in the hallux.¹¹

Indications for this bone graft procedure include a mid-waist fracture location with minimal diastasis, preferably 1 to 2 mm. The articular surface of the sesamoid should be free of disease, and the two parts should not demonstrate gross motion between them. A plantar medial incision is centered at the hallux MTP joint. The capsule is incised along the superior border of the abductor hallucis tendon, and the joint is examined. Should there be cartilage damage on the sesamoid or gross motion between the two halves, then sesamoidectomy is completed. Otherwise, an extra-articular approach to the sesamoid is performed with reflection of overlying periosteum but preserving the FHB tendon. The fibrous material of the nonunion is curettaged back to viable bone surfaces. Care is taken to avoid disruption of the overlying articular surface. Through the capsulotomy, a window is made in the medial cortex of the MT head, and a small amount of cancellous bone is harvested. Alternatively, cancellous bone graft may be acquired through a small incision in the lateral calcaneus. This graft is packed into the nonunion defect created, and the overlying soft tissues are approximated with absorbable suture. There is no need for internal fixation because the two fragments should remain stable. The capsulotomy is repaired and the wound closed. Postoperatively, the patient is placed in a posterior splint with the distal portion enveloping the hallux itself. At 2 weeks the sutures are removed and a short-leg cast with a toe spica extension is applied. The patient is allowed to weight bear in such a device after 6 weeks, advancing to a shoe protected with a turf-toe plate at 8 weeks. A CT scan at 12 weeks should confirm union, and if accomplished, running is initiated with continued orthotic protection. We previously have reported on this technique in a series of 21 patients, 19 of which were successful.⁵²

Osteochondrosis of the sesamoid may occur with progressive fragmentation. This process may occur insidiously or as the sequela of a stress fracture nonunion⁴⁸ or osteonecrosis.^{53,54} Subchondral cysts may characterize early stages. Patients will present with chronic discomfort worsened by weight-bearing activity. Attempts can be made at nonoperative management using a period of rest and immobilization followed by orthotic management. However, a sesamoidectomy often is necessary in order for a return to recreational activities.^{48,53,54}

Sesamoidectomy is the only option for the surgical management of a number of sesamoid disorders, including osteochondrosis, osteomyelitis, advanced degeneration, or the rare tumor. A tibial hallux sesamoidectomy is achieved through a medial or plantar medial approach, avoiding the plantar medial digital nerve. The sesamoid can be excised from within the joint or extra-articularly. As discussed for nonunions of the sesamoid, it often is helpful to assess the articular surfaces before excision; this can be accomplished by entering the joint along the

superior border of the abductor hallucis tendon. By performing the excision through an extra-articular approach, the overlying FHB tendon can be repaired. A longitudinal incision and reflection of overlying soft tissues (subperiosteal) allows for full exposure of the sesamoid; the bone then can be shelled out circumferentially with a no. 69 Beaver blade. The defect then is repaired side to side with absorbable suture (i.e., 4-0 Vicryl). The surgeon must be aware of the proximity to the FHL tendon, protecting this structure during the dissection. Although rarely performed because of the risk of residual pain, partial sesamoid excisions can be considered if there is a small proximal or distal fragment. The abductor hallucis tendon can be transferred into large defects created by total excision of bipartite or fractured sesamoids. This transfer is performed by dissecting the distal tendon off the capsule at the base of the proximal phalanx. A fasciotomy is performed proximally to allow for rerouting of the tendon to the plantar aspect of the joint, where it is sutured into the defect with absorbable material. An adductor hallucis tenotomy should accompany this abductor transfer to remove the now unopposed valgus force. A concomitant bunionectomy should be considered if significant hallux valgus is present at the time of tibial hallux sesamoidectomy, because a progressive valgus deformity otherwise may develop.⁵⁵

When performing a fibular hallux sesamoidectomy, the decision must be made whether to approach from dorsal or plantar surface. A dorsal approach is difficult unless there is a large intermetatarsal angle with lateral subluxation of the sesamoid complex (i.e., bunion/hallux valgus). A longitudinal first webspace incision is used in performing a dorsal-based excision. Following superficial dissection, a laminar spreader placed between the MT heads is helpful. This approach requires the release of the adductor hallucis tendon and other lateral soft-tissue structures. The sesamoid is shelled out of the FHB tendon, taking care to avoid the neurovascular structures plantarly.

The plantar-based approach to fibular sesamoidectomy is preferable in that the soft-tissue structures balancing the hallux MTP joint are not disrupted. In this approach, a curvilinear incision is placed over the palpable fibular sesamoid, but just off of the weight-bearing pad of hallux MTP joint itself. It is necessary to identify and protect the plantar lateral digital nerve (Fig. 21.8, A and B). Following the sesamoidectomy, the reflected periosteum and FHB tendon (lateral head) are repaired. Skin closure must carefully approximate the dermal edges to minimize hypertrophic scar formation.

Postoperatively soft dressings are applied in such a manner as to maintain plantar flexion and either varus (tibial sesamoidectomy) or valgus (fibular sesamoidectomy). Weight bearing is allowed in a hard-soled sandal or short walker boot for a tibial sesamoidectomy, whereas nonweight-bearing or heel touch protection is recommended for a fibular sesamoidectomy performed through a plantar incision. With the latter, the patient is allowed to begin full weight bearing with the sutures in place at 2 weeks postoperatively. The sutures then are removed 1 week thereafter. Removable bunion splints help to maintain the desired hallux alignment between the second and sixth week. A gradual return to hard-soled shoes follows, using a turf-toe plate in athletic or training shoes.

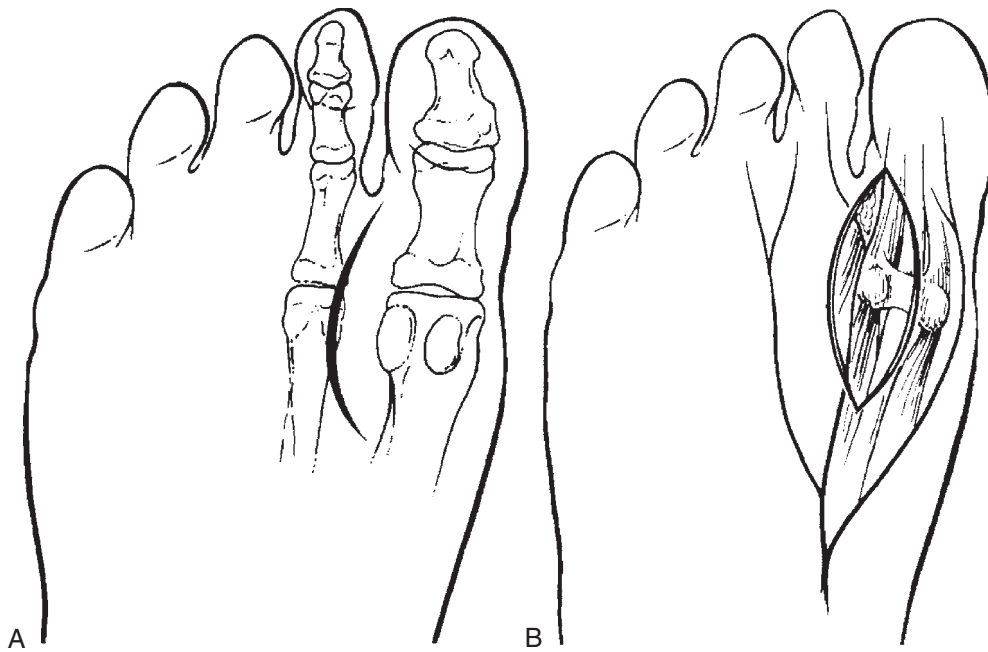


Fig. 21.8 (A) A curvilinear incision is made just lateral to the fibular sesamoid, just off the weight-bearing pad of the hallux metatarsophalangeal joint. (B) Care must be taken to identify and protect the plantar lateral digital nerve. (Drawn by Robert B. Anderson.)

The results of sesamoidectomy have been provided by a number of authors. Inge and Ferguson⁵⁶ reviewed 41 feet, 25 in which both sesamoids were excised. Complete pain relief was noted in 42%, whereas partial relief was noted in 82% with single sesamoid excision and in 64% of those in whom both sesamoids were excised. Leventen⁵⁷ found complete satisfaction in 18 of 23 sesamoidectomies. Mann et al.⁵⁸ identified 19 of his 21 sesamoidectomies “improved,” but only 50% had complete pain relief and 66% had full motion. In this group, 1 of 13 tibial sesamoidectomies developed hallux valgus, 1 of 8 fibular sesamoidectomies developed hallux varus, and 12 patients developed “weakness.” We assessed 12 patients who underwent a fibular sesamoidectomy via a plantar approach and identified 9 who were very satisfied and 2 who were satisfied. In addition, all would do it again, and 11 of 12 returned to preinjury activity level, citing no complications (for example, scar, neuroma).⁵⁹ Our results with isolated tibial sesamoidectomy were similar, with no loss of overall alignment or perceptible loss of push-off strength.⁶⁰

Sesamoidectomy is a good procedure that provides reliable results. The surgeon and patient must be aware that there is the potential for biomechanical implications such as the loss of push-off strength. This is especially important in the running athlete or elite dancer and must be discussed before intervening surgically.

- X-rays: AP and lateral weight-bearing foot, axial/tangential sesamoid views, skin marker over tenderness, contralateral views.
- MRI: differentiates soft-tissue from bone abnormality.
- Bone scan: high false-positive rate, use three-phase with pin-hole images to isolate problem area.
- Fractures: tibial sesamoid more common.

- AVN: fibular sesamoid more common.
- Nonoperative treatment: NSAIDs, rest, boot/cast in more severe injuries, turf-toe plate, arch support, and/or MT pad.
- Surgical treatment: varies depending on diagnosis.

Turf-toe

Since the term “turf-toe” was first used in the literature by Bowers and Martin⁶¹ in 1976, soft-tissue hyperextension injuries to the first MTP joint have received increasing attention from physicians, trainers, and athletes. Although these injuries have been grouped under the general heading of turf-toe, they actually represent a spectrum of injuries from the mild to the severe. In addition to the straight hyperextension injury of the first MTP joint, we now recognize there are variations that account for injury to specific anatomic structures in the capsular-ligamentous-sesamoid complex.

The true incidence of turf-toe injuries is difficult to quantify. At major universities, these injuries rank number three behind knee and ankle injuries.^{1, 62} When Coker et al.⁶³ looked at the Arkansas football players, they found ankle injuries to be four times more common than hallux MTP joint injuries; however, the latter were more severe, accounting for a disproportionate number of missed practices and games. Over a 3-year period, 18 of their players had a hallux MTP joint injury, equating to six turf-toe injuries per year. At Rice University, over a 14-year period, the average was 4.5 turf-toe injuries per year and included all sports.⁶⁴

The mechanism of injury can be direct or indirect and requires a basic knowledge of the mechanics required of the great toe during athletics. When an athlete rises on the ball of the foot for such activities as initiating a jump, blocking, or running, the hallux MTP joint extends upward of 100 degrees. As

the proximal phalanx extends, the sesamoids are drawn distally, and the more dorsal portion of the MT head articular surface bears most of the load. As this plantar complex attenuates or ruptures, unrestricted dorsiflexion can lead to impaction of the proximal phalanx on the dorsal articular surface of the MT head. This leads to a spectrum of joint injuries from partial tearing of the plantar structures to frank dislocation. The typical scenario leading to this injury in the athlete involves an axial load on a foot fixed in equinus. As an impact or force is placed on the heel, the forefoot progresses into dorsiflexion, creating hyperextension at the hallux MTP joint (Fig. 21.9).

However, not all turf-toe injuries are purely hyperextension. Numerous variations have been identified. For instance, a valgus component to the hyperextension of the hallux MTP joint results in injury to the plantarmedial ligamentous structures, occasionally to the tibial sesamoid, and the eventual development of a traumatic bunion with contracture of the lateral structures (Fig. 21.10). Douglas et al.⁶⁵ reported the case of a soccer player who sustained a hallux MTP joint injury when he was slide-tackled during practice. He continued to complain of joint instability and he failed conservative measures. MRI and operative findings were consistent with a medial collateral ligament tear, which was repaired.

Like valgus injuries, varus injuries also are rare. Mullis and Miller⁶⁶ reported on a basketball player with an injury to the hallux MTP joint 3 months before presentation. He had difficulty with running and was unable to return to sports participation. On physical examination, he was noted to have significant varus instability of the hallux MTP joint. Surgical findings included a torn conjoined tendon, lateral capsule, and lateral collateral ligament. The plantar structures were noted to be intact. All structures were repaired primarily, and the conjoined tendon was fixed to the base of the proximal phalanx through drill holes.

Over the years many theories have been investigated as causative factors in hallux MTP joint injuries. By far the two most common etiologic factors mentioned in the literature are the playing surface and flexibility of footwear. In a study by Rodeo et al.,⁶⁷ 80 active professional football players were surveyed, and of those with a turf-toe injury, 83% sustained the initial injury on artificial turf. Bowers and Martin⁶¹ addressed this

relationship by studying the impact of AstroTurf on the West Virginia University's football team. They coined the term "turf-toe" to describe injuries of the hallux MTP joint capsular-ligamentous complex sustained on artificial turf that previously had not been encountered on grass playing surfaces. The AstroTurf was alleged as a causative factor because of the hardness encountered with aging of the surface. However, Clanton and Ford¹ and others investigated the relationship of turf-toe injuries to aging artificial turf and found no significant correlation. In the three seasons preceding the replacement of the artificial turf in Rice Stadium, there were 13 turf-toe injuries, versus 12 injuries in the three seasons following replacement with a more modern synthetic playing surface. Nigg and Segesser⁶⁸ demonstrated an increased incidence of hallux MTP injuries on artificial turf and attributed this to the enhanced friction inherent in the surface. This may account for the forefoot's becoming fixed to the artificial surface with applied external forces, causing hyperextension and resulting hallux MTP injury.

Bowers and Martin,⁶¹ as well as Clanton and Ford,¹ have postulated that the shoe-surface interface most likely is responsible for these injuries. The majority of injuries are encountered on artificial turf in athletes wearing flexible, soccer-style shoes. The abandonment of the traditional grass shoe for the lighter, more flexible, soccer-style shoe seems to have been a major contributing factor in the evolution of the turf-toe problem. The trainers and physicians at Rice University could not recall a single instance of a severe MTP joint sprain occurring in a football player wearing the traditional grass shoe during the 25 years before 1986. This is most likely the result of the steel plate incorporated into the sole of the shoe for the attachment

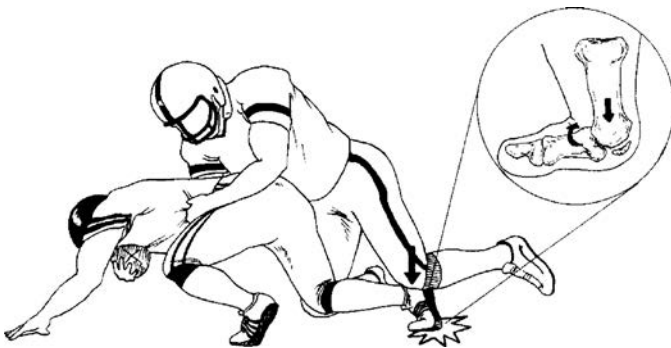


Fig. 21.9 An axial load applied to a foot fixed in equinus. As an impact or force is placed on the heel, the forefoot progresses into dorsiflexion, creating hyperextension at the hallux metatarsophalangeal joint. (From Adelaar RS, ed: *Disorders of the great toe*, Rosemont, IL: American Academy of Orthopaedic Surgeons; 1997.)



Fig. 21.10 Valgus component to the hyperextension causing injury to the plantarmedial structures, resulting in a traumatic bunion. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:693.)

of cleats, which has the secondary benefit of limiting forefoot motion.^{1,69,70} In the study by Rodeo et al.,⁶⁷ shoe type was not associated with turf-toe injury in professional football players. However, the number of players wearing traditional grass cleats in this study was small (15 out of 80) and perhaps influenced the outcome.

TABLE 21.1 Classification of Turf-Toe

Type of Injury	Grade	Description
Hyperextension (turf-toe)	I	Stretching of plantar complex Localized tenderness, minimal swelling, no ecchymosis
	II	Partial tear Diffuse tenderness, moderate swelling, ecchymosis, restricted movement with pain
	III	Frank tear Severe tenderness to palpation, marked swelling and ecchymosis, limited movement with pain, (+) vertical Lachman's if pain allows Possible associated injuries Medial/lateral injury Sesamoid fracture/bipartite diastasis Articular cartilage/subchondral bone bruise These may represent spontaneously reduced dislocations
Hyperflexion (sand toe)		Hyperflexion injury to hallux MTP or interphalangeal joint May also involve injury to additional MTP joints (lesser toes)
Dislocations	I	Dislocation of the hallux with the sesamoid No disruption of the intersesamoid ligament Frequently irreducible
	IIA	Associated disruption of intersesamoid ligament Usually reducible
	IIB	Associated transverse fracture of one or both of the sesamoids Usually reducible
	IIC	Complete disruption of intersesamoid ligament, fracture of one of the sesamoids Usually reducible

MTP, metatarsophalangeal.

Some authors have suggested that hallux MTP joint ROM may play a role in turf-toe injuries. Many studies have looked specifically at this factor and concluded that there is no relationship between hallux MTP joint ROM and subsequent turf-toe injury.^{1,62,67} However, there may exist a relationship between increased ankle ROM and turf-toe injuries. In the study by Rodeo et al.,⁶⁷ players with a turf-toe injury had mean ankle dorsiflexion of 13.3 degrees, versus 7.9 degrees for uninjured players, a statistically significant difference. It can be postulated that an increased ankle ROM places the hallux MTP joint at risk for hyperextension injuries. Still other causative factors contributing to turf-toe have been suggested. These include player position, weight, age, years of participation, pes planus, hallux IP DJD, and a flattened first MT head.^{1,62,67} The data for these variables are largely inconclusive, and it is unlikely that any of these factors play a significant role in the etiology of turf-toe.

Acute injuries to the hallux MTP joint have been classified into one of three general categories (Table 21.1).⁷¹ Hyperextension injuries usually can be differentiated from hyperflexion injuries by history and physical examination. The clinician should recognize that turf-toe constitutes a broad spectrum of injury with marked variability in the extent of soft-tissue involvement. To plan treatment and predict return to activity, a clinical classification system has been devised (Table 21.2). The mechanism for each of these injuries was discussed previously. At the extremes of hyperextension, frank dislocation of the hallux MTP joint can be seen.

To determine the extent of the injured structures in the hallux, the clinician must start by taking a history from the athlete. An exact determination of the events leading to the injury should be sought in each case. Reviewing the video of the game sometimes can aid in determining the mechanism. As with most athletic injuries, an examination of the involved extremity shortly after the injury is ideal. The examination should begin with observation of the hallux MTP joint for ecchymosis and swelling, with particular attention paid to the location (Fig. 21.11). Palpation of the dorsal capsule, medial and lateral collateral ligaments, and the plantar structures, including the sesamoid complex, should help the physician to elucidate the injured structures. The hallux MTP joint then can be placed through an ROM and compared with the opposite side. Abnormalities such as a mechanical block, hypermobility resulting from a plantar

TABLE 21.2 Clinical Classification System

Grade	Objective Findings	Activity Level	Treatment
I	Localized plantar or medial tenderness Minimal swelling No ecchymosis	Continued athletic participation	Symptomatic
II	More diffuse and intense tenderness Mild to moderate swelling Mild to moderate ecchymosis	Loss of playing time for 3–14 days	Walking boot and crutches as needed
III	Severe and diffuse tenderness Marked swelling Moderate to severe ecchymosis Range of motion painful and limited	Loss of playing time for at least 4–6 weeks	Long-term immobilization in boot or cast versus surgical repair



Fig. 21.11 (A) Plantar ecchymosis after injury to the plantar structures. (B) Medial ecchymosis after valgus injury.

plate tear, or gross instability can be appreciated. Varus and valgus stress testing then should be performed and also compared with the contralateral side. A dorsoplantar drawer test (Lachman) of the MTP joint will test the integrity of the plantar capsular-ligamentous complex. Plantarflexion and dorsiflexion of the hallux MTP joint against resistance should be performed to check the integrity of the flexor and extensor tendons of the hallux. In reality, this detailed examination can be difficult in the acutely injured athlete because of pain.

Following clinical evaluation, radiographic analysis is mandatory for all hyperextension injuries. In addition to the soft-tissue disruption, bony abnormalities may include capsular avulsions, sesamoid fractures, impaction fractures, diastasis of bipartite sesamoids, and proximal migration of the sesamoids. Recommended radiographs include a weight-bearing AP and lateral and a sesamoid axial view. A comparison AP view of the opposite foot is helpful. Prieskorn et al.⁷² found that patients with a complete plantar plate rupture had proximal migration of the sesamoids. The easiest way to evaluate the radiograph is to compare the distal aspect of the sesamoid-to-joint distance on the affected side with the unaffected side. The difference between sides should be within 3.0 mm (tibial) and 2.7 mm (fibular) 99.7% (3 SD) of the time. Looking at absolute numbers, if there was greater than 10.4 mm from the distal tip of the tibial sesamoid to the joint and greater than 13.3 mm from the distal tip of the fibular sesamoid to the joint, then there was a 99.7% chance of plantar plate rupture.

In addition to the standard views, special views and studies may be indicated, depending on a clinician's suspicion. Rodeo et al.⁷³ have suggested a forced dorsiflexion lateral view (Fig. 21.12, A and B), which may delineate joint subluxation, sesamoid migration, or separation of a bipartite sesamoid. Stress radiographs may help to define complete disruption of the

medial or lateral capsular-ligamentous complex. In addition, two oblique radiographs may be obtained. Other studies previously used in the diagnosis of turf-toe injuries include bone scintigraphy to rule out stress fractures or arthrography to document capsular tears. However, in our experience, MRI best defines soft-tissue injury and the presence of osseous and articular damage (Fig. 21.13). The use of a 1.5-Tesla MRI scanner (or 3T now if available) with paired 3-inch-round phased array surface coils can be used to obtain proton density and T2-weighted images. These images, obtained in the coronal, axial, and sagittal planes, provide anatomic detail of the nature and extent of soft-tissue injuries in acute turf-toe injuries.⁷⁴ We are liberal in performing this test because it assists in grading, identifies subtle injuries, provides timely decision making, and helps to formulate a prognosis.

The treatment of all grades of turf-toe injuries in early stages is similar.⁷⁵ Principles, which apply to most acute sprains of the musculoskeletal system, apply to the hallux MTP joint as well. Once the injury is recognized, immediate application of ice with a compressive-type dressing may aid in reducing swelling. Taping of the great toe in this acute stage is not recommended because swelling could lead to compromise of circulation. Clanton and Ford¹ suggest using the RICE formula of rest, ice, compression, and elevation. In addition, an NSAID may be prescribed. In some cases, a walker boot or a short-leg cast with a toe spica in slight plantarflexion may be helpful to alleviate symptoms during the first week (Fig. 21.14). Early joint motion may begin within 3 to 5 days after initial injury if symptoms permit. At this point, a severity grading must be applied so the athlete can be advised regarding prognosis and the time necessary for rehabilitation before a return to competition.⁷⁶

Athletes with a grade I injury usually are able to return to their sport with little or no loss of playing time. These athletes

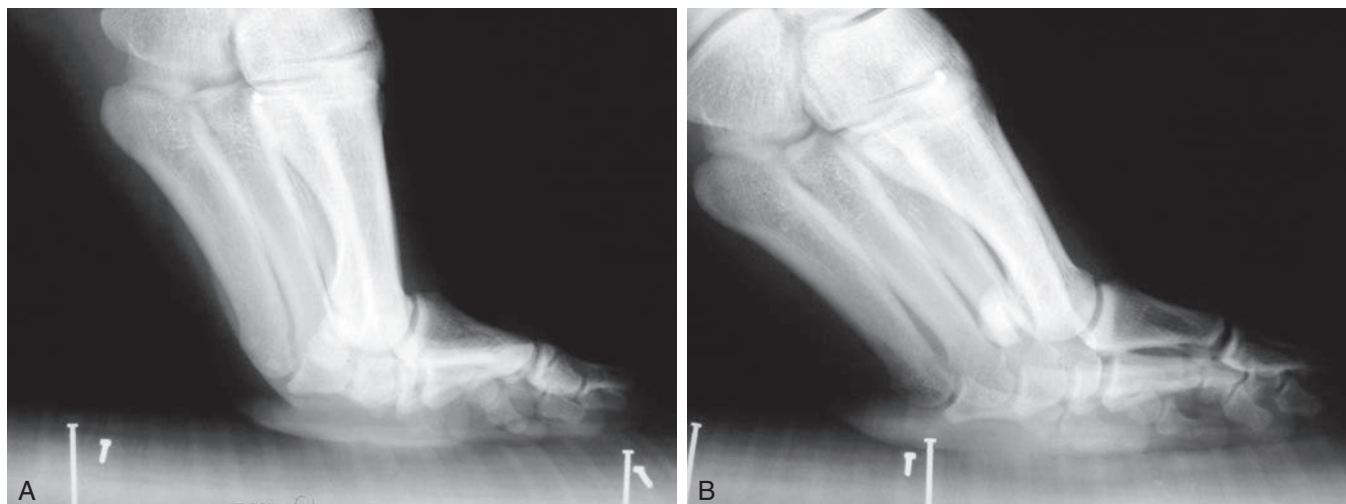


Fig. 21.12 (A) Normal dorsiflexion lateral. (B) Forced dorsiflexion lateral demonstrating proximal migration of the sesamoids.

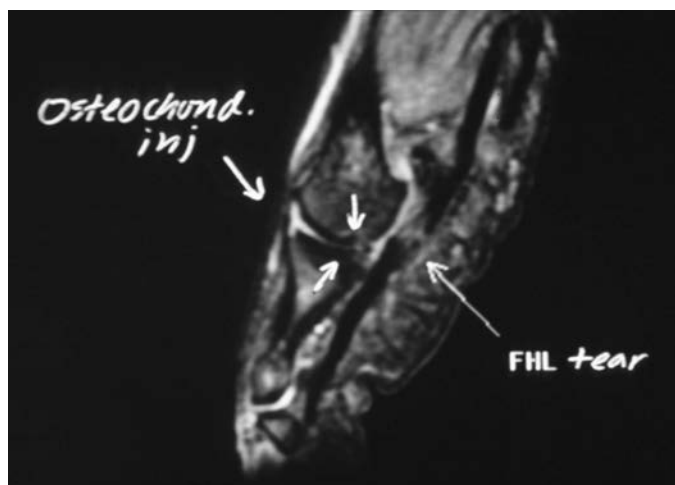


Fig. 21.13 Magnetic resonance imaging notes injury to bones and soft tissue. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:698.)

may benefit from taping of the great toe, as well as footwear modifications. The taping is designed to restrict hyperextension of the hallux MTP joint. Another technique used to restrict forefoot motion is the placement of an insole that includes a spring carbon-fiber steel plate in the forefoot region of the shoe. A custom insole with a Morton's extension may be better suited for the high-performance athlete but generally requires a longer shoe with a wider toe box. Factory-made turf-toe shoes are available that restrict forefoot bend, but most running athletes resist this treatment because of a perceived loss of mobility.

Grade II injuries usually result in loss of playing time ranging from 3 to 14 days, followed by the same modalities as mentioned previously. The grade III injuries may result in loss of playing time of at least 4 to 6 weeks, often requiring long-term immobilization and examinations weekly. In athletes who experience continued swelling and edema, modalities such as whirlpool and ultrasound with cold compression may be used as adjuncts to traditional therapy.⁷⁷ In general, return to play is dictated by symptoms, preferably with the athlete demonstrating 50 to



Fig. 21.14 Example of a short-leg walking cast with toe spica extension in slight plantarflexion. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:699.)

60 degrees of painless passive dorsiflexion. However, this return to athletics is individualized, dependent on the player's position, the level of discomfort, and healing potential.

There is a paucity of literature on the surgical management of hallux MTP joint injuries. This stems from the general notion that surgical management rarely is indicated in the treatment of this disorder. However, when an athlete fails to respond to conservative modalities, the treating physician should be suspicious for pathology that requires surgical intervention. Indications for surgery include a cartilage flap or loose body within the hallux MTP joint, acute sesamoid fracture, separation of a bipartite sesamoid, proximal migration of the sesamoids, evidence of gross instability resulting in persistent pain or synovitis, and hallux rigidus.

The study by Rodeo et al.⁷⁰ revealed that four athletes were noted to have diastasis of a bipartite tibial sesamoid and underwent excision of the distal fragment with repair of the capsule. One of these four athletes underwent acute excision, and the other three after failed conservative management. All of these players returned to their preinjury level of competition.

Our own experience in the repair or reconstruction of hyperextension injuries has been derived from a number of individuals who had sustained a turf-toe and subsequently were unable to perform athletically at their preinjury level. These athletes often complained of pain with running activity, along with the inability to cut from side to side. Clinical findings included malalignment of the hallux, traumatic and progressive bunion deformity, clawing of the great toe, diminished flexor strength, generalized joint synovitis, and advanced degeneration of the joint. Radiographic analysis often showed proximal migration of one or both sesamoids and cases of progressive diastasis of bipartite sesamoids (Fig. 21.15). MRI performed confirmed pathology through the plantar complex of this joint, often associated with injuries to the joint surface or FHL tendon. All the cases of proximal sesamoid migration associated with hyperextension injury have been associated with distal rupture. It appears that the sesamoids rupture distally and migrate proximally because of the preservation of the flexor tendons, along with the abductor and adductor tendons, and their ability to retract.

Our surgical experience with this injury has included 12 professional and collegiate athletes. Five surgeries were performed acutely for proximal migration or diastasis of a bipartite sesamoid, whereas seven were performed for chronic injuries, which included two traumatic bunions and one hallux varus deformity.⁷⁸

In the acute repair and reconstruction of these plantar complex injuries, exposure can be obtained through a medial, medial and plantar, or J-incision technique. Care is taken to avoid injury to the plantar medial digital nerve as it courses over the region at the tibial sesamoid. Plantarflexion of the joint can assist with plantar exposure of the joint. Once the defect has been identified in the plantar complex distal to the sesamoids, advancement and primary repair can be achieved with nonabsorbable sutures. Typically, sutures are placed into remnants of soft tissue on the base of the proximal phalanx. If found inadequate, then suture anchors or drill holes in the plantar lip of the proximal phalanx may be used. Fluoroscopic imaging should be utilized to confirm proper location of the anchor as excessive medial placement may create a supinated toe deformity. On rare occasions we have encountered a soft-tissue avulsion off of the distal pole of the tibial sesamoid. In this instance a transverse drill hole is placed and filled with a small nonabsorbable suture that is used to advance the sesamoid distal to the base of the phalanx. Fluoroscopic imaging, with comparison to preoperative images of the contralateral foot, ensure restoration of proper sesamoid position.

In cases of a progressive diastasis of a bipartite sesamoid, it is our recommendation to preserve one pole of the sesamoid only if that pole represents 75% of the entire sesamoid. Typically, the distal pole is excised and soft tissues are repaired through drill holes in the remaining proximal pole (Fig. 21.16). Should both poles of this sesamoid be fairly equal in size, degenerated or fragmented, then a complete sesamoidectomy is preferred. An MRI may assist in determining the extent of pathology within each pole. In these instances where the entire sesamoid is excised, a large soft-tissue defect will result leading to an incompetent FHB and potential loss of plantar restraints; we recommend that



Fig. 21.15 (A) Anterior-posterior (AP) radiographs of a professional football player following a turf-toe injury. Note the diastasis of the tibial sesamoid. (B) AP radiograph repeated 1 year later demonstrating progression of diastasis, which was associated with early clawing of the toe. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:701.)

an abductor hallucis tendon transfer be performed (Fig. 21.17). As previously mentioned, this transfer will act not only dynamically, helping to restore flexion power to the hallux, but also as a plantar restraint to dorsiflexion forces. It is accompanied by an adductor hallucis release via a separate dorsal first web space incision.

There are situations in which late reconstruction of these injuries is necessary, for example, when the athlete continues to perform despite injury or when the injury has been inadequately treated and protected. In these situations, the sesamoids may migrate well proximal, a problem often associated with hallux valgus, varus, or cock-up deformity. Reconstruction may include attempts at distal advancement of the sesamoids with soft-tissue reconstruction. This requires significant mobilization of the soft tissues proximal to the sesamoids, necessitating fasciotomies or fractional lengthenings of the FHB and abductor hallucis muscles. Joint debridement and cheilectomy may be necessary in cases of associated synovitis and osteochondral injury. Reconstruction of traumatic bunion deformities necessitates not only reconstruction of the plantar medial soft tissues but also a release of the lateral soft-tissue contractures.

The reconstruction of the claw toe deformity that occurs as a late sequela to hyperextension injuries is difficult. If the deformity is passively correctable at both the hallux MTP and IP joint levels, a flexor-to-extensor tendon transfer can be performed successfully. This transfer can be achieved either by splitting the flexor tendon and reapproximating dorsally into the extensor hood, as described by Girdlestone-Taylor, or by transferring directly through a drill hole into the base of the proximal phalanx (Fig. 21.18). This is our preferred method as it allows for use of an interference screw for secure fixation. Occasionally, a claw toe deformity will include a fixed contracture of the IP joint. This situation can be addressed and corrected by performing a concomitant hallux IP arthrodesis.

The postoperative management of athletes undergoing surgical reconstruction of hyperextension injuries is difficult because of the delicate balance between soft-tissue protection and early ROM. First, it is important to avoid placing the hallux in greater than 10 degrees of plantarflexion, either through surgical reconstruction techniques or with postoperative external immobilization modes. Excessive plantarflexion to this joint may become fixed and difficult to compensate for in the running athlete. Our

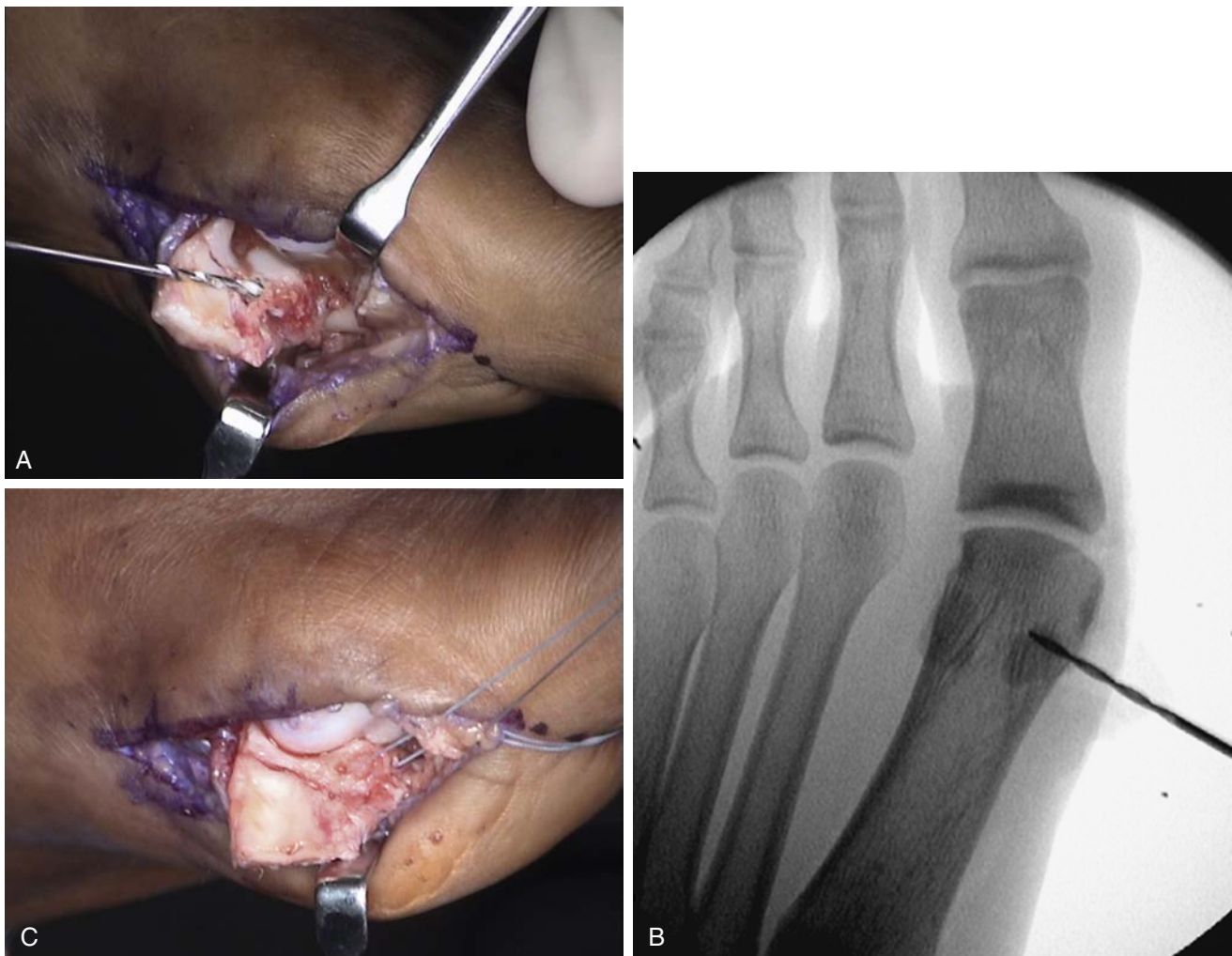


Fig. 21.16 (A) Avulsion injury with distal fragment excised. Small drill for transverse hole for fixation. (B) C-arm fluoroscopy of transverse orientation of drill. (C) Permanent suture passed through drill hole in distal sesamoid.

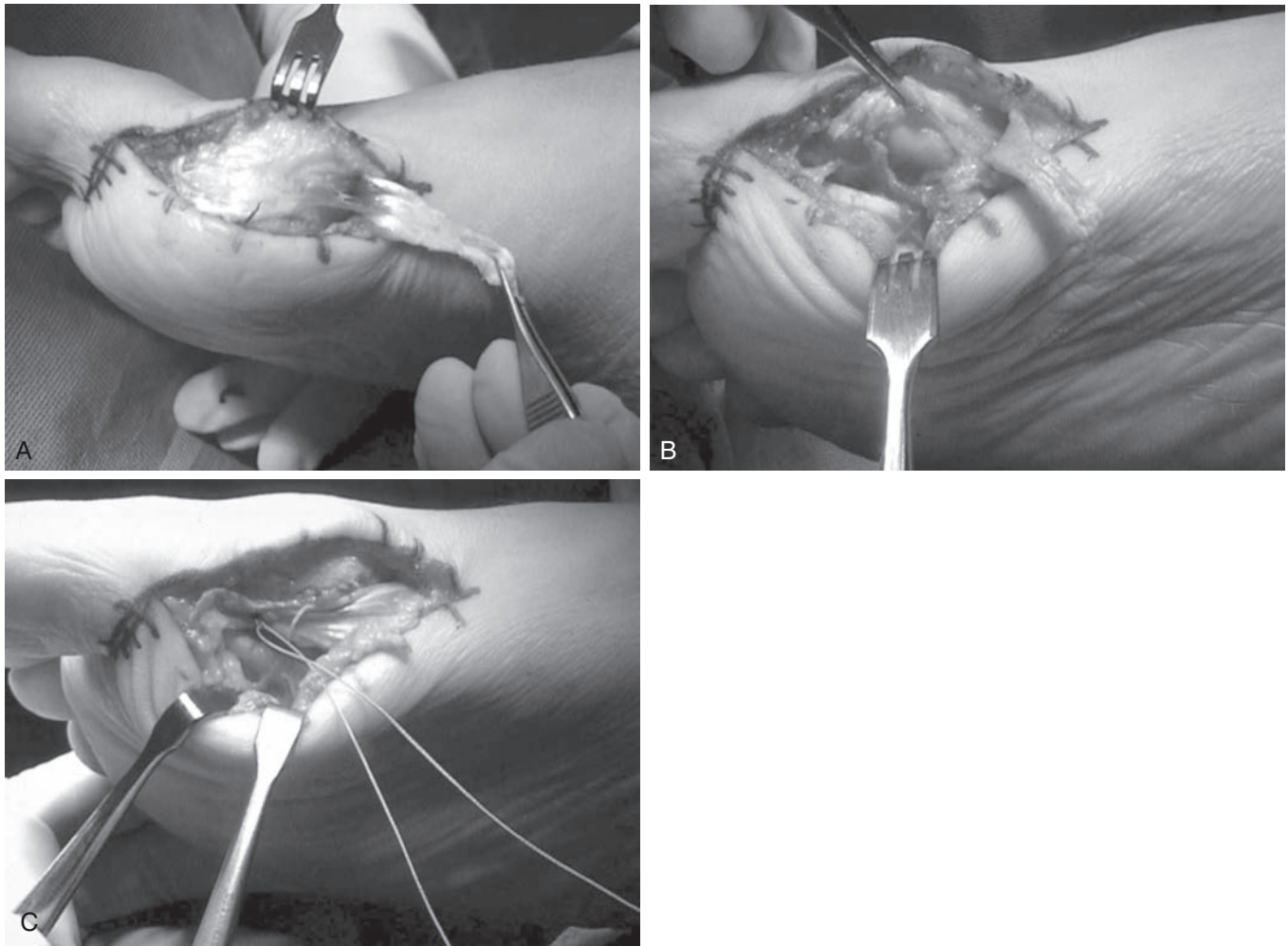


Fig. 21.17 Technique of abductor hallucis tendon transfer for reconstruction of hallux metatarsophalangeal joint. (A) Abductor hallucis tendon dissected from underlying capsule and immobilized proximally. (B) Plantar defect following sesamoid excision. (C) Transfer of abductor hallucis tendon completed with attachment to proximal phalanx. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:703.)

protocol includes external immobilization with a toe spica splint set in approximately 5 to 10 degrees of plantarflexion for a period of 5–7 days. At that time the athlete initiates protective, passive plantarflexion under the direct guidance of the athletic trainer or physical therapist. We avoid active and passive dorsiflexion and active plantarflexion maneuvers. When at rest, the toe is protected with a bunion splint using a plantar Velcro restraint and a removable posterior splint or cast boot. Nonweight-bearing ambulation is continued for a period of 4 weeks. ROM of the hallux is increased gradually at that time, along with protected ambulation in a cast boot. At 2 months postoperative, the patient is placed into an accommodative athletic shoe with the protection of an insole plate that limits dorsiflexion. Active ROM is instituted, with running by 3 months. At 4 months postoperative, the patient is allowed to return to contact activity with the continued protection of taping techniques and footwear modifications. We have found that it takes approximately 6 to 12 months before the athlete can perform at the preinjury level of function.

Late sequelae of turf-toe injuries may occur after conservative management or, less commonly, after surgical treatment has been rendered. Coker et al.⁶² reported on nine athletes who

had sustained a hyperextension injury. The most commonly reported late sequelae were joint stiffness and pain with athletic activity. Clanton et al.,⁶⁴ in their study of 20 athletes with turf-toe injury and 5 years of follow-up, noted a 50% incidence of persistent symptoms. Other late sequelae include cock-up deformity, hallux valgus, hallux rigidus, arthrofibrosis, loose bodies, and loss of push-off strength.

- Turf-toe constitutes a broad spectrum of injury with marked variability in the extent of soft-tissue involvement.
- Hyperextension injury to the plantar capsular-ligamentous-sesamoid complex.
- Can have varus or valgus component to injury pattern.
- Note ecchymosis, hypermobility, and varus/valgus on physical examination.
- X-rays: weight-bearing AP and lateral with contralateral views, sesamoid view, forced dorsiflexion lateral with contralateral view. Note sesamoid-to-joint distance.
- MRI: coronal, axial, and sagittal planes. May identify subtle injuries.
- Treatment: rest, ice, compression, elevation. Return to activity depends on severity of injury (see [Table 21.2](#)).

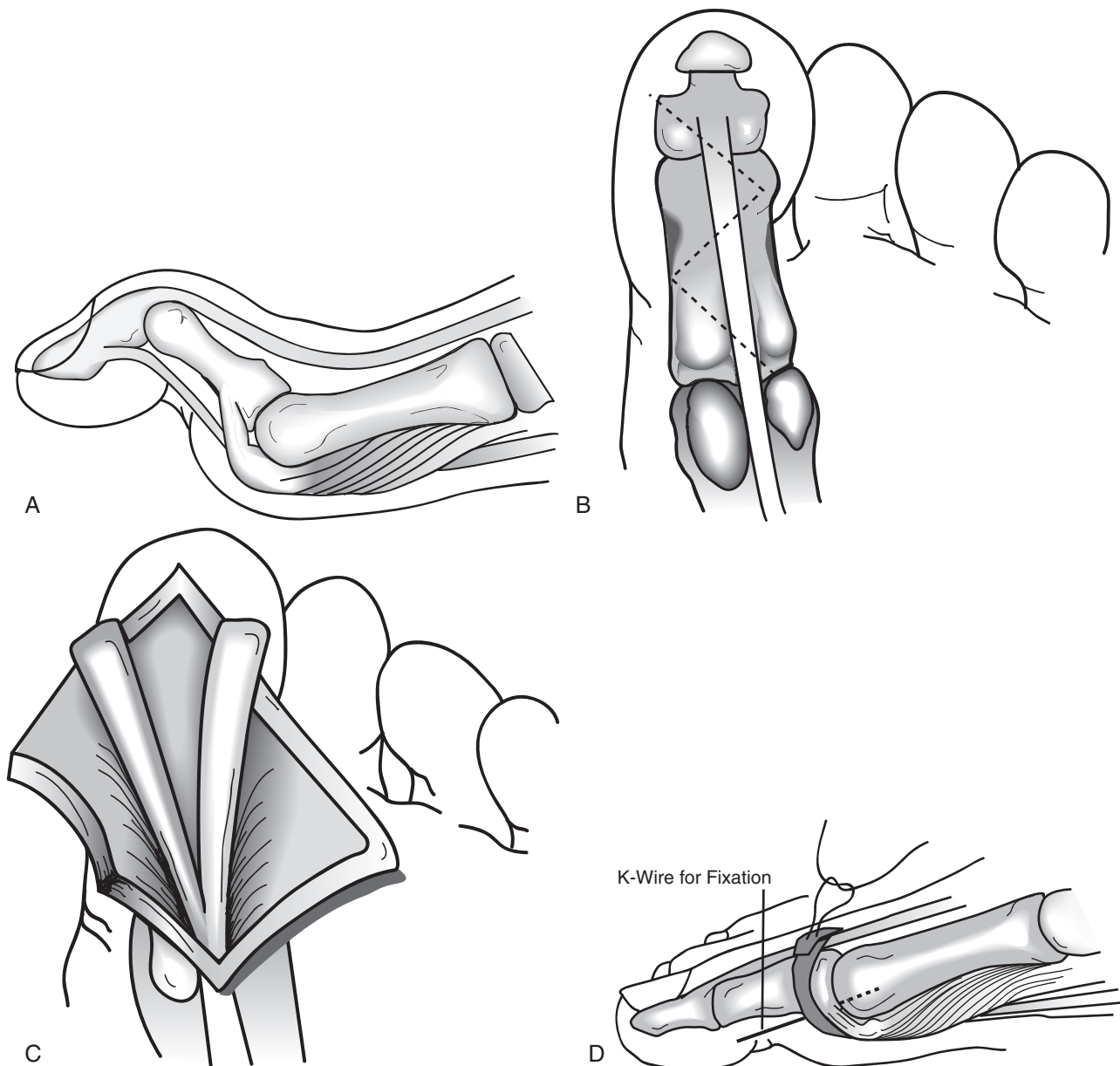


Fig. 21.18 (A through D) Technique for reconstruction of a claw-toe deformity that is passively correctable. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:706.)

- Shoe modifications and/or turf-toe insert to prevent hallux hyperextension.
- Surgical indications include a cartilage flap or loose body within the hallux MTP joint, sesamoid fracture, separation of a bipartite sesamoid, proximal migration of the sesamoids, evidence of gross instability resulting in persistent pain or synovitis, and hallux rigidus.

Dislocations of the Hallux MTP Joint

Frank dislocation of the hallux MTP joint most likely represents the extreme along the spectrum of hyperextension injuries. Dislocation in the dorsal direction is by far most common, yet plantar and lateral dislocations have been described. Jahss classified dislocation of the hallux MTP joint into two types.⁷⁹

In the type I dislocation, the MT head buttonholes through the weak capsular tissue proximal to the sesamoids. The distal

plantar plate, sesamoids, and intersesamoid ligament remain intact and attached to the phalanx distally. This intact complex comes to lie just dorsal to the MT head, with the FHB tendon dorsally translated. A closed reduction in the emergency department always should be attempted under local anesthesia. However, this injury typically is irreducible and requires an open reduction of the MTP joint through a dorsal approach.⁸⁰ If reduction cannot be obtained by reducing the sesamoids with an elevator, release of the adductor tendon and the deep transverse MT ligament or intersesamoid ligament may be required.⁸¹ If the joint is unstable after reduction, stabilization with a Kirschner wire is recommended; this can be removed after 3 to 4 weeks.⁸¹

Type II injuries are subclassified into types IIA and IIB (Fig. 21.19). In type IIA dislocations, the intersesamoid ligament is disrupted and radiographs reveal widening of the space between



Fig. 21.19 Dislocations. (A and B) Anterior-posterior (AP) and lateral radiograph of a type IIA hallux metatarsophalangeal (MTP) dislocation. (C and D) AP and lateral radiograph of a type IIB hallux MTP dislocation. (From Watson TS, Anderson RB, Davis WH: *Foot Ankle Clin.* 2000;5:710.)

sesamoids and dislocation of the MT head into or through the sesamoid split. Type IIB injuries produce a transverse fracture through one (usually tibial) or both sesamoids. In the situation of a single sesamoid fracture, the proximal fragment remains aligned with the intact sesamoid, and the distal fragment often becomes a loose body in the joint, usually requiring surgical removal. In addition to these types described by Jahss,⁶⁹ Copeland and Kanat⁸² defined a type IIC that is a combination of both IIA and IIB. The type IIC dislocation represents both a complete disruption of the intersesamoid ligament and a transverse fracture of either sesamoid (Table 21.3).

Differentiating between type I and type II dislocations is important because operative intervention typically is required for type I but not for type II dislocations. The general reduction maneuver is performed by placing gentle distraction with hyperextension on the MTP joint. If the joint is reducible, it typically is stable and is placed into a cast or hard-soled shoe for 3 to 4 weeks. A postreduction radiograph is required to confirm an anatomic reduction or to rule out the presence of any loose bodies.⁸³

TABLE 21.3 Radiographic Findings in Hallux Metatarsophalangeal Joint Dislocations

Dislocation Type	Radiographic Findings
I	No widening between sesamoids on AP view
IIA	Wide separation between sesamoids on AP view
IIB	Fracture of sesamoid (usually tibial)
IIC	Combination of type IIA and type IIB

AP, Anterior-posterior.

Occasionally, gross instability will follow a type II dislocation, particularly when a fracture of the sesamoid(s) has occurred. In this instance, the patient will experience pain with push-off and hallux rigidus type symptoms. A positive drawer sign is elicited, along with signs of generalized synovitis. Surgical correction in this setting includes plantar reconstruction to restore a restraint to dorsiflexion forces. Specifically, sesamoidectomy and abductor hallucis tendon transfer may be indicated. In the case of late

clawing, an FHL transfer should be considered, as described previously.

- Most extreme hyperextension injury with two main types (see Table 21.1).
- Type I: MT head buttonholes through intact plantar complex and likely irreducible. Surgical intervention to release blocks to reduction. Usually stable but may require K-wire fixation.
- Type II: three subtypes with injury to the plantar complex. Usually reducible, may require delayed reconstruction.

Hyperflexion Injuries of the Hallux MTP Joint

As stated previously, turf-toe injuries involve primarily a hyperextension injury to the hallux MTP joint with a possible varus or, more commonly, a valgus component. Rodeo et al.,⁶⁷ in their report on turf-toe injuries in professional football players, concluded that 12% of the players had a hyperflexion injury to this joint. Hyperflexion injuries clearly do not fit into the classification system for turf-toe. In fact, the mechanism and pathology are much different, and these injuries should not be grouped together.

Frey et al.⁸⁴ reported on a series of professional beach volleyball players with a hyperplantarflexion injury to the hallux MTP joint, an injury referred to as “sand toe.” This injury can result in significant functional disability noted with push-off, forward drive, running, and jumping. Although described in volleyball players, it also can be seen in football players, soccer players, and dancers.

The hyperflexion injury occurs when the weight of the body lands on a neutral or slightly plantarflexed hallux MTP joint. Frey et al.⁸⁴ reported on 12 volleyball players, 11 of whom had sustained an injury to the hallux MTP joint. The treatment for this injury mainly is conservative: taping, rest, ice, and NSAIDs. Once the inflammation has resolved, the athlete should undergo a rehabilitation program that includes strengthening of the intrinsic and extrinsic muscles of the foot. However, the time to recovery was, on average, 6 months (range 1–12 months). The most common problem after injury was loss of dorsiflexion of 25% to 50% at the hallux MTP joint, as well as residual pain. No toe deformities were noted. The authors attribute the loss of motion to capsular damage, synovitis, and arthrofibrosis. Whether or not arthroscopic debridement would benefit these athletes remains a question for future study.

- Different mechanism and injury pattern than turf-toe.
- Often referred to as “sand toe.”
- Treatment normally nonoperative with rest, ice, and NSAIDs.
- Rehabilitation after inflammation subsides with intrinsic/extrinsic strengthening.
- Motion loss of from 25% to 50% is common.

CONCLUSION

The great toe and its articulations are of paramount importance to the athlete. Great forces are transferred through this area with running, jumping, changing direction, and landing. Minor injuries can affect the ability even to walk or stand. A complete knowledge of the anatomy, forces involved, and treatment regimens are paramount when treating patients with these disorders.

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Bunions in the Elite Athlete: The Philosophy and Principles

Don Baxter

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INTRODUCTION

Sports medicine physicians treat many problems. One important area is the foot and ankle, with bunions being a common complaint. What do you do when an athlete comes to your office and says “My great toe hurts and has developed a prominence”? This chapter is not an explanation of bunions and all the various treatments. You can review those treatments in other textbooks and articles.

EXPERIENCE

What I want to do is share my experience of treating bunions, as well as secondary and tertiary problems, which develop as a result of bunions. I will also discuss other foot and ankle problems that support my philosophical approach to bunion treatment in the elite athlete. I don’t really care to review the literature, but rather prefer to outline my observations and outcomes of treating many elite athletes.

Between 1969 and 1974, all I saw was a bumpectomy, or a Keller bunionectomy during my internship and residency. In San Francisco I came under the influence of Henri DuVries, MD and Roger Mann, MD with a DuVries modification of the McBride procedure. I was also shown the Lapidus procedure where the tarso-metatarsal joint was fused along with the soft-tissue bunion reconstruction (McBride). Later Mann taught me his proximal first metatarsal (MT) osteotomy with a McBride procedure.

I came back to Houston, started jogging and running marathons during the running boom of the 1970s. Some of my friends and I started a running club in 1975, The Houston Harriers. I became the “go to” physician for joggers. I started a fellowship training program, and with my 35 fellows at University of Texas medical school and Baylor we started making observations and writing 27 peer review medical articles, many related to athletic injuries.

We looked at heel pain, bunions, Achilles tendon problems, as well as primary, secondary, and tertiary problems.

I learned the Chevron bunionectomy from Ken Johnson at the Mayo clinic in Arizona, later adding an occasional proximal phalanx osteotomy (Aiken). Bill Hamilton, MD in New York taught me about treating ballet dancers’ bunions conservatively.

PHILOSOPHY

The trick to medicine, as I see it, is to first take a complaint, read everything there is in the literature about that problem, and ask yourself if the recommended treatments can be improved with better outcomes.

My philosophy of foot and ankle surgery of athletes evolved. It is not the final answer. Your treatments will continue to evolve during the many years of your practice.

THE WORKUP

Take a history (see [Chapter 1](#)). Find out not only about static problems but more importantly the functional problems. What other problems exist? Where is the pain? When does it occur?

Are other problems primary or secondary?

Can the problems be corrected with shoe changes, orthoses, or medication or injections?

Is there a Morton’s foot or hypermobile first ray? Is there forefoot supination causing a hindfoot valgus?

For example, a lateral wedge was placed in a baseball player’s spikes who had genu varus. That year he led the majors in hitting, improving his back-foot stability.

For example, an Olympic gold medalist in the long jump sent me a Russian jumper, and friend, who had foot pain. I noticed he had a forefoot supination and a slight hallux valgus. We simply inserted a very lightweight Morton’s extension into his jumping shoe. Not only was the pain eliminated but he jumped 6 inches further, and almost beat my American friend and gold

medalist. The gold medalist later told me he did not plan on such a favorable outcome in his competitor!

In the early days there were one-piece shoes, and I experimented with pieces of felt glued into the insole.

Later, the shoe companies made shoes with removable insoles so corrections could be made on the removable liner.

X-RAY/MAGNETIC RESONANCE IMAGING

Look at a standing x-ray of the feet. Is the articulation of the bunion congruous or incongruous, and is the fibular sesamoid more than 50% uncovered by the 1st MT?

Is the first metatarsal rotated (Fig. 22.1)?

Occasionally, magnetic resonance imaging (MRI) is needed to review the tendons, ligaments, and articular cartilage. Is there an intra-osseous stress fracture? What is the status of the sesamoids? Is there a volar plate injury?

PLAN TREATMENT

Ask yourself if the bunion is compensated or decompensated (Figs. 22.2 and 22.3). If the bunion is compensated, such that no pain occurs with appropriate shoes and orthosis during athletic activity, then no surgery should be done. The dancer can dance, the runner run, the jumper jump, and the pitcher pitch. Now if the bunion is decompensated, with severe pain, unrelieved by shoes or orthosis, or requires injections to play, then the decompensated bunion must be made compensated. In other words, restore function. Don't try to make the anatomy perfect, but rather restore the function as it was before the decompensation occurred.

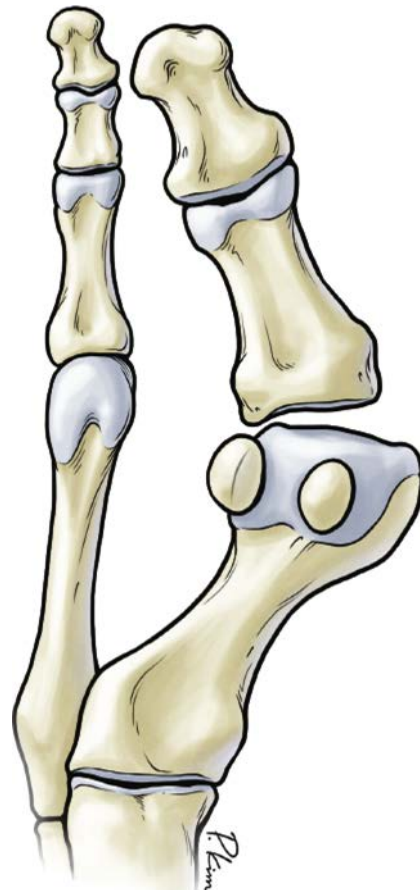


Fig. 22.1 With a decompensated bunion the joint is incongruous, and the fibular sesamoid is more than 50% uncovered by the first metatarsal head.

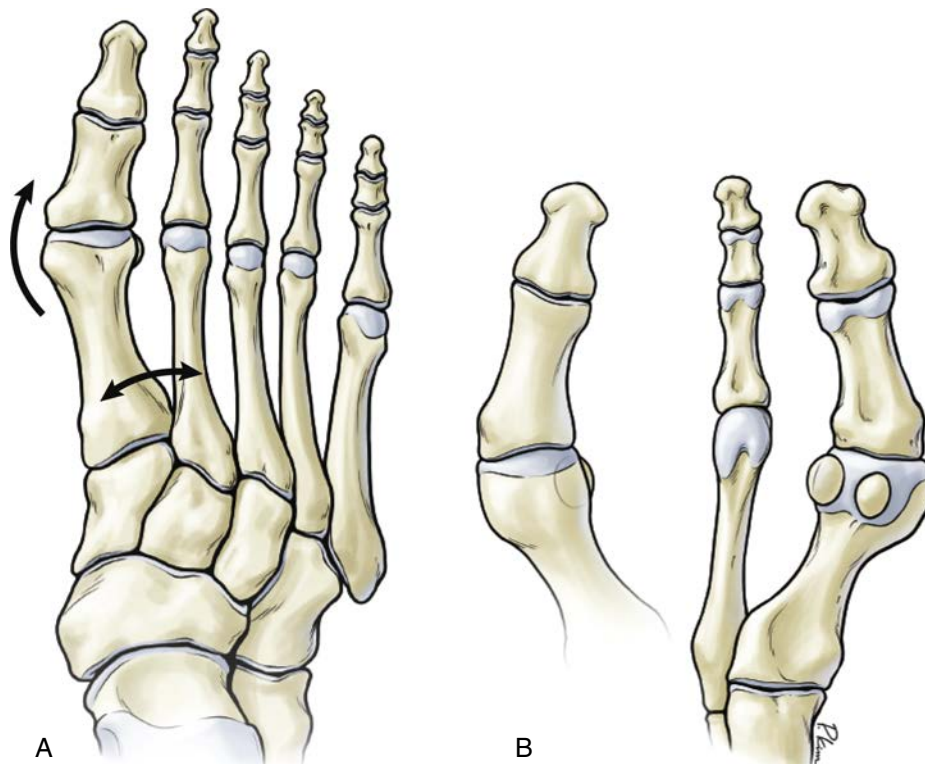


Fig. 22.2 (A) The compensated bunion has a hallux valgus that is usually less than 20 degrees and an intermetatarsal angle less than 11 degrees. The articular facet is congruous. (B) With a compensated bunion the articular facet is congruous, and the fibular sesamoid is less than 50% uncovered by the first metatarsal head.

In the plan, how can that be done without negatively affecting function? Sometimes restoration of the anatomic alignment is the enemy of better function, especially in the elite athlete. Roger Mann in the 2nd edition of this book discussed at length congruous and incongruous great toe joints (Fig. 22.4).

Often a congruent joint bunion is not symptomatic. In the elite athlete, footwear and lightweight flexible orthoses can be prescribed, but no surgery should be done.

Roger Mann has recommended, with incongruous, painful bunions, restoration of the anatomy fully to avoid re-occurrence (Fig. 22.5). I agree with Roger Mann in the nonelite athlete, but, as I will describe, I approach the elite athlete differently.

PATIENT

Let me tell you about an elite runner who I saw in 1983. This young runner had been to two Olympics as a middle-distance runner. Her foot had a severe bunion that had become decompensated, incongruent, and there was a secondary painful neuroma between the second and third MTs. Daily injections of numbing agents were needed to enable her to run.

I feared that a perfect realignment might adversely affect her function. So, after discussing with the patient, we decided to remove the neuroma, and do a chevron procedure on the bunion, making the decompensated incongruent bunion a compensated congruent bunion. Thirty-five years later, the bunion has progressed. It is unsightly and affects footwear. The thought now becomes that the operation was a failure. Recently, I called her and asked how she was doing.

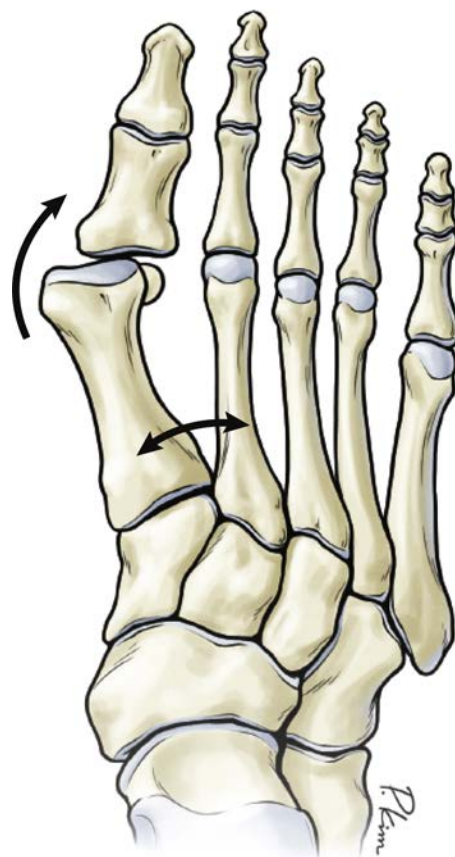


Fig. 22.3 The decompensated bunion usually has more than 25 degrees hallux valgus and an intermetatarsal angle more than 12 degrees. The joint is incongruous.



A



B

Fig. 22.4 (A) Radiograph of a congruent metatarsophalangeal joint. There is no lateral subluxation of the proximal phalanx on the metatarsal head. This maintains the function of the plantar aponeurosis and hence the stability of the foot. (B) An incongruent metatarsophalangeal joint has lateral subluxation of the proximal phalanx on the metatarsal head. This creates an unstable situation, progressive in nature, giving rise to decreased function of the plantar aponeurosis and increasing instability of the medial longitudinal arch.



Fig. 22.5 Preoperative and postoperative radiograph demonstrating correction following a distal soft-tissue procedure and proximal metatarsal osteotomy.

She said, “my bunion has returned, but there is no pain. I went to two additional Olympics and set an American record for 10 km on the road after the bunionectomy.” She was totally happy with what I did in 1983.

In 20 years, I never did a bunionectomy in an active ballet dancer. I have done bunionectomies in dancers after their career has been completed.

There are some philosophical thoughts I have in elite athletes: 1) be conservative; 2) be a minimalist; 3) don't be a perfectionist, but a functionalist; 4) always make the decision regarding surgery with other experts and the athlete (see [Chapter 30](#)); 5) each athlete is an outcome study regarding the athletes' preinjury performance and his/her postinjury performance.

My current approach to elite athletes with bunions is to treat the symptoms conservatively, with shoe changes and orthotic foot control. This takes care of 85% of bunions that are compensated. Fifteen percent become decompensated, so I do either a chevron or occasionally a Chevron-Akin procedure. I have not done a proximal osteotomy or a Lapidus fusion in the active elite athlete.

There is definitely a place for the more definitive bunionectomy. There is a current trend to do a derotational fusion of the tarso-MT joint. This Lapiplasty procedure perfectly realigns the sesamoids and the rotation of the first MT. It perfectly realigns the 1–2 MT angle. It is a very good procedure for a severe bunion.

Jim Nunley, MD feels there is a place for this procedure in the elite athlete, but the lapiplasty procedure has not been done in the elite athlete as of 2018, Lew Shon is concerned that a stress fracture might develop as a result of the biomechanical changes or synovitis and arthritis in adjacent joints. David Porter, MD PhD and Bob Anderson, MD do not do fusions in the elite athlete.

Possibly in our next edition more outcomes may be reported.

I feel most problems in elite athletes are biomechanical. The thousands of training miles accentuate and break down the weakest link.

Years ago, I had an American and world record holder in my office. She had a long foot with a slight forefoot supination. I suggested a Morton's extension thinking it might avoid secondary and tertiary problems. Over the years four major problems occurred, all from this forefoot supination. **The first** was a medial insertional achilles tendinosis, which took months to resolve. **The second** was a medial plantar fasciitis, with entrapment of the first branch of the lateral plantar nerve. This required releasing the nerve 2 months before the olympic trials. She missed qualifying for the national team by a few hundredths of a second. **The third** was a stress fracture of the fibula a month before the world games. Her coach and I let her run in a limited manner until the games. With a healing stress fracture of the fibula, this world-class athlete won the biggest race of her life beating the Russians as they dove for her at the finish. **The fourth** was the worst, a rupture of the posterior tibial tendon. Even with repair, her long successful career came to an end. I always wondered if a proper shoe, and Morton's extension in the shoe, could have avoided all of these problems. The most likely problem with a forefoot supination would be a hallux valgus.

But everything broke down because of the forefoot supination except the great toe. I also wonder if making the great toe perfectly aligned, such as with a first tarso-MT fusion, might affect other structures such as with this elite athlete.

These are all concerns for the next generation of foot surgeons.

HALLUS RIGIDUS

Occasionally, bunions in the elite athlete have a rigid element. Restricted dorsiflexion may affect function. One athlete, who had won the national cross-country three times, became affected by a rigid, painful hallux rigidus. His bunion was minimal. One of four of the dorsal first MT was removed. He returned to be a national-level runner, and an olympian.

REHABILITATION

All athletes are different as to their rehab. Most are highly motivated. My regimen is to have the athlete stay nonweight bearing for 2–4 days, use a postop shoe, and start a stationary bike on the fifth postop day, only using the heel to pedal. Minimal resistance is used for 3 days, and then progressive resistance. Forefoot pressure is not used for 3 weeks on the stationary bike. At 4 weeks a loose athletic shoe may be worn with a toe spacer. The toe spacer is worn until 8 weeks postop when easy running can be resumed. Unlimited training is resumed at 12 weeks. The hallux rigidus cheilectomy athlete resumes running by 3 weeks. But some variation is necessary for each athlete depending on symptoms and healing. A lightweight orthosis is usually prescribed to eliminate any minor biomechanical imbalances.

OUTCOME

When the runner or jumper returns to his or her preinjury times or heights, a good outcome is achieved. When the elite

athlete surpasses his or her personal best or sets a record, you have a great outcome.

That is what the treatment of elite athletes is all about—great outcomes, not perfection!

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Treace Medical Concepts-lapiplasty Treace Medical Concepts, Inc. 203 Fort Wade Road, Suite 150 Ponte Vedra, FL 32081 www.treace.com/lapiplasty.

Chronic Leg Pain

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INTRODUCTION

Orthopaedic surgeons continue to see an increase in the incidence of sports-related injuries with much of the population becoming active in more strenuous activity. Nonspecific complaints of pain in the foot, ankle, calf, or shin are often reported, with shin pain as the most common presentation.^{1,2} Evaluation of leg pain requires not only knowledge of the anatomy and biomechanics of the lower extremity but also an understanding of the pathology of the injury. Conducting a thorough history and physical examination and appropriately interpreting diagnostic tests are essential to the establishment of an accurate diagnosis. In addition, specific details regarding physical activity, including training regimens, surface conditions, and footwear, must be determined because these factors also play a significant role in the diagnosis.

Because several etiologies may present with similar characteristics, patients must be evaluated for multiple conditions.³ The differential diagnosis of chronic leg pain includes the following conditions: bony or soft-tissue tumors, chronic exertional compartment syndrome (CECS), claudication, isolated leg trauma, medial tibial stress syndrome (MTSS), muscle strains, nerve entrapment, popliteal artery entrapment syndrome (PAES), radiculopathy, referred pain from meniscal pathology, stress fractures, and tendinitis. Despite this wide range of diagnoses, several studies demonstrate that certain conditions are more prevalent among athletes, in particular.^{1,4–6} There have been reports in the literature of exertional leg pain

occurring in 12.8%–82.4% of athletes.⁷ In a previous retrospective study of 150 patients with exercise-induced leg pain, CECS was the most prevalent cause of pain, representing 33% of cases; stress fractures and MTSS accounted for 25% and 13% of cases, respectively.⁶ More recently, however, studies have shown MTSS to be the most common cause of chronic leg pain in the athlete, accounting for 13%–22% of injuries in runners and dancers, 35% of injuries in Naval recruits, and 5% of all athletic injuries.⁸ This chapter focuses on the most common causes of chronic leg pain in athletes, including MTSS, stress fractures, CECS, nerve entrapment, and PAES. The incidence, pathology, clinical presentation, and treatment options are discussed for each condition.

MEDIAL TIBIAL STRESS SYNDROME

“Shin splits” is a nonspecific diagnosis of posteromedial leg pain commonly used to describe a wide variety of lower leg pain conditions, including chronic ECS, fascial hernia, muscle strains, periostitis, and stress fractures.^{2,3,5,9–11} One of the most common sites of overuse pain is the distal one-third of the medial border of the tibia.^{11–14} Because of this, MTSS may be the most accurate term for this because it describes both the location and the likely pathophysiology of the syndrome.^{5,13–15}

MTSS typically is observed in runners and individuals involved in jumping activities such as basketball, volleyball, and dancers.^{3,10,13,16–18} Both biologic and biomechanical factors have been reported as possible causes of MTSS.^{12–14} Although

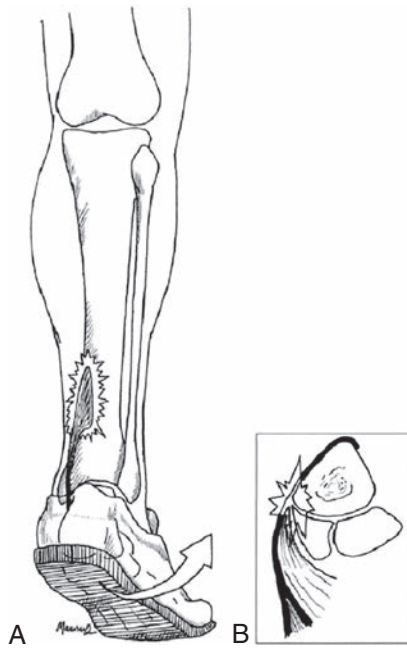


Fig. 23.1 (A) During running, the medial portion of the soleus contracts eccentrically as the foot pronates. Hyperpronating athletes, in particular, are at an increased risk for developing medial tibial stress syndrome (MTSS). (B) The source of pain is at the origin of the flexor digitorum longus (FDL) and soleus fascial bridge on the posteromedial aspect of the tibia.

the tibialis posterior muscle historically has been implicated as the source of this condition,^{11,18–21} a study of 50 cadaveric legs revealed that the tibial posterior muscle was more lateral, indicating that this muscle was not a likely source of MTSS.¹² Other studies have identified the soleus, flexor digitorum longus (FDL), and crural fascia as sources of the pain, with more focus on the soleus.^{12,22,23} Another theory suggests that the pain is due to bony overload of the tibia due to a bending stress produced with weight bearing.^{24–26}

During running, heel strike occurs in relative supination, with pronation of the foot increasing until midstance.^{13,27,28} Because the soleus is the primary plantarflexor and inverter of the foot, it has been theorized that the medial portion of this muscle contracts eccentrically as the foot pronates (Fig. 23.1).²⁷ The repetitive eccentric contraction that occurs in hyperpronating athletes may explain the increased incidence of MTSS observed in such athletes.^{5,11,13,23,27,29–32} In addition, hyperpronation is a compensatory mechanism that occur in patients with hindfoot and forefoot varus,^{23,28} tibia vara,²⁸ tight Achilles tendon,^{28,31,32} and tight gastrocnemius and soleus muscles,²⁸ therefore such patients also are at increased risk for developing MTSS. Other risk factors for MTSS include female sex, increased weight, higher navicular drop, previous running injury, and greater hip external rotation with the hip in flexion.³³

History

The most common complaint associated with MTSS is a recurring, dull ache localized over the distal one-third posteromedial cortex of the tibia (Case Study 23.1). MTSS tends to occur late in the sport season after prolonged activity, whereas stress

fractures tend to occur early in an athletic season as stresses increase rapidly. Early in the development of MTSS, patients may experience pain at the beginning of a workout or run but feel a relief of symptoms with continued activity, only to be followed by a recurrence of pain either at the conclusion of the activity or some time afterward. Pain usually is alleviated with rest and generally does not occur at night. However, as this condition progresses, pain may occur throughout training or during low activity, such as walking, and possibly may continue during rest.

Physical Examination

The pathognomonic physical finding in MTSS is palpable tenderness along the posteromedial edge of the distal one-third of the tibia. In rare cases, erythema or localized swelling over the medial tibia also may be observed. Although studies have reported conflicting ranges of motion associated with MTSS, in theory, hypermobile pronating feet are at increased risk of MTSS. Therefore evaluation for foot pronation or subtalar varus also is recommended. Abnormal pulse, diffuse swelling, firm compartments, neurologic deficits, and vibratory pain are not associated with this syndrome.

Diagnostic Studies

Roentgenograms generally are normal in patients with MTSS^{3,9,13,27,34,35} but are recommended to rule out abnormalities associated with other conditions such as stress fractures and tumors.^{3,13,14} A three-phase bone scan is warranted to rule out stress fractures if a conservative treatment program does not alleviate pain. This type of bone scan is a valuable diagnostic tool used to differentiate between MTSS and stress fractures, because each condition has a distinct scintigraphic pattern.^{9,11,13,22,27,36,37} A bone scan demonstrating a longitudinal and diffuse pattern in the distal one-third of the tibia is indicative of MTSS (Fig. 23.2).^{11,13,22,37} In general, only delayed images are positive in cases of MTSS, whereas both early and delayed images demonstrate uptake in cases of stress fracture.^{13,22} In addition, magnetic resonance imaging (MRI) is another diagnostic tool for MTSS. MRI will show periosteal reaction and bony edema with a sensitivity of 78%–89% and specificity of 33%–100%.⁸

Treatment

Conservative

The recommended management of MTSS is multimodal, consisting of rest, nonsteroidal antiinflammatory drugs (NSAIDs), and ice.^{3,14} Physical therapy modalities such as iontophoresis and ultrasound also may be used.^{3,14} Initially, rest or a decrease in training for 2 to 3 weeks is suggested and may be curative without further workup.¹⁴ Cardiovascular conditioning may be maintained during this period with swimming, upper body weightlifting, and deep-water running.^{13,14} Stationary biking is another option but should be performed with the heel on the pedal, a precaution that will diminish muscular stress transmission to the leg. NSAIDs often are prescribed to relieve pain^{13,14} and to decrease possible inflammation. Ice may be used to further reduce swelling

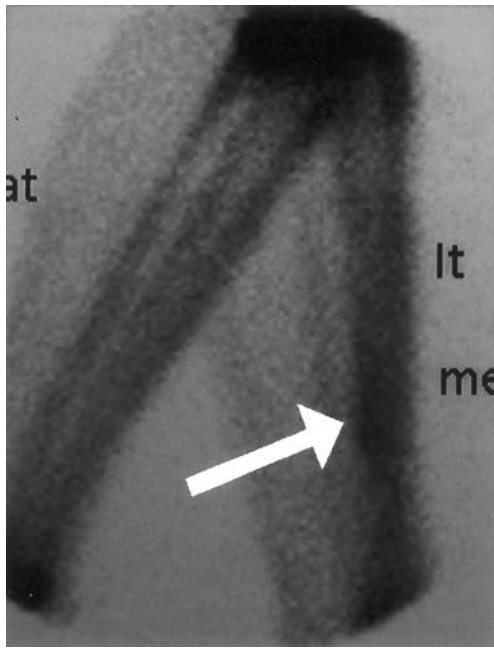


Fig. 23.2 Classic bone scan demonstrating the increased linear uptake along the posteromedial aspect of the tibia in the delayed phase indicative of medial tibial stress syndrome (MTSS). The linear uptake is most commonly observed in the distal one third of the leg; however, in this specific case, the location is slightly more proximal.

and inflammation.^{14,36} Addressing biomechanical abnormalities is also recommended.^{14,38,39} For example, excessive pronation may be corrected with the use of custom or off-the-shelf orthotics.³⁸ Physical therapy modalities including massage, electrical stimulation, iontophoresis, ultrasound, and kinesio taping have also been used.^{12,36,40,41} A combination a home training program with extra-corporeal shock wave therapy has also been shown to be more effective than a home training program alone.⁴² If pain is present with walking or at rest, range-of-motion boots and/or walkers are used. In rare cases, crutches may be necessary.

If the patient has not experienced pain during conservative treatment, a gradual return to training may be initiated. Warm-up and cool-down routines, including stretching, are advised with each workout to prevent recurrence of symptoms. If the patient remains asymptomatic, progression of training is recommended at increments of 10% to 25% for 3 to 6 weeks.¹³ If symptoms return, activity should cease for at least 2 weeks before training is resumed at a lower intensity and duration.

Operative

Fasciotomies of the posterior compartments of the tibia are possible treatment options in patients with intractable MTSS.^{6,13,29,39,43} In these rare cases, fasciotomies may alleviate the pull of the soleus and deep compartment muscles on the corresponding fascial insertions.^{6,13,29,39,43} However, conservative management alone has been successful in treating MTSS cases, eliminating the need for surgical intervention.

PEARL

MTSS pain actually may subside during workout but will recur following cessation of activity. Conversely, pain associated with chronic ECS and PAES does not subside during activity and tends to remain until activity is completed.

Pain is localized to the distal one third of the tibia in MTSS but is usually more proximal in the typical stress fracture.

CASE STUDY 23.1

A 16-year-old female cross-country runner presented for evaluation of progressive right leg pain. Over the preceding 3 weeks, training intensity had been increased in preparation for a season-ending tournament. During that time, increasing pain developed over the distal medial leg. Initially, pain was present only at the conclusion of training but progressed to include soreness on first arising in the morning and with daily activities, forcing the patient to decrease training. She denied constitutional symptoms, history of trauma, or recent footwear change.

Physical examination was remarkable only for tenderness along the posteromedial cortex of the distal one-third of the tibia. Plain radiographs were normal. On the basis of a clinical diagnosis of MTSS, conservative treatment, consisting of cessation of training for 2 weeks, NSAIDs, and ice, was recommended. At the 2-week follow-up, only minor improvement had been achieved and the patient remained in significant pain. Consequently, a range-of-motion boot was implemented and a bone scan was ordered to rule out a possible stress fracture. Because the bone scan was negative, as indicated by a diffuse uptake in the delayed phase, the patient remained in the range-of-motion boot for an additional 4 weeks. After this period, activities of daily living were conducted without pain, permitting a gradual return to training over the ensuing 6 weeks.

STRESS FRACTURES

Repetitive loading caused by overuse or overloading of the lower leg results in microtrauma to the bone that eventually may lead to stress fracture.^{6,40,44–46} Stress fractures of the tibia are more frequent^{3,13,44,47–51} and more problematic to treat than those of the fibula. Fibula stress fractures tend to heal more rapidly and generally do not require adjunctive therapy.^{48,50} Therefore, this section will focus on tibial stress fractures.

Athletes are particularly prone to stress fractures of the leg, and specific stress fractures are related to certain types of activities. For example, the more common posteromedial stress fracture usually is associated with running activities.^{13,40,52} Conversely, midanterior tibial cortex stress fractures often are associated with dancers and athletes involved in cutting and jumping activities.^{40,45,53–57}

Risk factors for developing a stress fracture include excessive training, training errors, biomechanical variants, and menstrual irregularities with corresponding changes in bone density.^{11,13,47,49,52,58,59} Excessive training, particularly common early in the athlete's season, causes overuse or overloading, which may result in stress fracture.^{6,40,52} In addition, overlapping of sport seasons, which often occurs when teenage athletes participate in multiple sports, also may lead to an overuse scenario. Training errors, including changes in training surface, footwear, and technique, often result in overloading, which may result in stress fracture.^{6,40,44} Weather and seasonal differences

affect surface conditions for many outdoor sport activities and may increase the risk of stress fracture. For example, dry conditions and the fall season are both associated with hard ground surfaces, which result in an overloading environment for soccer players. Simple measures, such as watering soccer fields when the ground is hard, may reduce the risk of stress fracture and other injuries by minimizing loading conditions. Biomechanical factors, such as cavus feet, leg-length inequality, and muscular imbalance, also may increase the risk of developing a stress fracture.^{11,13,47,51} Finally, the female athletic triad of amenorrhea, disordered eating, and low bone mineral density have been associated with an increased incidence of stress fractures.^{46,47,49,58}

The cause of stress fractures is multifactorial in nature and often results from an imbalance of natural bone formation and resorption cycle because of repetitive loading.^{44,51,54,59} One theory proposed to explain the mechanism of stress fracture suggests that muscle fatigue results in the transmission of excessive forces to the underlying bone, ultimately leading to stress fracture.^{15,44,51,54,58} Another hypothesis asserts that simple, repetitive weight bearing leads to a concentrated rhythmic muscle action, which causes excessive transmission of forces beyond the threshold of bone, thereby resulting in fracture.^{15,44,51,54,60} Forces from large posterior muscle groups, in particular, may cause increased tension on the anterior cortex of the tibia, possibly leading to the problematic midanterior tibial stress fracture.⁵⁴

History

Pain associated with tibial stress fractures is typically more proximal than that caused by MTSS (*Case Study 23.2*). Although pain is normally localized to the fracture site, diffuse pain also may occur. Stress fracture pain will develop gradually, occurring initially as a mild ache following a specific amount of exercise and then subsiding. As the condition progresses, pain may become severe and occur during earlier stages of exercise and after cessation of activity. In rare cases, night pain also is possible. Any complaints of constitutional symptoms, including fever and fatigue, should raise concern of a possible tumor or infectious process.

In addition to obtaining a history of pain and symptoms, training and activity also should be investigated to identify possible errors that may increase the risk of stress fracture. Recent changes in activity level, such as increased quantity or intensity of training, modifications in training surface, footwear alterations, and technique should be noted. Inquiries regarding diet also should be conducted because the presence of eating disorders increases the risk for stress fracture. Furthermore, obtaining menstrual histories of female athletes also is pertinent because oligomenorrhea and delayed menarche both increase the risk of stress fracture. Finally, a review of systems is suggested to assess general health, medications, and personal habits to identify any additional factors possibly influencing bone health.

Physical Examination

On gross physical examination, the leg will appear normal. Compartments should be soft and the posteromedial aspect of the middle to distal one-third of the tibia should not be tender.

Joint range of motion usually is normal, but gait analysis may reveal biomechanical risk factors. Neurovascular examination typically is normal in the absence of any associated abnormalities. Palpation will reveal tenderness localized to the fracture site. In addition, erythema or localized swelling also may be noted. An ultrasound or a tuning fork will produce vibratory pain over the site of the stress fracture. Hopping on one foot may also reproduce pain. In long-standing fractures, a palpable bony thickening may be present.

Diagnostic Studies

A clinical diagnosis of stress fracture often may be made solely on the basis of the history and physical examination,^{44,59} but diagnostic imaging may confirm the diagnosis or assist in identifying the stress fracture in questionable cases. Plain radiographs should be performed as the first imaging step but may be negative, because radiographic abnormalities often are not observed until 2 to 3 weeks after the onset of symptoms and may not appear at all if activity modification has been performed.^{3,44,51,52,61} Radiographic abnormalities may appear as a faint periosteal reaction, a fluffy area of callus, or a cortical lucency.⁴⁶ If radiographic examination demonstrates the presence of a stress fracture, no further imaging is necessary.

A three-phase bone scan is indicated when suspicion of stress fracture remains despite negative radiographs.^{46,52} The specific scintigraphic pattern of a stress fracture demonstrates focal uptake in the area of fracture (*Fig. 23.3*).^{44,62} MRI, another diagnostic option, differentiates among fracture, tumor, and infection and also localizes the pathology.^{36,51,52,59,63} An MRI also is useful in differentiating between longitudinal stress fractures and MTSS, the more commonly observed overuse injury, because bone scans of these conditions demonstrate identical diffuse uptake in the distal one third of the tibia (*Case Study 23.3*).^{64–66}

In addition to its diagnostic capabilities, imaging also assists in differentiating among the various types of stress fractures. For

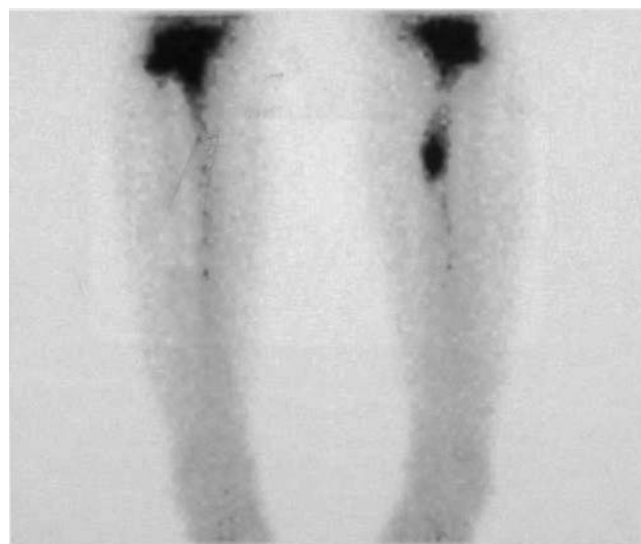


Fig. 23.3 Bilateral bone scan demonstrating normal scintigraphy (*left*) versus the focal uptake pattern of a typical tibial stress fracture (*right*).

example, radiographs depicting a small lucency or a “dreaded black line” in the midanterior cortex of the tibia are indicative of a midanterior cortex tibial stress fracture (Fig. 23.4, A).^{40,52} Because of the relatively avascular nature of this portion of the tibia, a bone scan initially may be interpreted as negative, but closer examination will depict an area of decreased uptake at the fracture site.^{52,57} If this type of fracture is not initially diagnosed and treated, a complete fracture may result. Conversely, plain radiographs of longitudinal tibial stress fractures often are normal, whereas bone scans will demonstrate increased uptake in the lower tibia.⁶⁷

Treatment

Conservative

Conservative treatment for stress fracture is focused on pain relief and protection from further injury.^{46,52} Improvement in muscular strength and endurance, continuation of cardiovascular fitness, and management of biomechanical factors also are important. Relative rest, possibly with weight-bearing restriction, is recommended for a minimum of 2 to 4 weeks. Cardiovascular fitness should be maintained with cycling, swimming, deep-water running, or other nonloading activities.^{46,51,52,62} Upper body strength training is recommended to maintain muscle mass and is not likely to jeopardize fracture healing.⁵¹ Bracing or casting may be required for 3 to 12 weeks to immobilize the fracture adequately in severe cases or if pain is not relieved after the initial 2- to 4-week rest period.^{38,68} Because prompt return to activity is a priority for elite athletes, electrical stimulation is highly recommended. Electrical stimulation also has been effective in healing traumatic fracture nonunions.^{40,52–54}

In addition to rest, treatment should emphasize the importance of modifying intrinsic risk factors to prevent future injury. This includes drawing lab work such as serum calcium, albumin, alkaline phosphatase, and serum vitamin D levels. Hormone levels should be drawn for patients with a history of hormonal imbalances or endocrinopathies.⁶⁹ Contributing factors, such as training errors, improper footwear, and muscle imbalances that were identified in the history and physical examination, also must be addressed.^{6,46,51,70} Training regimens should be individualized for each patient including examination of shoes for signs of wear and inadequate support. Shoes should be replaced every 500 km and, if necessary, appropriate orthotics should be implemented.^{45,52} Treatment plans for athletes with eating disorders or females with menstrual irregularities should involve dietary counseling and/or estrogen replacement therapy to accelerate healing and to prevent future problems.^{44,52,71}

Use of pharmacologic agents in the treatment of stress fractures. Teriparatide and other bisphosphonates are pharmacologic agents that increase bone formation in the early stages of fracture healing, increase bone mass, and decrease the risk of fractures in osteoporotic bone. It has been approved for treatment of osteoporosis only, and there has been frequent anecdotal cases of physicians using the medication off-label for treatment of chronic tibial stress fractures and nonunion.

Animal models and basic science research seem to support the use of pharmacologic intervention. Significant improvements in callus volume, callus mineralization, bone mineral content, strength and rate of successful union at the fracture site in both normal and delayed healing models has been

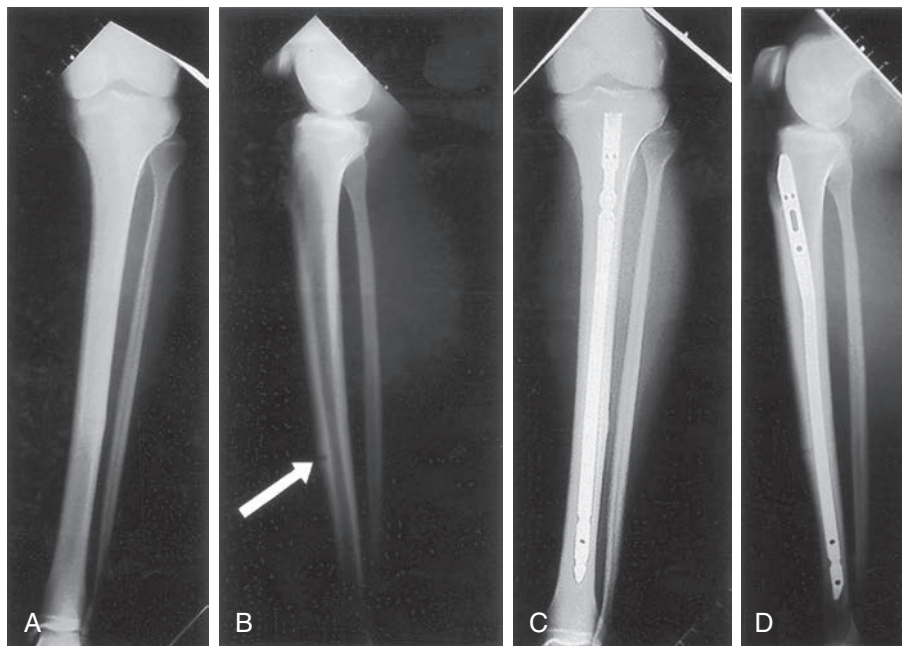


Fig. 23.4 (A and B) Preoperative radiographs of a male runner who presented with a midanterior cortex tibial stress fracture, also referred to as a “dreaded black line” which is visible in the lateral radiograph (B). (C and D) Because of the severity of the fracture, intramedullary nailing was required. As demonstrated in the 2-month postoperative radiographs, the fracture healed completely without the need for bone grafting.

demonstrated.¹⁴⁸ Unfortunately, clinical studies are sparse. Aspenberg et al. found no significant statistical difference when teriparatide was compared to placebo in normal healing of distal radius fractures.⁷²

Various small clinical series have shown some benefit in the treatment of fracture nonunion, but unfortunately no studies have demonstrated accelerated healing in human subjects with tibial stress fractures. The literature is replete only with anecdotal use of the medication to assist in healing fracture union. Therefore, at this time, no definitive clinical evidence can support pharmacologic management of tibial stress fractures.^{73–75}

Return to activity should be gradual and individualized according to symptoms, with an emphasis on progress only when activity is accomplished without pain.^{51,52} It must be stressed that activity should cease if any pain occurs and should not be reattempted until the pain is alleviated.^{52,62} In addition, once the pain is alleviated, the patient must return to the lower-loading activity and not advance until each successive activity has been accomplished without pain. A period of rest also must be implemented between activities before advancing to a higher-loading activity. Although athletes may resume full training in 8 to 16 weeks, patients must be aware that a prolonged recovery period may be required for more severe stress fractures.^{54,57} Midanterior cortex tibial stress fractures, in particular, require a significant period of rehabilitation.^{3,45,52–54,62} Despite this prolonged rehabilitation, conservative treatment is similar to that for other tibial stress fractures and includes avoidance of activity, bracing or casting, and possible electrical stimulation.^{52,54,57}

Operative

Although most stress fractures heal successfully with conservative treatment, surgery may be warranted for severe stress fractures, such as midanterior or longitudinal tibial stress fractures, or for chronic nonunions of proximal medial stress fractures.^{45,53,54,57} Intramedullary nailing has yielded promising results in high-demand patients with problematic stress fractures.^{45,53,54,57} Our experience with intramedullary nailing also has been positive and involves the treatment of three midanterior tibial stress fractures, all of which healed completely without the need for bone grafting (Fig. 23.4, B).

PEARL

Vibration from a tuning fork or ultrasound will produce pain corresponding to the stress fracture site but will not elicit pain in cases of MTSS or chronic ECS.

If a stress fracture is suspected on the basis of the history and physical examination despite negative plain radiographs, additional imaging, such as a three-phase bone scan, is recommended to confirm the diagnosis.

Pain and swelling in the subcutaneous border of the tibia is indicative of a midanterior tibial stress fracture, which requires careful radiographic evaluation to confirm the presence of the subtle “dreaded black line.” If diagnosis remains questionable, a three-phase bone scan demonstrating a focal area of decreased uptake in the anterior tibial cortex will confirm the diagnosis.

CASE STUDY 23.2

A 16-year-old female soccer player related a 3-week history of anterior tibial pain localized approximately 7 cm below the tibial tubercle. Initially, pain was mild and occurred only with prolonged training. When the patient continued her training intensity, the pain progressed to the point at which training became difficult and persisted with daily activities; however, the patient did not seek treatment at this time. Before her final game, the patient stated that her pain was so severe she was unsure whether she should/could continue to play. Despite constant pain, the patient competed in the final game and experienced a noncontact tibial fracture while running.

Roentgenograms confirmed the presence of a tibial fracture with an intact fibular that was located at the anterior tibial cortex approximately 7 cm below the tibial tubercle, corresponding to the site of a presumed existing stress fracture. Conservative treatment was recommended and involved long-leg casting for 3 months. Because of minimal bone healing, determined by radiographic evaluation, long-leg casting continued and pulsed electromagnetic stimulation was added for 1 month. Following the use of the long-leg cast, a long-leg fracture brace was used with continued pulsed electromagnetic stimulation.

After 6 months of conservative treatment, aching continued at the fracture site on weight-bearing ambulation. Subsequent plain radiographs and computed tomography (CT) scan indicated small areas of spot weld healing but a largely inadequate bridging callus. Consequently, operative treatment involving reamed intramedullary nailing of the tibia without fibular osteotomy was performed. Approximately 4 months following surgery the fracture was completely healed, and by 8 months postsurgery the patient returned to playing soccer.

CASE STUDY 23.3

A 47-year-old woman who regularly walks for cardiovascular fitness presented with complaints of left lower leg pain. The patient described a “deep-aching” pain in the lower one-third of her leg. Over the past 3 months, pain increased with continuation of the patient’s walking program and began to occur at night, eventually resulting in limitation of activity. Her medical history was significant for osteoporosis and systemic lupus erythematosus, which was treated with multiple medications.

Neurovascular and physical examinations were grossly normal. No swelling was observed, but palpation revealed mild tenderness along the distal tibia. Plain radiographs did not reveal the presence of a fracture or periosteal reaction. An MRI was ordered to differentiate between the suspected longitudinal stress fracture and possible MTSS and subsequently demonstrated a longitudinal stress fracture in the tibial metaphysis with surrounding bone edema (Fig. 23.5). Conservative treatment involving a range-of-motion boot and nonweight-bearing ambulation was recommended. Six weeks following treatment, plain radiographs demonstrated a slight callus formation, indicative of the healing process. As a result, the patient was instructed to progress from partial to full weight-bearing ambulation over a 4-week period. Full weight-bearing ambulation in a range-of-motion boot continued for an additional 4 weeks, with subsequent introduction of a regular shoe. At 4½ months, the patient resumed her walking program with a gradual increase in mileage.

CHRONIC EXERTIONAL COMPARTMENT SYNDROME

Compartment syndrome of the lower leg generally is characterized by severe intracompartmental swelling that impairs neuromuscular function within the involved compartment which produces pain.^{76,77} This syndrome is classified into



Fig. 23.5 Magnetic resonance imaging portraying the longitudinal signal change in the distal tibia typical of a longitudinal tibial stress fracture.

two forms: acute, the more severe form requiring immediate surgical intervention; and chronic.^{76,78–86} Acute compartment syndrome, commonly caused by trauma, occurs when intracompartmental pressure is elevated to such a degree that immediate decompression is necessary to prevent intracompartmental necrosis.^{76,78,79,83} Conversely, chronic ECS develops when exercise sufficiently raises intracompartmental pressure to produce small vessel compromise, which subsequently causes ischemia and pain,^{80,82,87} but not to the degree exhibited in the acute form.^{76, 79} Athletes exhibiting chronic ECS who continue or increase training are at greater risk of developing acute compartment syndrome.^{79,81,85} Chronic ECS often presents in bilateral form in young athletes with equal incidence in males and females and typically is observed in runners or participants in sports involving ball or puck.^{3,10, 77,88–90} Anterior chronic ECS is more common than the lateral and posterior forms of this syndrome (Fig. 23.6).^{3,10,35,77,80,88,90–92} Although symptoms of chronic ECS, such as pain, muscle weakness, numbness, and swelling, are general, the onset and subsidence patterns are specific to the condition.^{78–80,88} Symptoms resolve after activity is discontinued but generally return at the same interval or intensity at the next training session.^{3,10}

Although the etiology of chronic ECS is not as well understood as that of the acute form, raised intracompartmental pressure resulting in relative ischemia of the involved muscles is likely the pathophysiologic mechanism producing this condition.^{1,77,83,84,88,89,93} Repeated muscle contractions during exercise cause an increase in muscle volume by as much as 20% because of fiber swelling and increased intracompartmental blood volume.^{76,79,80, 88,92} The resulting increase in compartmental pressures is transient and typically will normalize within 5 minutes of completing exercise in asymptomatic people.^{80,90,94,95} In chronic ECS, however, intracompartmental

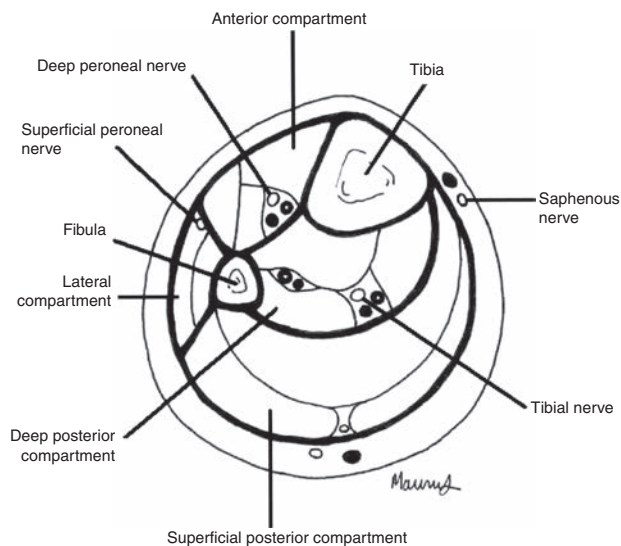


Fig. 23.6 Cross-sectional view demonstrating the compartments of the lower leg and associated anatomy.

pressures may remain abnormally high for 20 minutes or longer after exercise before returning to normal.^{78,91,96}

Several theories have been proposed to explain tissue ischemia, the main symptom of chronic ECS. The first theory suggests that increased compartmental pressure during exercise causes arterial spasm, which results in decreased arterial inflow.^{88,97} An alternative hypothesis asserts that transmural pressure disturbances produce arteriolar or venous collapse, which subsequently leads to ischemia.^{88,97–99} Finally, and perhaps more pertinent to athletes, venous obstruction recently has been advocated as a possible cause of tissue ischemia.^{78,79,83,88} According to this theory, eccentric exercise results in myofiber damage, which causes release of protein-bound ions into the compartment. Such repetitive eccentric contractions therefore cause not only an increase in ion concentration within the compartment but also a subsequent increase in osmotic pressure. This resulting arteriovenous gradient, in which venous pressure is increased and arterial blood flow is decreased, consequently leads to tissue ischemia.^{78,79,83,88} The association between repetitive eccentric contraction in the anterior compartment of runners and the increased incidence of chronic ECS in the anterior compartment lends support to this theory.^{77,79,80,88,90,91,100,101}

History

Patients experiencing chronic ECS may complain of cramping, burning, or pain over the involved compartment(s) with exercise (Case Study 23.4). Pain associated with anterior chronic ECS may not be limited to the compartment but also may radiate to the ankle and foot. The most characteristic symptom of chronic ECS is pain occurring at a fixed point in the patient activity. The pain will become progressive with continued exercise or increased intensity but often will dissipate or cease with rest, usually within 20 minutes of completion of activity. Although this pattern of pain relief is observed in the majority of athletes with chronic ECS, it is not unusual for pain to ensue for a longer period. In extreme cases, pain may be constant. In addition, patients

with anterior and deep posterior compartment syndromes occasionally describe paresthesia in the dorsum of the foot or in the instep, respectively. In severe cases, transient foot-drop may occur.

Physical Examination

Results of physical and neurocirculatory examinations in patients exhibiting chronic ECS are normal before exercise. Because pre-exercise examinations may not yield insight into the condition, examinations also must be conducted after the patient has performed the exercise that initiates the symptoms. Following exercise, a sensation of increased fullness, swelling, tension, or increased leg girth may be produced in the involved compartments. The leg also may be tender over the involved muscles. This diffuse muscular tenderness must be distinguished from that associated with superficial nerve entrapment, which usually is focal at the site of entrapment. In cases of severe chronic ECS, muscle weakness and paresthesia to a light touch may be observed. Pulses, however, will remain normal in all cases of chronic ECS.

Diagnostic Studies

In addition to physical examination, diagnostic testing, such as radiographs, bone scans, electrophysiologic testing, and MRI/magnetic resonance angiography (MRA), may assist in differentiating other possible lower leg conditions from chronic ECS. Radiographs typically are normal in cases of chronic ECS. Although rarely positive in chronic ECS, bone scans also should

be obtained to eliminate MTSS and stress fracture diagnoses. Electrophysiologic testing generally is not necessary but may be beneficial in documenting the extent of motor loss in patients with footdrop. An MRI/MRA is recommended only when symptoms are accompanied by a visible or palpable mass in the leg or when clinical evidence suggests possible popliteal artery compression.

The most useful diagnostic tool to confirm chronic ECS is compartmental pressure testing.^{3,15,101} Although many authors advocate performing pressure tests before, during, and after exercise,^{15,79,93,95,102–108} we prefer pre-exercise and postexercise testing only and do not recommend that measurements be obtained during exercise because of technical difficulties and the unreliability of measurements. The side ported needle technique, involves the injection of small amounts of normal saline into the compartments using an 18-gauge needle and a handheld compartmental measurement device (Fig. 23.7, A). Patients are placed in a supine position with the knee extended and the ankle in neutral dorsiflexion (Fig. 23.7, B). The needle tip location and depth of penetration must be controlled to obtain reliable measurements.¹⁰³ Pressure measurements are taken before exercise and at 1 minute and 5 minutes following exercise. If 5-minute measurements are borderline, 15-minute compartmental pressure measurements are obtained following exercise. We use the compartmental pressure measurement guidelines to establish a diagnosis of chronic ECS and are supported by other surgeons, as summarized in Table

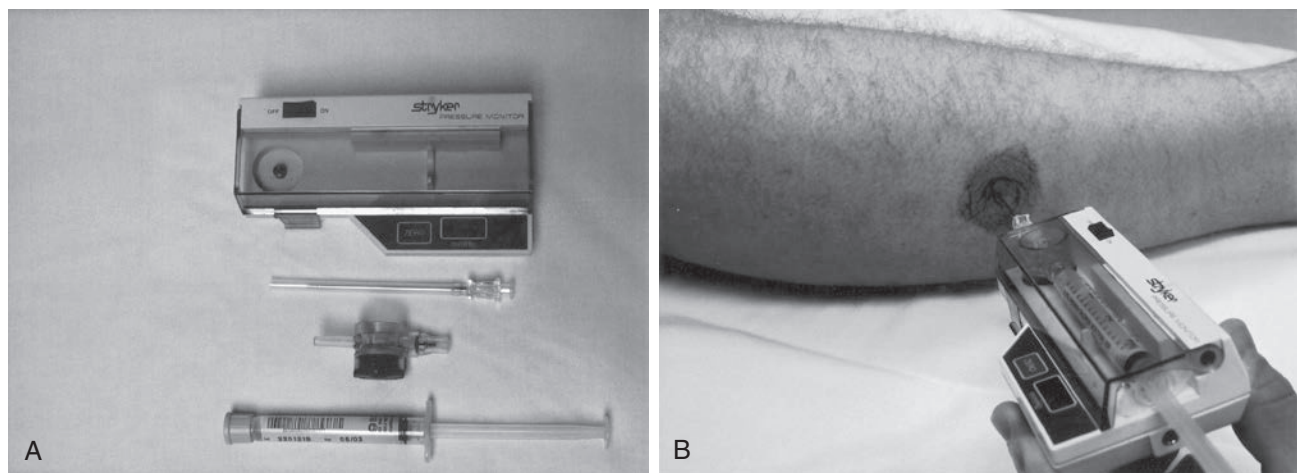


Fig. 23.7 (A) Handheld compartmental pressure measurement device (Stryker Instruments, Kalamazoo, Mich.). (B) To ensure accurate compartmental pressure measurements, the patient should be placed in a supine position with the knee extended.

TABLE 23.1 Compartmental Pressure Measurement Guidelines for Establishing Chronic Exertional Compartment Syndrome

Source	Pre-exercise	1-min Postexercise	5-min Postexercise	15-min Postexercise
Edwards PH, Myerson MS ⁷⁹	≥15 mm Hg	≥30 mm Hg	≥20 mm Hg	N/A
Pedowitz RA, et al. ⁹⁰	≥15 mm Hg	≥30 mm Hg	≥20 mm Hg	N/A
Rorabeck CH ⁹⁶	≥15 mm Hg	N/A	N/A	≥15 mm Hg

23.1.^{79,90,95} Pressures usually return to normal within 3 to 5 minutes after exercise in patients without this condition.¹⁵ If elevated pressures continue for 5 to 10 minutes, chronic ECS is diagnosed.

Treatment

Conservative

Although some authors solely advocate surgical management for the treatment of chronic ECS, we recommend beginning with nonoperative treatment to address the extrinsic and intrinsic factors that contribute to the condition.¹⁰ Modification of extrinsic factors, including training surface, shoe design, and training intensity may decrease the symptoms of chronic ECS. Muscle imbalance, flexibility, and limb alignments are intrinsic factors that may be addressed with either strengthening and stretching exercises or orthoses. A short-leg cast used for approximately 4 weeks may cause atrophy of the leg musculature that in turn may alleviate symptoms. Biomechanical abnormalities also should be addressed and corrected, usually with an orthotic, before training is resumed. Because identifying and modifying all risk factors contributing to chronic ECS is difficult, many athletes may continue to have symptoms of chronic ECS on resumption of activity and may be unable to return to competition.⁸⁷ In such cases, an operative approach may be warranted to enable return to the previous level of intensity.

Operative

The surgical technique for treating chronic ECS involves decreasing intracompartmental pressure, as depicted in Fig. 23.8.^{6,86,88,100} Fasciotomy generally is recommended if symptoms persist for at least 3 months and produces favorable results, especially in the anterior and lateral forms of the condition.^{43,77,79,82,87–89, 91,95,103,108–110} Care must be taken to identify and protect the superficial peroneal nerve. In case of concomitant superficial peroneal nerve entrapment, release of any fascial tethering or compression also may be performed. To prevent postoperative fascial scarring, early passive and active range-of-motion exercises are implemented and weight-bearing ambulation as tolerated is permitted within 2 weeks following surgery.^{79,88,91} Patients may begin exercise on a stationary bicycle at 2 weeks postoperatively, followed by isokinetic strengthening exercise 3 to 4 weeks after surgery. Running may be initiated 5 to 6 weeks postoperatively, with speed and agility drills added during the eighth week.^{79,88} Athletes generally return to full sports participation within 8 to 12 weeks following surgery.

PEARL

Patients are able to predict the time of symptom onset.
The physical examination typically is normal at rest.
For the most accurate diagnosis, it is imperative to perform compartmental pressure testing after activity that initiates symptoms.

CASE STUDY 23.4

A 15-year-old female soccer player presented with complaints of bilateral leg pain during activity. The patient had been diagnosed with chronic ECS approximately 1 year ago and underwent bilateral fasciotomies of four compartments at another facility. After her initial postoperative rehabilitation, pain recurred on exercise. A second bilateral fasciotomy of four compartments was performed, followed by recurrence of symptoms. Presently, pain developed approximately 15 minutes after beginning soccer practice and increased until activity ceased.

The current evaluation revealed a normal examination at rest, with well-healed surgical incisions. It was noted that the medial incision was quite proximal. Compartmental pressure measurements after provocative exercise confirmed bilateral compartment syndrome, based on pre-exercise and 1-minute and 15-minute postexercise readings. The 1-minute and 15-minute postexercise measurements were greater than 21 and 17 mm Hg, respectively, in all compartments.

Edwards performed a third surgery involving bilateral fasciotomies of four compartments. Because of the proximal position of the medial incision and the suspicion that the soleus bridge previously was unreleased, a new 10-cm medial incision was created to be used in addition to the previous midleg lateral incisions. The soleus bridge subsequently was released. Recurrent scarring of the anterior and lateral fasciotomy incisions was noted, and repeat extensile releases were completed. The patient recovered fully and returned to full activity at 12 weeks postoperatively, including twice-daily soccer practice.

NERVE ENTRAPMENT

Lower extremity nerve entrapment is a mechanical irritation of a peripheral nerve caused by impingement.^{111,112} The common peroneal, superficial peroneal, and saphenous nerves are the most at risk for entrapment, which may produce neurogenic leg pain in the athlete (Fig. 23.9).^{113–118} Trauma is a primary cause of all three forms of entrapment.^{115,118} Superficial peroneal nerve entrapment also is observed in dancers and athletes in a wide variety of sports, including bodybuilding, horse racing, running, soccer, and tennis.^{111 113,115,118} Common peroneal nerve entrapment often is associated with repetitive exercises involving inversion and eversion, which often occur in running and cycling.^{111,114,115} External compressive sources, such as tight plaster casts and anterior cruciate ligament (ACL) braces, and internal compressive sources, including osteophytes or proximal tibiofibular joint ganglion cysts, also may cause common peroneal nerve entrapment.^{115,118,119} Knee surgery also may cause common peroneal and saphenous nerve entrapments,^{115,120} the latter of which also may result from inflammatory conditions such as thrombophlebitis.¹²⁰ Superficial peroneal nerve entrapment, caused by either trauma or fascia hernias, is the most common type of nerve entrapment that we have observed.

Although the causes of nerve entrapment are well established, the mechanism responsible for this syndrome is unknown.^{114,117} Certain factors, however, can predispose nerves to entrapment. Nerves coursing through soft tissues are particularly at risk for entrapment. Nerves branching near joints also are at increased risk for entrapment because joints are associated with a high volume of movement and are common sites of trauma.^{112,119}

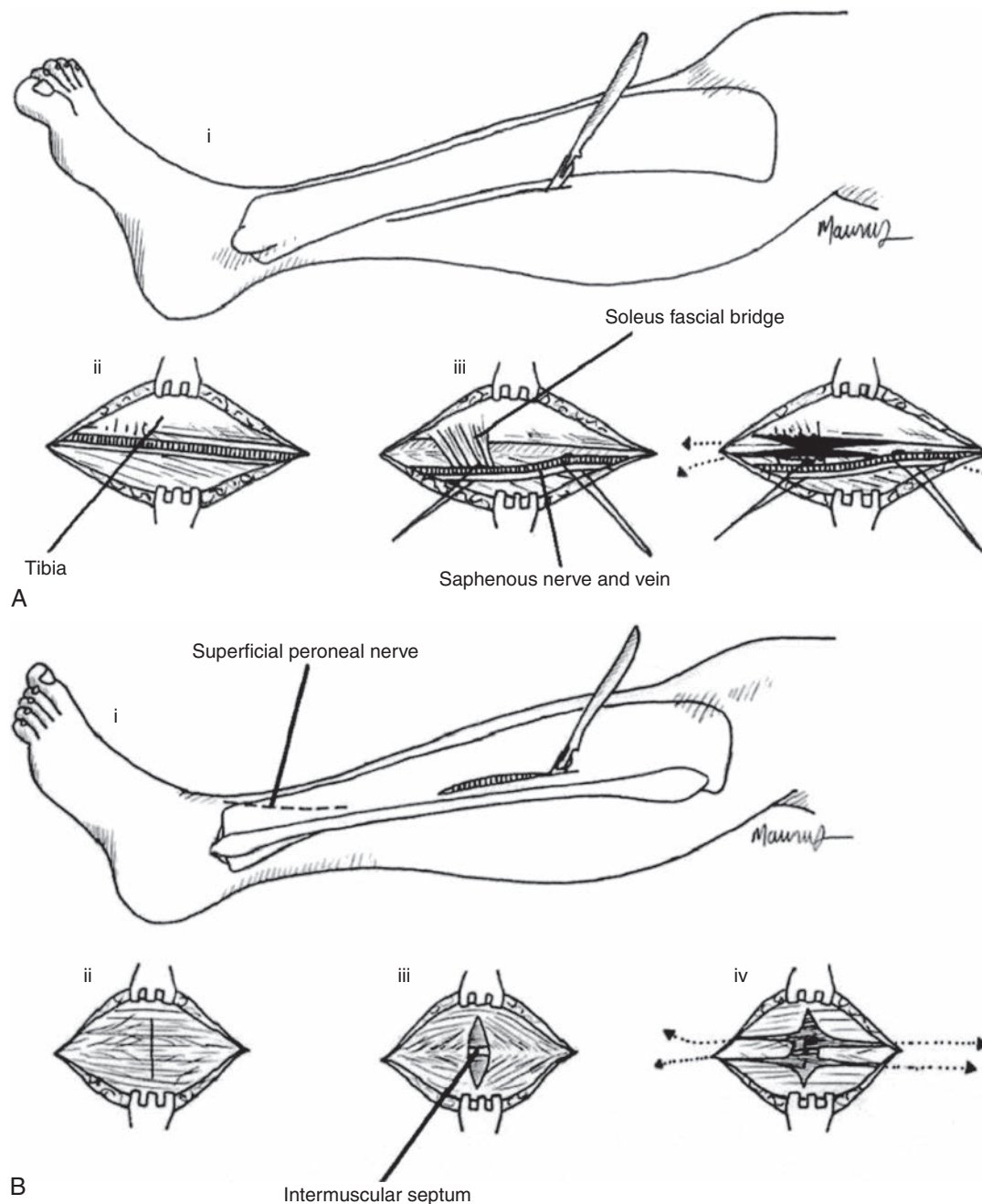


Fig. 23.8 (A) Fasciotomy technique for decompression of superficial and deep posterior compartments used for the treatment of chronic exertional compartment syndrome (ECS). *i*, A longitudinal incision is created on the posteromedial aspect of the leg. *ii*, The tibial posterior border is exposed, allowing full visibility of the saphenous vein and nerve. *iii*, The soleus bridge is released providing exposure of the posterior compartments. *iv*, The affected compartment is incised, using scissors or a fasciotome to extend the fasciotomies proximally and distally. (B) Fasciotomy technique for decompression of anterior and lateral compartments used for the treatment of chronic ECS. *i*, A longitudinal incision is created on the anterolateral aspect of the leg, midway between the tibia and fibula. *ii*, Following exposure of the fascia, a transverse incision is created. *iii*, The intermuscular septum is identified to assist in locating the superficial peroneal nerve. Care must be taken to avoid the superficial branch of the peroneal nerve, which crosses laterally to anteriorly approximately 10 cm above the ankle. *iv*, The appropriate compartment is incised, using scissors or a fasciotome to extend the fasciotomies proximally and distally.

Additionally, nerves, as opposed to circulatory and lymphatic vessels, are susceptible to impingement because of inherent inelasticity.¹¹² Because nerves lack independent movement, impact or compression from either trauma or internal pressure may cause neuropathy.

History

Patients suffering from nerve entrapment of the lower extremity typically present with pain that is aggravated with continued exercise. Common peroneal nerve entrapment pain is located in the region of nerve compression and is referred to the lateral

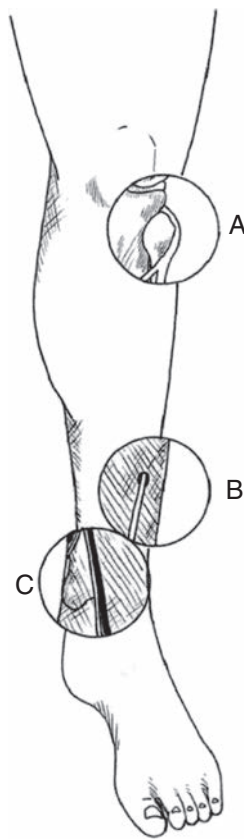


Fig. 23.9 Common sites of nerve entrapment in the lower extremity. (A) Common peroneal nerve entrapment occurs as the nerve wraps around the head of the fibula and exits the peroneal tunnel. (B) Entrapment of the superficial branch of the peroneal nerve typically occurs as it pierces the deep fascia of the lateral or anterior compartments of the leg. (C) A common site of saphenous nerve entrapment occurs where the nerve branches, approximately 15 cm proximal to the medial malleolus.

leg and foot. In contrast, pain associated with superficial peroneal nerve entrapment involves the lateral calf and/or dorsum of the foot (*Case Study 23.5*). Saphenous nerve entrapment often occurs just above the medial malleolus, leading to local pain and referred pain to the dorsum of the foot medially (*Case Study 23.6*). Numbness, often described as a burning sensation, also may be observed with all compressive neuropathies. In addition, some patients may experience localized swelling. Diffuse swelling, on the other hand, is indicative of chronic ECS or a systemic problem. Finally, motor weakness, such as footdrop, typically is observed late in common peroneal nerve entrapment.

Physical Examination

The lower back, hips, and ankle joints should be examined to confirm that an overriding neurologic condition is not present. Fascial hernia also should be ruled out. Range of motion of all leg joints and stability of the knee and ankle should be assessed. Compression or percussion of the nerve is the hallmark test used to determine a diagnosis of nerve entrapment. A tingling sensation along the nerve or at its exit from the fascia is indicative of entrapment syndrome. Tingling typically will be elicited at the level of the fibular neck radiating distally in common peroneal nerve entrapment. Alternatively, in superficial

nerve entrapment, tingling will occur 7 cm to 12 cm above the lateral malleolus, whereas tingling will radiate from just above the medial malleolus and more distally on the medial foot in saphenous nerve entrapment.

Diagnostic Studies

Roentgenograms, MRI, compartmental pressure tests, electromyography (EMG), nerve conduction, and/or nerve block are possible diagnostic tests conducted to confirm the diagnosis of nerve entrapment.^{115,116,121} Radiographs typically are normal in nerve entrapment syndromes but assist in identifying possible compressing bony lesions and in excluding stress fractures and bone tumors.^{116,121} An MRI is recommended if a pressure-causing mass is suspected. Compartmental pressure tests may be conducted to distinguish between chronic ECS and nerve entrapments,^{113, 116,121} because elevated compartment pressures are indicative of chronic ECS. To differentiate between common and superficial peroneal nerve entrapments and to locate the anatomic point of compression, EMG and nerve-conduction studies are recommended and should be performed before and after exercise.^{115,116} A nerve-conduction velocity of less than 40 m/sec is considered abnormal and is indicative of nerve entrapment of the lower extremity.¹²² If superficial nerve entrapment is suspected on the basis of any of the aforementioned diagnostic tests, a nerve block should be performed. The anesthetic should be injected where the Tinel's sign is the strongest or at the location corresponding to maximal pain on pressure. Immediate pain relief following injection is suggestive of nerve entrapment.^{111, 112,117,118,120}

Treatment

Conservative

Conservative treatment for nerve entrapment includes modification of precipitating activity, biomechanical correction, physiotherapy, and/or soft-tissue massage.^{113,115,118} NSAIDs used in conjunction with tricyclic medications such as amitriptyline and, occasionally, gabapentin may alleviate the pain and associated swelling of all three forms of nerve entrapment.¹¹⁸ Iontophoresis is another option that we prefer because of its less invasive nature in comparison with a nerve block. However, nerve blocks may be necessary if iontophoresis fails. Because constrictive clothing and/or devices, including ACL braces or patellar tendinitis straps, place additional stress on the nerves, the use of these devices is not recommended during treatment.^{115,118} In our experience, application of local anesthetic patches may also help to alleviate symptoms.

Operative

Although common peroneal and saphenous nerve entrapments often are successfully treated by conservative measures, superficial peroneal nerve entrapment typically requires surgical treatment.^{115,118} If surgery is warranted, fasciotomy is performed to expose the nerve, and, if necessary, is followed by external neurolysis.^{113–115,120,121} In common peroneal nerve entrapment, resection of osteophytes, ganglion cysts, or other obstructions may be necessary before neurolysis is performed.^{115,118} In rare cases of trauma-induced saphenous nerve entrapment,

neuroectomy may be required.^{12,117,120} Because of the increased risks associated with neurologic surgical procedures, including neuromas and reflex sympathetic dystrophy, surgical treatment requires a thorough knowledge of the peripheral neuroanatomy.¹¹⁸ To minimize such risks, the nerve should be manipulated as little as possible and the surrounding soft tissue should be relatively undisturbed.¹¹⁸ Activity may be increased gradually on wound healing.

PEARL

A careful history and physical examination should be conducted to rule out referred pain or an overriding neurologic condition.

A positive Tinel's sign is highly suggestive of nerve entrapment.

If physical examination and all diagnostic tests, including compartmental pressure measurements, are normal, nerve compression often is the source of the pain.

The fascial exit of the superficial peroneal nerve is variable, ranging from approximately 7.5 cm to 12.5 cm from the tip of the lateral malleolus.

CASE STUDY 23.5

A 30-year-old male runner presented with complaints of lateral leg pain and foot numbness. The symptoms progressed after he began an aggressive running program during the prior year. The pain was described as sharp and tingling and typically occurred over the mid to distal aspect of his lateral leg during running. He denied a history of injury.

On physical examination, a small prominence of soft tissue was noted over the painful area. "Lightning-like" sensations and paresthesias corresponding to the superficial branch of the peroneal nerve were elicited on percussion. Gross neurovascular examination, including sensation, otherwise was normal. After conservative treatment, including failed iontophoresis, a fasciotomy was performed to release the nerve. Postoperatively, activity gradually was increased, with resumption of training at 6 weeks.

CASE STUDY 23.6

A 52-year-old avid golfer presented with a 3-month history of distal medial leg pain. The pain increased with activity and radiated to the dorsum of the foot. Initially, pain was mild but progressed to the point at which the patient was unable to complete a round of golf without significant pain. NSAIDs and ice were implemented without improvement. Although the patient initially denied a history of trauma, on further inquiry, he recalled that he had hit his distal tibia approximately about the medial malleolus on his daughter's bicycle 3 months before the present complaint.

Physical examination revealed full range of motion, with no swelling or cutaneous changes about the distal third of the leg. In addition, no tenderness was observed over the medial distal tibial cortex, and a vibratory test was negative. However, a positive Tinel's sign over the saphenous nerve above the medial malleolus was elicited, reproducing the distal-radiating pain. On the basis of these clinical findings and the traumatic nature of the injury, a diagnosis of posttraumatic saphenous neuritis was established. Conservative treatment comprising NSAIDs, ice, and iontophoresis was prescribed. Symptoms improved markedly at 2 weeks following treatment and completely resolved by 4 weeks, enabling the patient to return to regular activity.

POPLITEAL ARTERY AND VEIN ENTRAPMENT SYNDROME

PAES is more common in athletes than in the general population, especially as a result of increased participation in competitive sports.^{6,123} This condition results from an abnormal relationship between the popliteal artery and the surrounding myofascial structures (Fig. 23.10), producing calf pain on exertion.^{6,123–126} PAES is progressive and, in more severe cases, may result in occlusion of the popliteal because of compression from the medial head of the gastrocnemius muscle.^{6,123}

Although PAES is a possible diagnosis in any athlete with calf pain and intermittent claudication, it is predominantly observed in males under the age of 30.^{6,123–125,127–137} This condition typically occurs unilaterally, but may be observed bilaterally at an incidence of 25%–67%.^{129,138,139} Although relatively rare, it can affect athletes with more hypertrophied gastrocnemius and calf musculature.

It is generally accepted that there are two types of popliteal artery entrapment. The first type is anatomic, in which there can be embryologic abnormalities within the gastrocnemius muscle.^{6,123,124,129} The medial head of the gastrocnemius can have an anomalous attachment to the distal femur, causing tight stricture of the popliteal artery as it passes through the muscle. This variant can be present in up to 1% of patients, and is usually clinically silent. Compression can also possibly be caused by either the soleus or plantaris muscles.^{123,140,141} Secondly, patients can also have functional popliteal artery entrapment. This occurs due to compression of the artery due to a hypertrophy gastrocnemius muscle or excessive positioning in passive dorsiflexion of the ankle or excessive active plantar flexion of the ankle.^{139,142}

Although anatomically ubiquitous in the population, popliteal venous entrapment syndrome is a much more rare vascular cause of chronic calf pain in the athlete. In a wide range of studies in asymptomatic individuals, utilizing ascending duplex ultrasonography or ascending contrast venography, entrapment of the popliteal vein was seen in 27% to up to 47% of healthy individuals. Anatomically, the entrapment can occur from the medial head of the gastrocnemius muscle, or an anomalous fibrous band over the vein. It can be more common in individuals or athletes with significant hypertrophy of the gastrocnemius muscle.¹³⁹

History

Patients usually present with claudication symptoms, with vague calf pain present with exercise (Case Study 23.7). There is usually no pain at rest. Physical exam findings are usually unremarkable. Diagnosis is usually confirmed by CT, MRI, MRA, or, more than likely, lower extremity arteriography.

PAES should be considered in the differential diagnosis of healthy young patients presenting with complaints of intermittent pain typically involving the foot and leg. Pain, described as a deep ache or cramping, generally is posterior in location and typically occurs after vigorous exercise. It is important to note, however, that claudication may be atypical

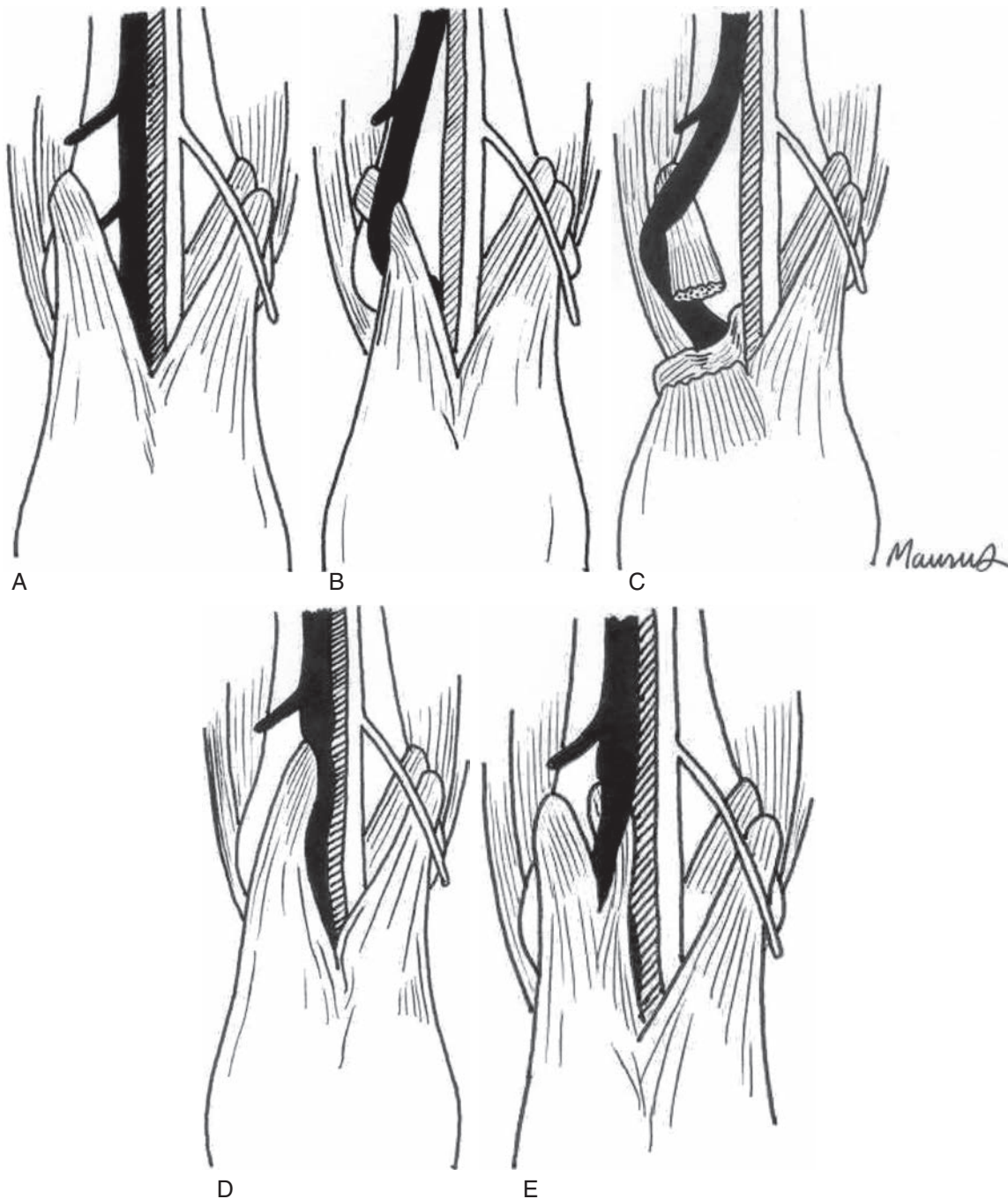


Fig. 23.10 Normal course of the popliteal artery versus possible aberrant pathways involving the medial head of the gastrocnemius muscle that cause popliteal artery entrapment syndrome (PAES) (*popliteal artery = dark, popliteal vein = striped, tibial nerve = white*). (A) Normal course of the popliteal artery in which the artery and vein course distally between the heads of the gastrocnemius muscle, over the popliteus muscle, and beneath the soleus muscle. (B) The popliteal artery deviates medially, wraps around the medial head of the gastrocnemius muscle, and then resumes the normal distal course. (C) The popliteal artery deviates medially, wraps around the medial head of the gastrocnemius muscle, and abnormally courses beneath the popliteus muscle, consequently becoming entrapped. (D) The popliteal artery courses normally but is compressed by the medial head of the gastrocnemius muscle, which is positioned laterally to its normal insertion. (E) The popliteal artery courses normally but is entrapped between the medial head and an accessory tail of the gastrocnemius muscle. (Modified from Rich NM, et al: *Arch Surg*. 1979;114:1377.)

early in the course of this condition, because it may occur with walking and not with prolonged leg exercise. Symptoms occurring less frequently include numbness, tingling, or coolness of the foot; these symptoms may be relieved by changing leg positions.

Frequently, popliteal venous entrapment syndrome is a diagnosis of exclusion, once more common etiologies of calf pain have been ruled out. Patients may present with painful edema, vague calf pain, stasis dermatitis, or, less likely, venous stasis ulceration. The finding can be unilateral or bilateral. There are no predisposing factors, although it has been suggested that a past medical history of deep vein thrombosis can predispose an individual to popliteal venous entrapment syndrome.¹³⁹

Physical Examination

Physical examination often is normal at rest in PAES cases, especially if the artery is still patent. Compartments may be soft, and palpation of the bone and soft tissues may not elicit tenderness. The only possible pathognomonic physical exam finding would be diminishment or loss of popliteal, dorsalis pedis, and posterior tibial pulses with full passive dorsiflexion or the extremes of plantar flexion when compared to the normal contralateral leg. On auscultation, a bruit may be heard after provocative exercise, but the significance of this observation is unclear because it also may be observed in a normal athlete.

Diagnostic Studies

Diagnosis is usually confirmed by CT, MRI, MR angiogram, or, more than likely, lower extremity arteriography. If Doppler sonography indicates PAES, arteriography is recommended to confirm the diagnosis.^{123,128,134,143,144} Often referred to as the “gold standard test” of PAES, arteriography is an invasive procedure involving radiographic imaging after injection of a radiopaque material into the suspected arterial segment.^{123,133,135} Because arteriography may be normal in PAES when the ankle is in the neutral position and the knee is extended, it is important to repeat the studies bilaterally after exercise or with the ankle in positions of provocation, because extrinsic arterial obstruction may be demonstrated with ankle plantarflexion.^{6,127,134,137,143,145,146} MRI/MRA also may be beneficial in evaluating PAES and offers the added benefit of showing the offending soft tissue structure in many cases.^{143,146,147} Compartmental pressure measurement testing and three-phase bone scans are recommended to rule out chronic ECS and stress fractures, respectively.

The diagnosis of popliteal venous entrapment can be made diagnostically with dynamic duplex ultrasonography, with active and passive dorsiflexion and plantar flexion maneuvers of the calf. Infrequently, venography or MRA can be utilized.

Treatment

Treatment of popliteal artery entrapment is most likely conservative, with periods of rest, stretching, and activity modification. Surgical intervention involves resection of the abnormal

portion of the medial head of the gastrocnemius, with full dissection of the artery, and possible bypassed grafting if a portion of the artery is constricted or diseased.¹⁴⁸ Full recovery in athletes can be upward of 6–8 months.

Treatment of popliteal vein entrapment is almost always conservative with compression socks, activity modification, and therapy. Surgery is rarely indicated, and should be reserved for individuals with severe venous stasis ulceration. Surgical treatment consists of resection of the compressive medial head of the gastrocnemius musculature and release of the popliteal vein.

PEARL

The knee may be warm on palpation because of increased collateral circulation.

Bilateral pulses with provocation should be examined to determine whether reduction in pulse volume between limbs exists.

If PAES is suspected on the basis of Doppler sonography, arteriography should be performed to confirm the diagnosis.

CASE STUDY 23.7

A 19-year-old female competitive soccer player presented with complaints of bilateral leg pain. Pain, described as a dull ache in the posterior aspect of both legs, began during workouts. The pain continued to intensify until cessation of activity was required. However, the pain resolved after a short rest period of 5 to 10 minutes. This pattern of intense pain during activity followed by relief after rest continued without progression with every successive practice and competition.

The initial physical examination did not reveal any abnormalities, as demonstrated by soft compartments, no tenderness on palpation, and normal neurovascular findings. Radiographs and resting compartmental pressure measurements were normal. In an attempt to reproduce the patient's symptoms, the patient was instructed to exercise and subsequently returned with complaints of posterior calf pain and mild tenderness on deep palpation of the calf. During this symptomatic period, neurovascular examination and compartmental pressure measurements remained normal. A subsequent three-phase bone scan and MRI also were normal. As a result, conservative treatment consisting of rest was implemented for 1 month.

The patient returned for evaluation because of continued symptoms, but the physical examination remained normal. Compartmental pressures were reevaluated at pre-exercise and postexercise intervals and remained within normal limits. Examination of pedal pulses demonstrated normal dorsalis pedis and posterior tibial artery pulses. However, when this measurement was repeated with active plantarflexion or passive dorsiflexion with a straight leg, loss of all pulses was observed bilaterally. To confirm a diagnosis of PAES, an arteriogram with provocative maneuvers was performed and demonstrated loss of flow at both popliteal arteries (Fig. 23.11). Because the patient desired to continue competitive soccer, she elected to undergo surgical release of the entrapped popliteal artery. Surgical inspection revealed a popliteal artery coursing medially to the head of the gastrocnemius muscle and anteriorly to the popliteus muscle belly (Fig. 23.12). These areas of entrapment then were released. After wound healing, the patient gradually increased activity over a 6-week period and returned to competitive soccer 3 months postoperatively.

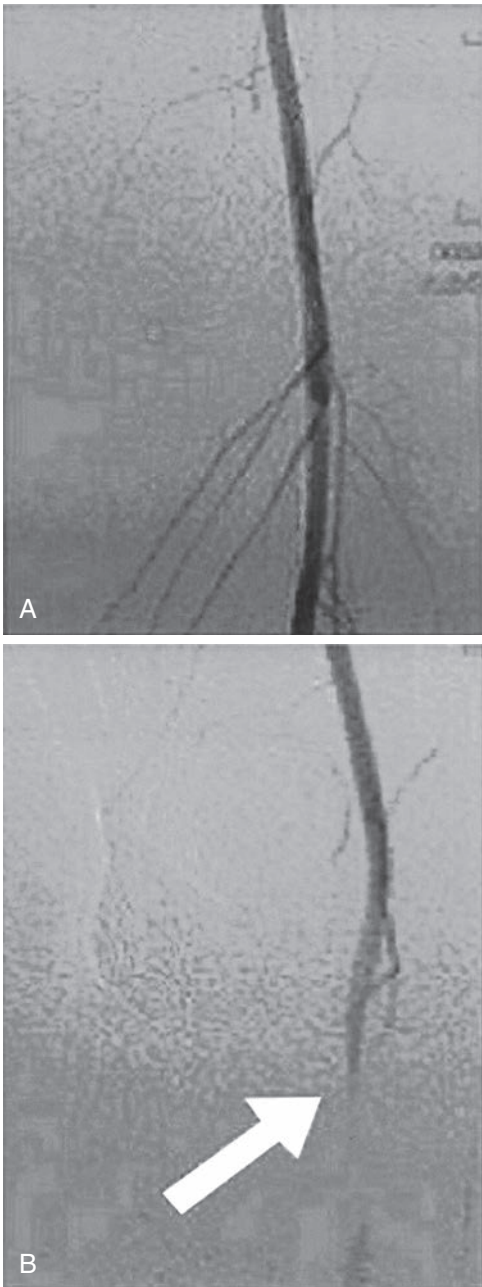


Fig. 23.11 Arteriograms obtained following provocative active plantarflexion and passive dorsiflexion with a straight leg, demonstrating normal flow (A) compared with the decreased flow (B) associated with popliteal artery entrapment syndrome (PAES). (Images courtesy Louis J. Unverferth, MD, Riverside Methodist Hospital, Columbus, Ohio.)

SUMMARY

The most common conditions involving lower leg pain in athletes are MTSS, stress fractures, chronic ECS, nerve entrapment, and PAES. Similarities of symptoms among these conditions make diagnosis difficult. The challenge for the sports medicine specialist is to differentiate among these similarities to establish an accurate diagnosis. Although pain

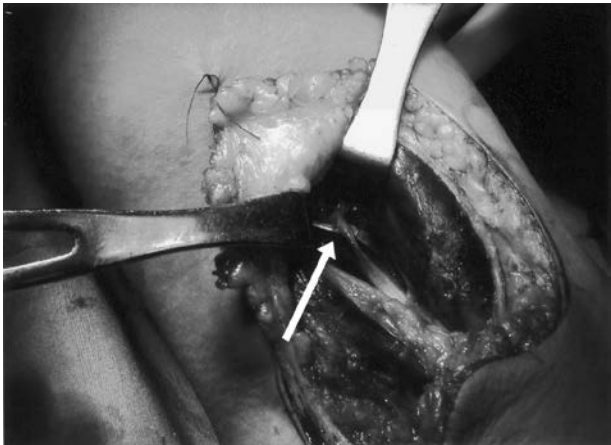


Fig. 23.12 Intraoperative photograph illustrates the abnormal pathway of the popliteal artery as it courses medially to the head of the gastrocnemius and anteriorly to the popliteus muscle indicative of popliteal artery entrapment syndrome (PAES). (Image courtesy Paul Cook, MD, Riverside Methodist Hospital, Columbus, Ohio.)

TABLE 23.2 Pain Locations of the Common Lower Leg Conditions		
Leg Condition	Localized or Generalized	Location of Pain
MTSS	Generalized	Posteromedial distal 1/3
Stress fracture	Localized	Bony tenderness above distal 1/3
Chronic ECS	Generalized	Involved compartments with exercise
Nerve entrapment	Localized	Fascial exit site
PAES	Generalized	Posterior with exercise

ECS, Exertional compartment syndrome; MTSS, medial tibial stress syndrome; PAES, popliteal artery entrapment syndrome.

is the hallmark symptom in all of these conditions, subtleties exist in the location and occurrence of pain among the various conditions. Therefore, determining whether the pain is generalized or localized and isolating the onset and diminishment of pain will assist in determining the appropriate diagnosis, as summarized in Table 23.2. Keys to making an accurate diagnosis include conducting a thorough history, performing an exhaustive physical examination (Table 23.3), and using the appropriate diagnostic tools to distinguish further among these conditions (Table 23.4). Once a diagnosis is established, the preferred treatment is conservative management, consisting of rest from activity and modification of extrinsic and intrinsic factors. Treatment should be individualized according to the patient’s symptoms and involve gradual rehabilitation and return to activity. Although a conservative approach typically is successful, surgical intervention may be required for cases in which conservative treatment has failed.

TABLE 23.3 Physical Examination Observations of Common Lower Leg Conditions

	MTSS	Stress Fracture	Chronic ECS	Nerve Entrapment	PAES
Edema/warmth	Posteromedial distal 1/3	Over site	No	No	Possible around knee
Paresthesias	No	No	Rarely	Often	Rarely
Pedal pulse	Normal	Normal	Normal	Normal	Pedal pulses with provocation
Palpation tenderness	Posteromedial distal 1/3	At site	Involved compartment(s) with exercise	Possible at site of compression	Posterior with exercise

ECS, Exertional compartment syndrome; MTSS, medial tibial stress syndrome; PAES, popliteal artery entrapment syndrome.

TABLE 23.4 Diagnostic Studies Useful in Distinguishing Among Common Lower Leg Conditions^a

Diagnostic Study	MTSS	Stress Fracture	Chronic ECS	Nerve Entrapment	PAES
Roentgenograms	Recommended Normal	Recommended Periosteal reaction/early callus after 10–14 days	Recommended Normal	Recommended Normal	Not recommended N/A
Bone scan	Recommended Linear uptake	Recommended Focal uptake	Not routinely recommended Normal	Not recommended N/A	Not routinely recommended Normal
MRI	Not routinely recommended Signal changes	Not routinely recommended Bone edema	Not routinely recommended Normal	Not routinely recommended Normal	Not recommended N/A
MRI/MRA	Not recommended N/A	Not recommended N/A	Not recommended N/A	Not recommended N/A	Recommended Flow with provocation
Compartmental pressure test	Not recommended N/A	Not recommended N/A	Recommended ≥15 mm Hg at rest; >20mm Hg 5-min postexercise	Not routinely recommended Normal	Not routinely recommended Normal
Arteriography	Not recommended N/A	Not recommended N/A	Not recommended N/A	Not recommended N/A	Recommended Obstruction with provocation

^aThe upper portion for each diagnostic study represents our recommendation; the lower portion indicates the results corresponding to the diagnosis.

ECS, Exertional compartment syndrome; MTSS, medial tibial stress syndrome; PAES, popliteal artery entrapment syndrome.

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Foot and Ankle Injuries in Dancers

Cesar de Cesar Netto, John G. Kennedy, William G. Hamilton, Martin O'Malley

OUTLINE

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INTRODUCTION

Ballet has all the elements of the arts in its makeup—drama, poetry, literature, painting, sculpture, design, music, and, of course, dance. Dancers, both male and female, are the physical means by which the choreographer sculpts a composition of expressive motion. The grace and art of the ballet performance belie the great physical strain on the body as a whole and the foot and ankle in particular. From an early age the dancer must learn to be an artist, a gymnast, and an athlete. Most ballet dancers train for a minimum of 10 years before attaining the skill set necessary to join a corps de ballet. Very few dancers develop into soloists and fewer still attain the role of principle ballerina. Throughout this time of training, the body is placed



Fig. 24.1 Illustration of the *en pointe* stance.

under great strain, and it is by a process of natural selection that those dancers who are flexible and technically proficient survive the rigors of training to advance further. Hamstring strains, foot and ankle pathologies, and low back pain are the most commonly diagnosed musculoskeletal injuries.^{1,2}

Female dancers spend a considerable time *en pointe*, or on the points of the toes (Fig. 24.1), whereas male dancers do not dance on their toes and spend much of their time in turning, lifting, and holding female dancers. As such, male and female dancers tend to present with distinct injuries. In addition to the myriad of physical injuries related to female dancers that follows, female dancers also are prone to the triad of anorexia, amenorrhea, and osteoporosis (see Chapters 4 and 28). This unfortunate triad stems from the significant pressure on dancers to weigh less and less. The most disturbing data suggest that female dancers weigh more than 15% below the ideal weight for height. This has metabolic consequences leading to stress fractures and slower union rates in injured female dancers.³ In contradistinction, male dancers have fewer metabolic problems but are prone to overuse injuries from repetitive motion and to stress fractures (see Chapter 3) from the sudden deceleration of large leaps, *volé*, *sauté*, or *jeté*. Dancers' feet are the instruments on which their art depends. They require, in addition to an extraordinary flexibility and strength, a particular anatomic profile.

Over time a dancer's foot will evolve and only the strongest will survive. Dancers' feet typically are "intrinsic plus;" they have narrow metatarsal width with straight toes. (Intrinsic-minus feet have wider metatarsal splaying and clawing of the toes.) Apart from muscle strength, dancers' feet require great flexibility. In the *relevé* position (Fig. 24.2) the ankle is in a vertical position: 90 degrees of plantarflexion of the ankle-foot complex. The dancer also requires 90 to 100 degrees of dorsiflexion in the first metatarsophalangeal (MTP) joint to go from *relevé* to *en pointe*. These are extraordinary ranges of motion and can only be achieved with years of practice, which mold the



Fig. 24.2 Illustration of the *relevé* stance (*demi-pointe*).

young ballet dancer's bones during the bone growth phase.⁴ As a result of endless practice *barres*, classes, and training, dancers' feet tend to be cavus and have thickened metatarsals to support when *en demi-pointe*. Calluses abound secondary to pressure demands on the skin.

In general, five types of dancer's feet have been described:

1. Grecian (also known as Morton) foot has a relatively long second and third metatarsal in relation to the first and fifth metatarsal. However, dancers do not have the same problems associated with transfer metatarsalgia as does the general population with this foot configuration.
2. Egyptian foot. Long first ray relative to the central metatarsals. This can predispose the first MTP joint to degenerative arthrosis or hallux rigidus.
3. Simian foot. Metatarsus primovarus with hypermobile first ray that causes transfer metatarsalgia to central metatarsal heads.
4. Peasant foot. Uniform metatarsal length, giving broad, square foot. Its stability makes it an ideal platform for dancing.
5. Model's foot. This foot is long and slender with a taper exaggerated cascade from first to fifth metatarsal head. As such, it bears weight unevenly *on demi-pointe* and is a poor foot for dance.

The following is a review of the more common dance injuries and problems in the foot and ankle.

METATARSOPHALANGEAL JOINT

Bunions (see Chapter 22)

Although dancing has been said to play a role in the pathogenesis of bunions, it is unlikely that this is the case. Dancers, like the rest of the population, can be either resistant or prone to develop bunions.⁵ In those dancers that are prone to develop bunions, it is imperative to delay surgical intervention for as long as possible. Bunion surgery adversely affects dorsiflexion of the first MTP joint, a critical motion in dancers. Most bunions, specially the asymptomatic ones, can be treated with



Fig. 24.3 Clinical picture of a ballet dancer treating hallux valgus deformity conservatively using a toe spacer in between first and second toes.

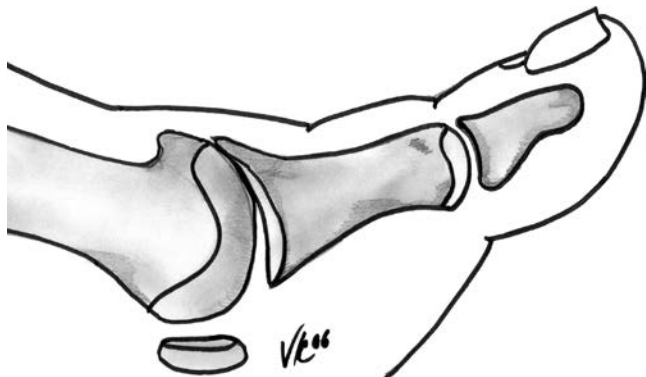


Fig. 24.4 Illustration of hallux rigidus.

conservative methods, including toe spacers and horseshoe pads (Fig. 24.3). The senior author has seen several aspiring young dancers whose careers were ended by well-meaning bunion surgery. If a bunion is precluding the dancer from activity and surgery is warranted, then a chevron or metatarsal osteotomy can provide pain relief and stability without compromising motion. A tarsometatarsal fusion will make the midfoot too stiff and limit the pointe position.

Hallux Rigidus (see Chapter 21)

Any restriction to full dorsiflexion of the first MTP joint will prevent the dancer from performing *relevé*. Many dancers can accommodate by rolling out onto the lateral border of the foot, a process known as “sickling.”

The treatment of hallux rigidus depends on the grade of the disease (Fig. 24.4). A frequently used classification system was proposed by Coughlin and Shurnas (Fig. 24.5)⁶:

In grade I disease, the joint is preserved and marginal osteophytes can be resected (cheilectomy), either arthroscopically or open dorsal approach, with excellent outcomes.

In grade II disease, the joint is involved. There is minor cartilage destruction evident as joint space narrowing on plain radiograph, especially in its dorsal aspect. Surgical treatment involves a more extensive surgical debridement with cheilectomy and resection of the dorsal one-third of the metatarsal head.⁷ Intraoperative dorsiflexion of the hallux greatly overestimates the degree of motion that can be expected following surgery. Just over half of what is achieved at the time of surgery will be evident in the postoperative follow-up examination. It is important that dancers understand that, although surgery will make the condition better, the joint will never be normal. In addition, the length of recovery time must be discussed with the dancer, because a full functional recovery often takes 6 months. To improve functional motion following surgery, a dorsally based closing osteotomy (Moberg) can be used. This procedure improves dorsiflexion but at the expense of plantarflexion, and the dancer should be warned of this.

Grades III and IV hallux rigidus presents with dorsal and lateral osteophytes in addition to clear degenerative arthrosis on both sides of the joint. Arthrodesis is an acceptable surgical option in the general population but not feasible in a dancer. To preserve motion, we recommend similar surgical treatment utilized for grade II, with cheilectomy and Moberg osteotomy (Fig. 24.6, A–C), with good clinical outcomes.⁸

INJURIES TO SESAMOID BONES (SEE CHAPTER 21)

The sesamoid bones lie within the substance of the flexor hallucis brevis tendons. They are commonly injured in dancers, particularly in those who fail to perform a *plié* on landing, absorbing the energy of the landing through partially flexed knees. Without such absorption built into a dancer's technique, sudden deceleration with high impact of the sesamoid bones predisposes to injury.

Sesamoiditis

The differential diagnosis of sesamoiditis is lengthy and requires careful history taking and clinical examination. Magnetic resonance imaging (MRI) aids diagnosis.

The following is a list of differentials:

- stress fracture of the sesamoid bone,
- avulsion fracture or sprain of the proximal pole of the sesamoid,
- sprain of the distal pole,
- sprain of a bipartite sesamoid,
- arthrosis of the sesamoid metatarsal joint, and
- preradiographic osteonecrosis of the sesamoids.

Several mechanisms are responsible for producing sesamoiditis in dancers. Most of these can be treated with a felt pad around the sesamoid for relief (“dancer's pad”) (Fig. 24.7). In general, symptoms resolve without any additional interventions,

Coughlin and Shurnas Classification		
Grade	Radiographic	Clinical
I	Dorsal spur is main finding Minimal joint narrowing Minimal periarticular sclerosis Minimal flattening of metatarsal head	Mild or occasional subjective pain and stiffness Pain at extreme of dorsiflexion and/or plantar flexion on examination
II	Dorsal, lateral, and possibly medial osteophytes give flattened appearance to metatarsal head. No more than 1/4 dorsal joint-space involvement on lateral radiograph Mild to moderate joint-space narrowing and sclerosis. Sesamoids not usually involved but may be irregular in appearance	Moderate to severe subjective pain and stiffness that may be constant. Pain extremes of dorsiflexion and/or plantar flexion on examination
III	Same as grade II but with substantial joint-space narrowing. Possibly periarticular cystic changes, more than 1/4 of dorsal joint may be involved on lateral side, sesamoids may be enlarged and/or cystic and/or irregular.	Nearly constant subjective pain and substantial stiffness Pain throughout range of motion on examination (but not at midrange)
IV	Same as grade III	Same as grade III but definite pain at midrange of motion

Fig. 24.5 Coughlin and Shurnas Staging for Hallux Rigidus (Grade I-IV). (From Coughlin, MJ, Shurnas PS. Hallux rigidus. *J Bone Joint Surg Am* 2004;86A S1(Pt 2): 119–130.)

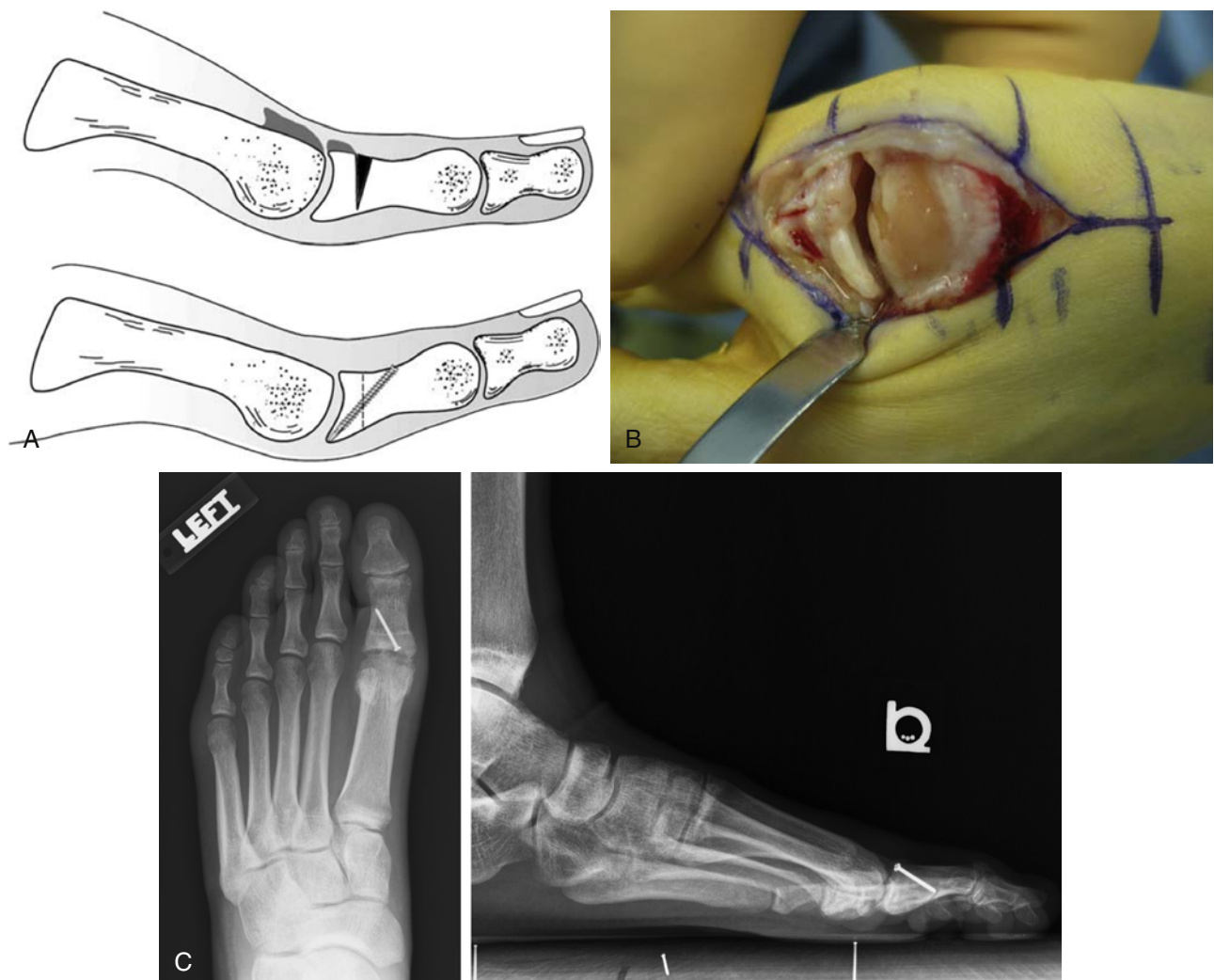


Fig. 24.6 Hallux Rigidus Surgical Treatment in a Dancer. (A) Illustration of cheilectomy and Moberg Osteotomy; (B) clinical photograph of first metatarsophalangeal joint illustrating 75% degeneration of articular cartilage, substantial osteophyte formation, and joint-space narrowing (Grade III hallux rigidus); (C) postoperative radiographs of a Grade III hallux rigidus treated with Cheilectomy and Moberg osteotomy.



Fig. 24.7 Dancer's pad for sesamoiditis.

although it may take up to 6 months for full resolution. In those cases requiring further diagnostic testing, an MRI can be useful. The medial sesamoid is often bipartite, with rounded edges on plain radiograph, distinguishing it from a recent fracture. In those cases with recalcitrant pain, surgery may be warranted. A medial-based incision can locate the medial sesamoid; in the surgical treatment of lateral sesamoid pathology we prefer a dorsal approach in the first web space. A plantar incision should always be avoided because of the increased risk of plantar scarring and chronic plantar pain. Surgery should be reserved only for patients with symptoms persisting for at least 6 months following initial conservative treatment.

Other conditions may mimic sesamoiditis, including bursitis and nerve entrapment:

1. **Sesamoid bursitis:** Swelling and inflammation within the sesamoid bursa may mimic sesamoiditis. However, careful clinical examination usually can identify a symptomatic bursa when present. Treatment consists of a well-directed local corticosteroid injection to the bursa. Bursitis still may take some time to resolve, and it can be complicated by a fibrous scar that causes repeated symptoms. In such cases, a bursectomy can be performed through a careful medial approach.
2. **Joplin's neuroma:** Entrapment of the proper digital nerve, adjacent to and, rarely, under the tibial sesamoid, will cause symptoms similar to sesamoiditis. Joplin's neuroma, however, will display a characteristic nerve compression sign with palpation. In those cases recalcitrant to conservative therapy, neurolysis, and transposition of the nerve are required.
3. **Lateral proper digital nerve entrapment:** The lateral proper digital nerve may be compressed under the deep transverse ligament, causing pain in the great toe on the lateral side. Because of the position of the nerve, a compression test cannot be performed. Diagnosis is made with a selective local anesthetic injection to the nerve. Surgical resection of the transverse ligament is often curative.

HALLUX INTERPHALANGEAL JOINT

In young ballet dancers, hyperflexion of the great toe interphalangeal joint (IPJ) can occur when attempting *en pointe*. Here, weight is distributed over the nail and dorsum of the toe in the pointe



Fig. 24.8 Taping of the toes.

shoe. Hyperextension of the great toe IPJ also occurs, usually to compensate for lack of motion in adjacent joints. Rarely does this need surgical intervention, despite radiologic appearances, because the joint is quite accommodating and typically asymptomatic. In those who do complain of symptoms, lamb wool wrapping can help to alleviate the discomfort problem (Fig. 24.8).

LESSER METATARSOPHALANGEAL JOINTS (SEE CHAPTER 20)

Metatarsalgia is uncommon in dancers, and when it is encountered, the differential diagnosis must include plantar plate injury with MTP instability and Freiberg's infraction.

Plantar Plate Tear and Metatarsophalangeal Instability

As the dancer *relevés*, the phalanx subluxes dorsally, pushing the metatarsal head plantarward and causing pain and overload of the plantar plate of the lesser MTP joints. In the *demi-pointe* position, excessive loads are transmitted through the second and third MTP joints. Clinical examination will elicit a translation in the anterior-posterior (AP) plane, positive drawer test, that is in excess of the adjacent joints.⁹ MRI can be a useful diagnostic tool, demonstrating either acute or chronic injury of the plantar plate (Fig. 24.9). Treatment initially is directed at taping to neighboring toes and stress-relieving padding. We have used platelet-rich plasma (PRP) injections and prolotherapy (saline solution) with success. Surgical correction includes a very limited resection arthroplasty with a plantar condylectomy, through dorsal approach. Alternately, a limited Weil osteotomy may be used with screw fixation. A formal plantar plate repair should be used with care, since it can cause joint stiffness, which is not tolerated by professional dancers. Motion should always begin early. Scarring at the plantar aspect of the wound facilitates tightening of the redundant plantar plate.



Fig. 24.9 Magnetic resonance image (sagittal plane) demonstrating injury of the plantar plate of the second metatarsophalangeal joint.

Freiberg's Infraction

Dancers have a propensity to develop Freiberg's infraction equal to that of the general population. In general, conventional radiography lags behind clinical symptoms by up to 6 months (Fig. 24.10, A, B and C). MRI facilitates early diagnosis.

Four types of infraction occur:

- Type I: A localized osteonecrosis of the metatarsal head that heals by creeping substitution. No cartilage defect is seen.
- Type II: Following metatarsal head osteonecrosis, the structural support of the head is lost. New bone formation occurs but is not sufficient to prevent collapse of the head. The articular cartilage is preserved; however, osteophytes on the dorsal lip limit dorsiflexion. Surgical debridement is curative, with exostectomy of the dorsal ridge to facilitate dorsiflexion.
- Type III: In addition to metatarsal head collapse, the articular cartilage is destroyed. Surgical management includes excision of the dead bone and cartilage and osteophyte resection. The plantar aspect of the joint usually is intact and can be left alone.
- Type IV: A rare entity with several heads involved. May represent a congenital epiphyseal dysplasia rather than a true infraction.

METATARSAL INJURIES

Second Metatarsal Base Stress Fracture (see Chapter 5)

Most high-level dancers have a mild cavus foot. Despite the mechanical advantages it brings, this anatomy creates vis-à-vis technique where the rigidity of the foot places high stresses on the bones during impact.¹⁰ In those dancers who start their careers early in life, the metatarsals hypertrophy and the cortices broaden to accommodate the increased stresses placed on them. In certain cases, however, stress fractures occur despite cortical hypertrophy, because the repeated microtrauma of dancing exceeds the reparative capacity of the bone.

Because of the cuneiforms' Roman arch configuration, the second metatarsal sits wedged between the medial and lateral cuneiform bones. This causes a relative rigidity to the second ray and consequently a potential site for a stress fracture. However, relative length of the second metatarsal is not considered a risk factor for fracture occurrence.¹¹ In fact, this is the most common site for a stress fracture in the dancer's foot, and when a patient complains of pain and tenderness in the base of the second metatarsal, it should be regarded as a stress fracture until proven otherwise.

The fracture line is usually transverse. Conventional radiographs may not show the fracture, but a computed tomography (CT) scan or an MRI will confirm clinical suspicion in such instances (Fig. 24.11). As the second metatarsal hypertrophies from years of pressure in the *demi-pointe* position, it may have the appearance of a healing fracture. Again, MRI can be useful in determining the true diagnosis.

Acute injuries require a cam walker for up to 6 weeks to allow time for the fracture to consolidate. Cast immobilization usually is not required, provided that the dancer can be trusted to keep the cam walker in place as prescribed. Rarely, a fracture may progress to a delayed union, and in these cases a small ultrasound bone stimulator can be used to accelerate healing. Occasionally, internal fixation with a small plate and screws, associated with bone grafting, may be required.

Nutcracker Fracture at the Base of the Second Metatarsal

Represents a different modality of common fractures in the base of the second metatarsal of professional dancers.¹² It is associated with specific anatomical findings of a wide forefoot and the use of tight shoes. There is repetitive impingement of the base of the first metatarsal into the base of the second metatarsal secondary to forefoot compression in the toe shoe. Often seen in young dancers with bunions and tight toe shoes who have not resized their toe shoes as they grow, but can also rarely occur in the absence of hallux valgus deformity, usually due to a valgus strain of the forefoot during *en pointe* (Fig. 24.12).

Fifth Metatarsal Spiral Diaphyseal Fracture (Dancer's Fracture)

These fractures occur when the dancer rolls over onto the lateral border of the foot from a *demi-pointe* position. The usual fracture pattern is a spiral, oblique fracture starting distal-lateral and running proximal-medial (Fig. 24.13). The average time to pain-free walking is 6.1 weeks; return to barre exercises around 11.6 weeks; and return to performance after 19 weeks.¹³ Recent literature supports excellent outcomes with conservative treatment,¹⁴ but surgical treatment might occasionally be needed in severely displaced fractures.

Proximal Fifth Metatarsal Spiral Fracture

The base of the fifth metatarsal has three different anatomic fracture zones: zone 1, tuberosity; zone 2, metaphyseal-diaphyseal junction (Jones fracture); and zone 3, diaphyseal stress. Proper identification of the zone affected is important, since healing characteristics and treatment modalities are usually different. Fractures can be classified as¹⁵:

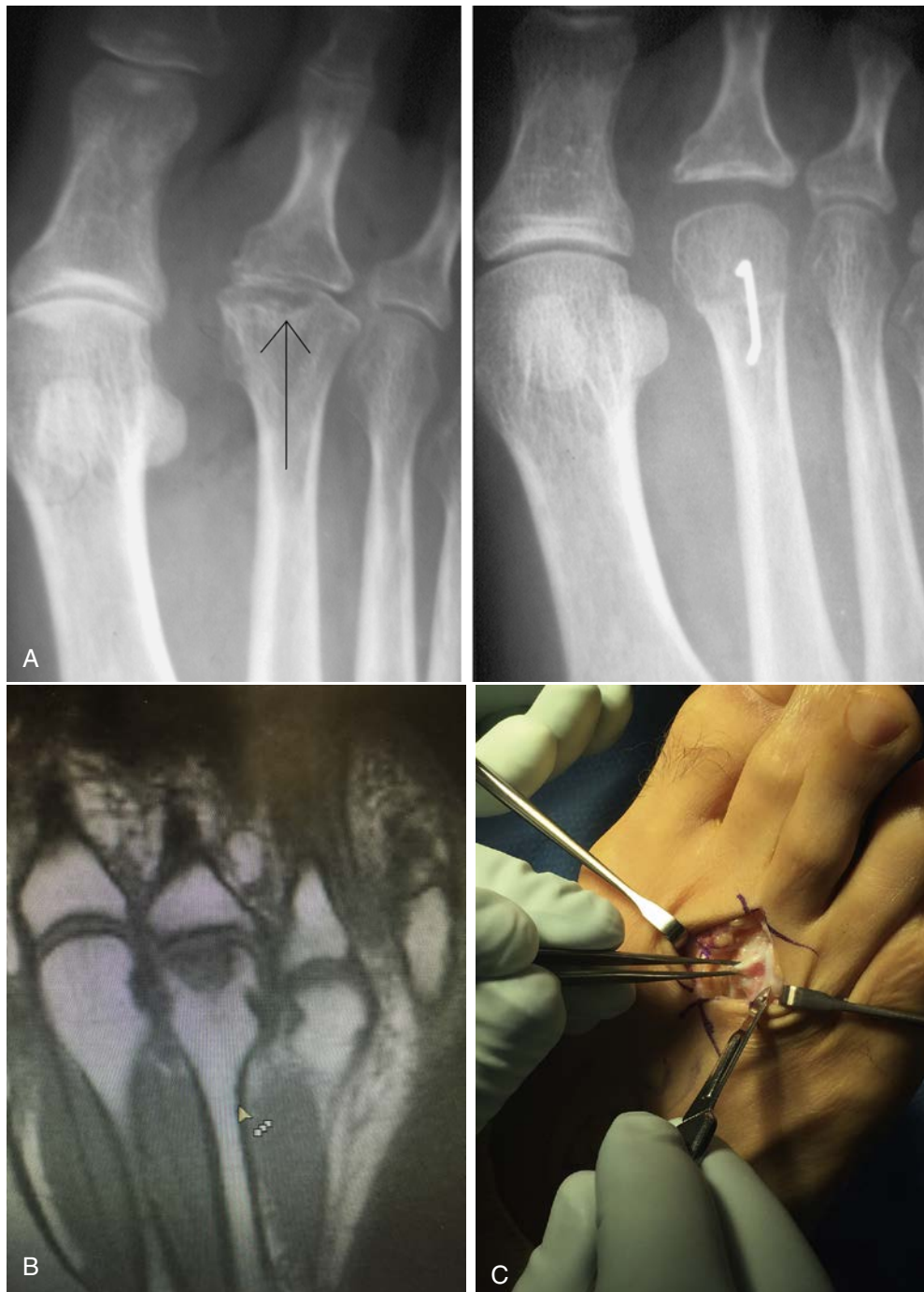


Fig. 24.10 Freiberg's Infraction. (A) Preoperative and postoperative anterior-posterior radiographs of the second metatarsal Freiberg's infraction; (B) transverse T1 magnetic resonance image of a third metatarsal Freiberg's infraction; (C) intraoperative findings with cartilage delamination.

- Type I: Avulsion fracture on the lateral aspect of the fifth metatarsal tuberosity, extending proximally into the metatarsocuboid joint. Usually caused by sudden inversion of the foot, the peroneus brevis attachment is avulsed, in addition to the lateral band of the plantar fascia and the abductor digiti minimi. In general, this injury can be treated with immobilization and functional rehabilitation and rarely requires surgical intervention. Fibrous union will invariably occur even in the presence of significant distraction of the fragments. In a skeletally immature dancer, this apophysis will

not have ossified and the fracture will not be visible on plain radiographs. The diagnosis must be made clinically. MRI can supplement the diagnosis. The treatment is similar.

- Type II (Jones fracture): Fracture line usually begins laterally in the distal part of the tuberosity and extends obliquely and proximally into the medial cortex at the level of the base of fourth and fifth metatarsal articulation. Subtle cavus-varus alignment and metatarsus adductus (supinated forefoot) are commonly associated anatomic deformities. These fractures are difficult to treat in a dancer because extensive

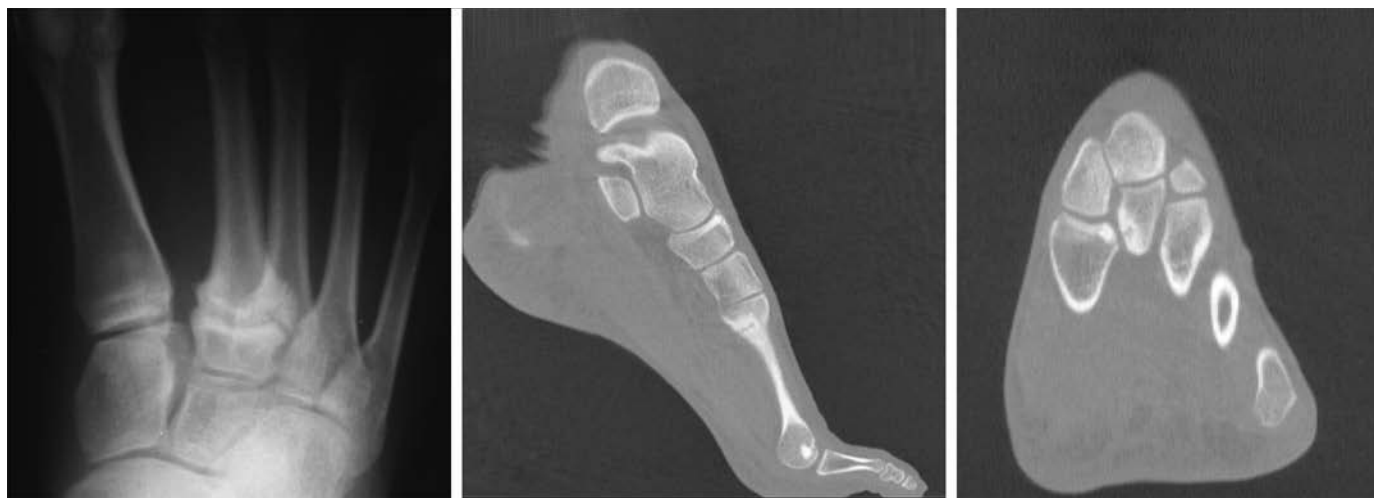


Fig. 24.11 Stress Fracture of the Base of the Second Metatarsal in Dancers. Radiographic anterior-posterior view and computed tomography scan, sagittal and transverse plane views demonstrating the transverse fracture line.



Fig. 24.12 Nutcracker Fracture at the Base of the Second Metatarsal. Radiographic anterior-posterior view and zoom demonstrating compression around the forefoot (arrows) driving the base of the first metatarsal to impinge in the base of the second metatarsal, creating a stress area and a traditionally oblique fracture line. Valgus strain of the forefoot during *en pointe* acts as a deforming force.



Fig. 24.13 Dancer's Fracture of the Fifth Metatarsal. Anterior-posterior and lateral radiographic views demonstrating the spiral oblique fracture line running from distal-lateral to proximal-medial.



Fig. 24.14 Jones Fracture in Dancers. (A) Radiographic oblique view of an acute fracture; (B) anterior-posterior and lateral views of a fracture fixed with single intramedullary screw and bone graft.

time in a nonweight-bearing immobilization is required. Because of the high rate of nonunion and refracture with conservative treatment, we recommend percutaneous intramedullary screw fixation to decrease time lost from dancing. Bone graft and or bone marrow aspirate concentrate may be used as an adjunct to help osteosynthesis.¹⁶ If the screw is removed, the fracture has a significant risk of recurring (Fig. 24.14 A and B).

- Type III (proximal diaphyseal stress): Fracture distal to the fourth and fifth metatarsal base articulation right at the metaphyseal-diaphyseal junction is usually caused by acute or chronic overload. Similar treatment options as for type II fractures, where surgical treatment with single intramedullary screw and bone graft expedites return to dance activities.

Bunionette Deformity

Pain over bunionettes usually can be diminished with soft padding or paper surgical tape adhesive taping to reduce friction and callus formation. Surgical resection usually is reserved for a retired

dancer because the time to recovery from the procedure is extensive.

THE MEDIAL ANKLE

Although posterior tibial tendon pathology is relatively common in other sports, it is rare in dancers. The reasons for this are multiple. Typically, a dancer's foot is cavus, which tends to protect him or her from tibialis posterior pathology in comparison to a more planus foot. Also, when a dancer is in equines, the posterior tibial tendon is relatively shortened as the subtalar joint is inverted.

Medial Ankle Sprains

Medial ankle sprains occur infrequently and are associated with a pronated foot landing off balance, being more common in male dancers. Injury pattern depends on the position of the foot during the injury. If the foot is in plantarflexion, the anterior deltoid is maximally affected, and the tension is greatest in the deltoid in this position. Similarly, when the foot is flat on the

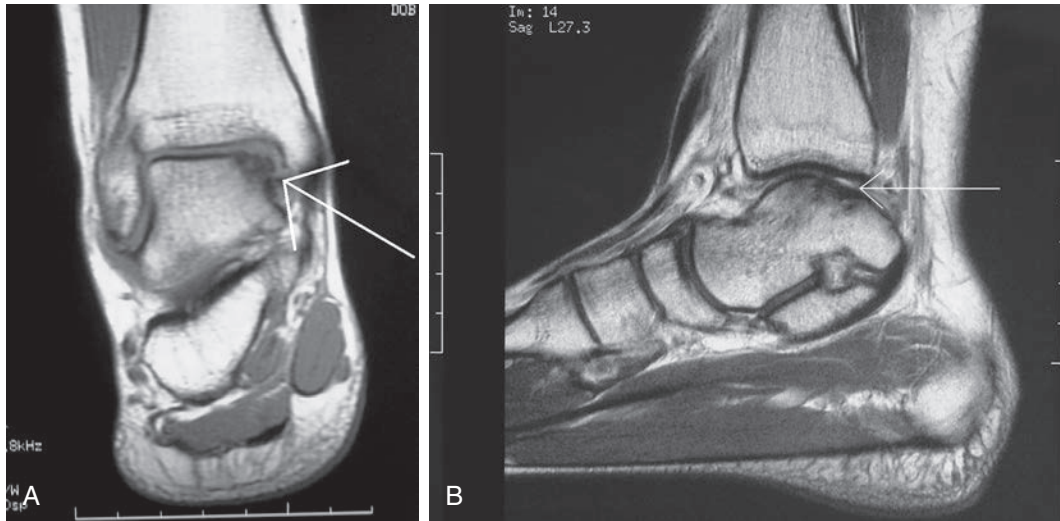


Fig. 24.15 Osteochondral Defect of the Talus in Dancers. Magnetic resonance image demonstrating a posteromedial talar dome lesion in (A) coronal and (B) sagittal T1 views.

ground and hyperpronated, the tear will occur in the midportion of the deltoid.

An accessory bone, the os subtile, can be occasionally found in the substance of the deltoid ligament. When injured, it may manifest as a trigger point of pain when ligamentous healing should be complete. A local injection of steroid is all that is required to treat this symptom.

Chronic strain of the deltoid from poor form in rolling in (pronation) of the foot is a common overuse injury in dancers. Chronic strain of the anterior aspect of the deltoid ligament, anchored to the capsule of the talonavicular joint, may predispose the ankle to chronic rotatory instability.

Recalcitrant medial ankle pain may also be caused by an osteochondral defect of the talus following a sprain. Clinical suspicion warrants further investigation with CT or MRI, which will demonstrate the extent of the lesion (Fig. 24.15 A and B). The size of the symptomatic osteochondral defect determines the most appropriate treatment. Arthroscopic debridement with microfractures, chondrocyte transplant, allograft implants, and osteochondral grafting are available techniques.

LATERAL ANKLE

Lateral Ankle Sprain

Lateral ankle sprain is a common injury in dancers and can often lead to recurrent instability and repetitive injuries.¹⁷ Chronic ankle instability can be found in up to 75.9% of the professional dancers, causing long-term limitations, especially in female dancers, who place extreme stress on their lateral ankle ligaments from being *en pointe* or *demi-pointe*.¹⁸

The anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL) are important stabilizers of the lateral ankle and are stressed at different ankle positions. The ATFL sprains in a plantarflexed and inverted foot, whereas the CFL is more prone to injury when the foot is dorsiflexed.

The subtalar joint can contribute to the occurrence of lateral ankle sprains. Dancers with decreased subtalar motion can transfer stresses to the ankle joint and being prone to lateral ankle sprains. It is important to check for decreased subtalar motion.

Another frequent associated condition in dancers with lateral ankle instability is hypermobility or generalized ligament laxity. Physical examination looking for findings of hypermobility is mandatory.

Acute lateral ankle ligament tears can be classified as:

- Grade I: Partial tear, usually of the ATFL. This is a stable injury, requiring rest, ice, compression, and elevation for 48 hours. Thereafter, motion is encouraged with a light compressive bandage. Dancers can begin light workouts at 48 hours with the aid of a brace or Aircast. Initially, therapy should concentrate on range of motion. After 4 or 5 days, dancers begin to wean out of the brace and initiate proprioception, balance, and peroneal-strengthening exercises.
- Grade II: Complete tear of the ATFL, occasionally including the CFL as well. A positive drawer sign is usually found, with a negative talar tilt test. Treatment is immobilization in a cam walker or Aircast for up to 6 weeks. Initially, physical therapy should focus on regaining appropriate range of motion. Thereafter, a triple-phase rehabilitation program including peroneal-strengthening, balance, and proprioceptive training should be initiated early.
- Grade III: Unstable injury. Both the ATFL and the CFL are injured. In addition to the drawer sign, the talar tilt test is positive. Ankle is severely unstable. Treatment traditionally is immobilization for up to 4 months. In a professional dancer, primary repair is preferred, and the Brostrom-Gould technique can be usually performed 1 week following the injury with predictable results and return of function.¹⁹

Regardless of the treatment used, attention must be paid to reestablishing a functionally stable ankle joint. Early functional treatment has been shown to produce the fastest recovery of ankle range of motion and earliest return to activity without affecting mechanical stability.²⁰ Closed chain balance and proprioception activities, along with peroneal muscle strengthening, will improve the neuromuscular control of the ankle. A therapist must be familiar with the modalities needed to achieve these goals to optimize outcomes in these dancers.

Residual symptoms following lateral ankle sprains in dancers may be secondary to:

- Avulsion fracture of the tip of the fibula,
- Avulsion fracture of the calcaneus at the calcaneocuboid joint,
- Accessory ossicle or os subfibularae,
- Calcaneus fracture or avulsion of extensor digitorum brevis,
- Fractured os peroneum,
- Fractured lateral process of talus,
- Fractured anterior process of the calcaneus,
- Talar dome osteochondral injury,
- Cuboid subluxation,
- Soft tissue entrapment,
- Sinus tarsi syndrome,
- Fractured os trigonum or Shepherd's fracture,
- Syndesmotric disruption,
- Maisonneuve injury,
- Bone edema,
- Anterolateral ankle impingement,
- Peroneal tendon dislocation or subluxation,
- Functional ankle instability, or
- Impingement of a lateral branch of the deep peroneal nerve (LBDPN).

The treatment for chronic lateral ankle instability in dancers is usually surgical. The Brostrom-Gould technique is again the preferred initial technique. It is always important to evaluate the patient for possible and frequent associated injuries that should be addressed concomitantly and include peroneal tendon pathology, talar osteochondral defects, and ankle impingement.²¹ In revision cases or patients with initial severe instability and generalized ligament laxity, the option is lateral ligament reconstruction with hamstring autograft.

Cuboid Subluxation

Cuboid subluxation is a common but poorly recognized condition in ballet dancers and might be resultant from prior repetitive lateral ankle sprains. Its symptoms include lateral midfoot pain and an inability to “work through the foot.” In addition, pressing on the plantar surface of the cuboid in a dorsal direction produces pain. The normal dorsal/plantar joint play is reduced or absent when compared with the uninjured side, and subtle forefoot abduction is present. Frequently, there is a shallow depression on the dorsal surface of the foot and palpable fullness on the plantar aspect of the cuboid. Radiographic, CT scan, or MRI evaluation is difficult because of the normal variations found in the relationship between the cuboid and its surrounding structures (Fig. 24.16). The diagnosis is primarily subjective, and must be made on the basis of the patient's history and physical findings. Treatment requires recognition of the condition and manual reduction. Close follow-up is imperative to certify that the cuboid remains in the correct reduced position.²²

ANTERIOR ANKLE (SEE CHAPTER 2)

The cavus foot is biomechanically ideal for dancers in that plantarflexion capacity is maximal. However, this is at the expense of dorsiflexion, which is frequently limited.

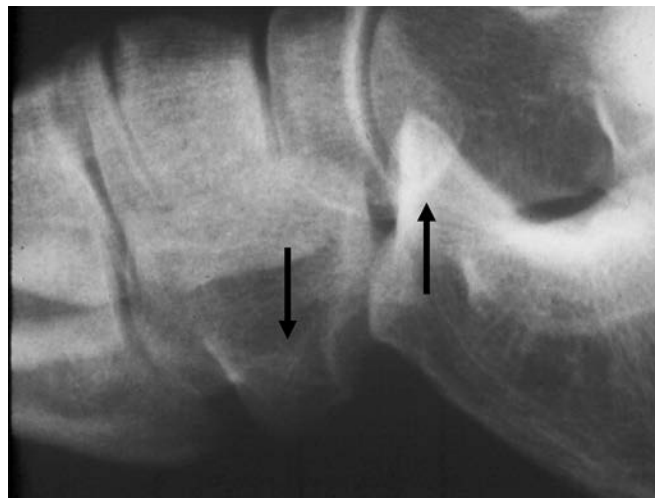


Fig. 24.16 Cuboid Subluxation in Dancers. Radiographic lateral view demonstrating the plantar subluxation of the cuboid at the calcaneocuboid joint.

Anterior impingement in dancers may be induced by repetitive dorsiflexion during *demi-plié*, where the anterior edge of the distal tibial articular surface contacts the dorsal neck of the talus, and is typically seen in male dancers who perform high jumps and deep *pliés* (bravura).²³ It also can be associated with the sequelae of lateral ankle sprain, including a hypertrophic tissue response, or simply by impingement of anatomically normal ligamentous structures. There are both osseous and soft-tissue causes of impingement symptoms. Impingement of the anterior lip of the tibia against the talus causes the cambial layer of the periosteum to produce reactive bone formation and osteophytes or “kissing lesions.”²⁴ This is a continuous cycle as more bone forms, and eventually motion is significantly restricted.

Three main types of lesions are seen (Fig. 24.17):

- Anterior tibial lip,
- Talar neck, or
- A combination of both.

Treatment for symptomatic cases is usually arthroscopic debridement, which has been shown to be an effective method for the treatment of bony and soft-tissue anterior ankle impingement in dancers, with minimal morbidity.²⁵ Special attention should always be directed to the medial aspect of the joint, where most of the osteophytes are usually located.

Extensive bone formation or degenerative arthritis might require an anterior or anteromedial arthrotomy.

POSTERIOR ANKLE (SEE CHAPTER 2)

Ideally, more than 100 degrees of plantarflexion should occur at the foot-ankle complex in a professional ballet dancer. Much of this has to be accomplished by the subtalar joint, and subtalar motion is facilitated by the turned-out position of mild forefoot pronation and abduction, unlocking the transverse tarsal joints. Any form of tarsal coalition, whether fibrous or bony, will prevent the subtalar joint from supplementing the ankle joint in full equinus. Consequently, most dancers with subtalar coalitions do not reach professional grade.



Fig. 24.17 Anterior Ankle Impingement in Dancers. (A) Radiographic lateral view, (B) computed tomography scan sagittal, and (C) three-dimensional reconstruction views of tibiotalar osteophytes.

Posterior Impingement Syndrome

The posterior tubercle of the talus varies greatly in size. In posterior impingement syndrome, either a large posterior tubercle or an os trigonum is caught between the posterior lip of the tibia and the calcaneus when the dancer is in *relevé*, resulting in repetitive and overuse injuries (Fig. 24.18 A, B and C).^{26–28} A simple clinical sign, the forced plantarflexion sign, confirms the diagnosis when pain is produced by full plantarflexion at the back of the ankle. The syndrome is usually a result of an os trigonum impinging the soft tissue rather than the bone itself.

The differential diagnosis includes Achilles tendinitis, peroneal tendinitis, or heel pain.

The os trigonum is present in up to 10% of the population and is bilateral in 50% of the patients. Anatomically, the os trigonum represents the nonfused lateral tubercle of the posterior process of the talus. The lateral and medial talar tubercles form a fibro-osseous tunnel where the flexor hallucis longus (FHL) tendon runs on the back of the ankle joint. Most cases of os trigonum are asymptomatic in the general population, and this is also true in dancers. However, in dancers this condition

is often unnecessarily operated. For this reason, a diagnostic anesthetic injection is advised before deciding on any surgical intervention. If there is no subsequent pain relief following the injection, differential diagnosis should be considered and include FHL tendinopathy, peroneal tendinopathy, and stress fracture of the posterior process of the talus.

Treatment of an os trigonum is generally nonsurgical. Once a diagnosis has been confirmed by local anesthetic injection, the next step is rest and activity modification. Local steroid can give dramatic relief of symptoms that often is long lasting or permanent. When surgery is required, arthroscopic or open resection can be performed with similar reported outcomes. However, patients treated endoscopically returned to full dance earlier (mean of 9.8 weeks) than those undergoing open excision (14.9 weeks).^{29,30} For open resection either a posteromedial or posterolateral approach can be used.³¹ In cases in which it is suspected that there is an associated FHL pathology, a posteromedial approach is preferred so that tenolysis can be performed safely. The tendon debridement can be also performed arthroscopically.³²

Posterior impingement may also occur in association with chronic lateral ankle instability. The talus slips forward and the posterior lip of the tibia impinges on the *calcaneus*. Treating the lateral ligament instability usually addresses this form of impingement.

Flexor Hallucis Longus Tendinopathy

The FHL is the most common site of lower extremity tendon disorders in ballet dancers. This entity has become known as “dancer’s tendinitis.” As the tendon passes between the fibro-osseous tunnel at the back of the talus, it runs deep to the sustentaculum tali. Within this pulley system it can become inflamed and cause irritation and swelling. Reduced vascularity is also an important factor contributing to tendon degeneration and rupture under strain.³³

When the tendon has a thickening or a partial tear at a particular area, it may cause triggering (Fig. 24.19). This condition is known as hallux saltans.^{34,35} When the tendon becomes completely stuck down within the pulley system, a

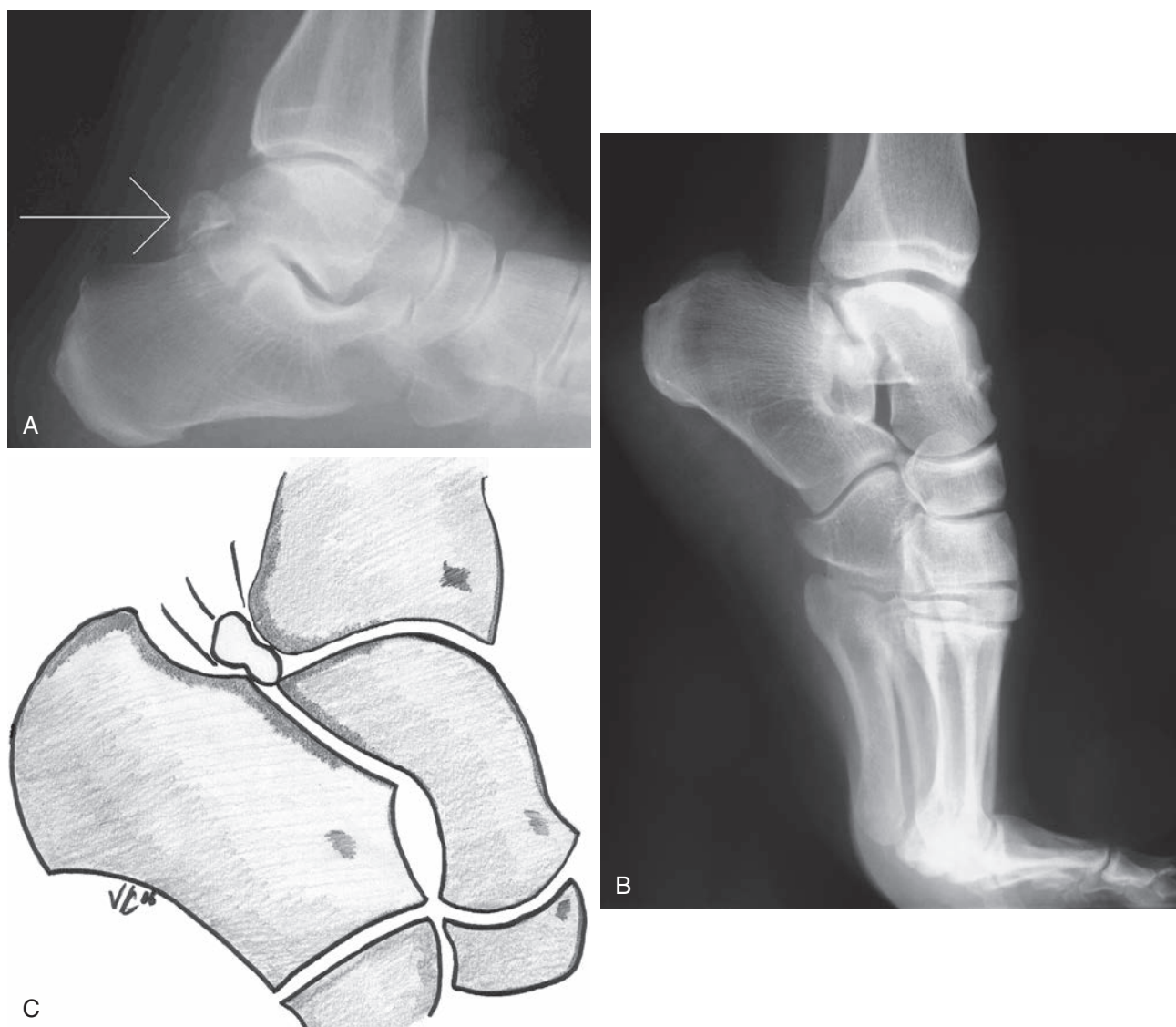


Fig. 24.18 Posterior Ankle Impingement in Dancers. (A) Lateral radiographic view of an os trigonum; (B) lateral radiograph of an os trigonum in *relevé* position; (C) illustration of os trigonum posterior impingement.

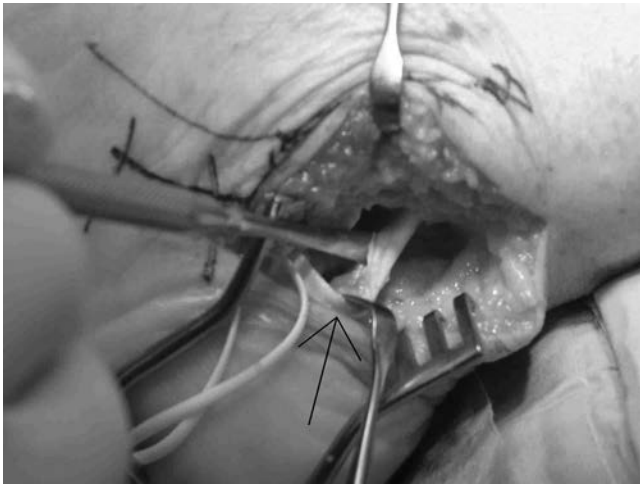


Fig. 24.19 Flexor Hallucis Longus Tendinopathy. Intraoperative finding.

pseudohallux rigidus can be seen. Dancers tend to have symptoms for a longer period of time before seeking treatment than non-dancers.³⁶

A trial of conservative treatment can be tried with resting and antiinflammatories. Local steroid injections should be avoided whenever possible secondary to increased risk of tendon rupture. However, this condition is frequently persistent and disabling, and open surgical tenolysis is frequently needed, with good reported results.^{36,37} Arthroscopic treatment is also described.³²

Three areas of tendon pathology are typically found. The most common location is behind the medial malleolus. It may also be found at the knot of Henry, or at the base of the first metatarsal where the tendon passes beneath the sesamoid bones.

ACHILLES TENDON (SEE CHAPTER 9)

As the largest tendon in the body, the Achilles tendon incurs forces up to six times body weight during running and jumping. Optimal lower leg function is critical for ballet dancers to meet their occupational requirements. The tendon is commonly injured in dancers either from repetitive overload or excess stress applied by poor technique. Although a common site of injury in dancers, the tendon is rarely ruptured in this group of athletes.³⁸

Peritendinitis of the Achilles Tendon

The Achilles tendon has no real synovial sheath and is surrounded by a peritendon, which can become inflamed from overuse or from the tight ribbons of ballet shoes. The peritendinitis is classically seen as a diffuse swelling along the Achilles tendon. When the tendon itself is inflamed, it presents as a discrete swelling along the tendon. Treatment of peritendinitis requires rest. A cam walker with a heel-raise insert worn for 23 hours/day should be worn for at least 2 weeks. This can break the cycle of inflammation and prevent the next step in the continuum of pathology—pathology of the tendon itself.



Fig. 24.20 Lateral radiographic view of a Haglund's deformity in a dancer with cavus-varus foot.

Tendinosis of the Achilles Tendon

Tendinosis is caused by microtears of the collagen fibers on the surface or in the substance of the tendon. Its most common form occurs at the isthmus of the tendon and involves a localized swelling of the pseudosheath. This may be felt clinically as crepitus when the tendon is stretched and relaxed—the so-called painful arc sign. Chronic tendinosis can be felt as multiple nodules on the surface of the tendon. More severe strains result in a classic fusiform swelling of the tendon. This is slow to heal and carries a guarded prognosis.

Treatment requires rest initially, usually in a cam walker with a heel-raise insert for up to 6 weeks. Failure to comply with strict immobilization initially can result in prolonged symptomatology and rupture of the tendon. Treatment can be supplemented with antiinflammatory medication. Rehabilitation consists of stretching exercises and a gradual strengthening program. In more chronic cases, use of an overnight splint to assist with a prolonged stretch in a dorsiflexed position can be helpful. Orthotic prescription may be considered to help correct any structural imbalances in the foot. A “stretch box” is a useful tool to prevent injury that is used by many ballet schools. This allows dorsiflexion of the ankle with stretching of the Achilles tendon before and between performances. However, dancers must exercise caution to avoid stretching the Achilles tendon too aggressively, causing more tears and thereby worsening the condition.

Certain factors can predispose to Achilles pathology in dancers:

- Cavus foot with associated Haglund's disease (Fig. 24.20), with tendinitis of the Achilles overlying the retrocalcaneal bursa. Cavus feet are common in this population because they afford the dancer a distinct anatomic advantage. For this reason, any prominence at the posterosuperior aspect of the calcaneus can cause irritation of the tendon and may necessitate surgical resection of the prominence and debridement of the tendon (Fig. 24.21 A and B).
- “Rolling in” or pronation of the foot.
- “Ribbon burn” from tight toe ribbon at the back of the leg.

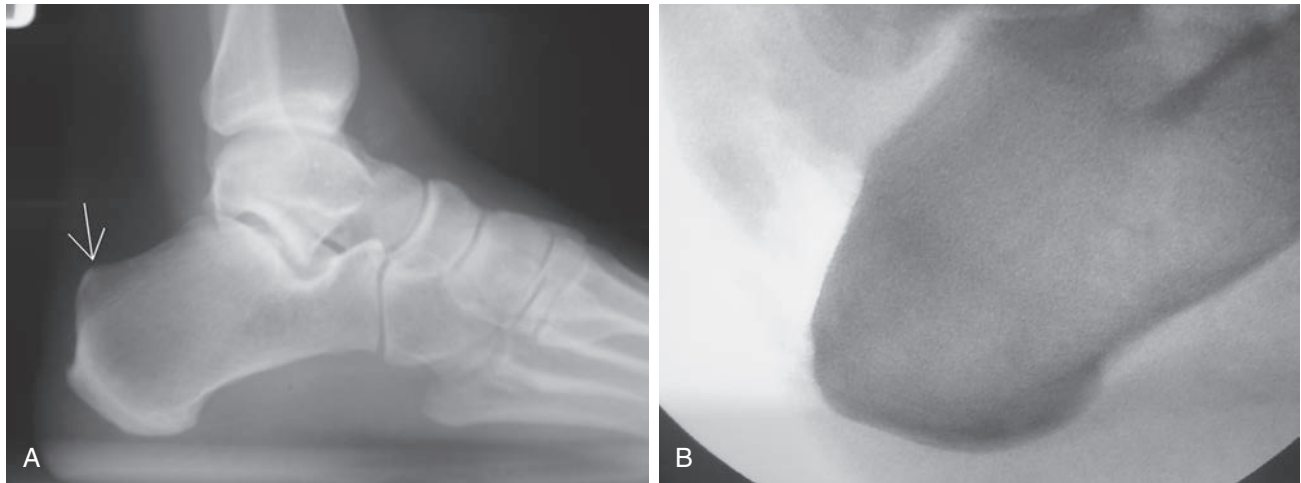


Fig. 24.21 Haglund's Deformity in Dancers. (A) Preoperative lateral radiographic view and (B) intraoperative lateral fluoroscopic view after resection of the superior aspect of the posterior calcaneal tuberosity.

- Congenitally thin tendon predisposed to overload injury.
- Gastrocnemius contracture.

Rupture of the Achilles Tendon

Achilles tendon ruptures are rare in female athletes and more common in male dancers older than 30 years. Typically, a tear presents as a sharp pain of sudden onset and an inability to walk on the toes. A Thompson test is the best clinical diagnostic test. Feeling for a defect along the tendon usually is diagnostic; however, an intact peritendon filled with hematoma may mimic an intact tendon. Ultrasound or MRI can confirm the diagnosis with a high degree of sensitivity and specificity. Treatment is dependent on the requirements of the patient. Conservative treatment was initially associated with a high rate of reruptures. However, more recent studies have demonstrated similar functional outcomes and rerupture rates when comparing conservative functional treatment and surgical treatment.³⁹ In athletes, the operative intervention is still preferred with the advantage of restoring the physiologic length of the tendon and optimizing functional outcome.⁴⁰ This requires up to a full year of treatment and rehabilitation before the dancer can return to preinjury levels of dance. Limited open techniques with percutaneous suturing facilitate early motion and reduce the risk of associated skin problems.⁴¹ Correct tensioning of the repair is critical to outcome regardless of the technique used.

Pseudotumor of the Calf

An accessory soleus muscle can present as a slowly enlarging mass on the medial side of the calf. It generally is painless, usually presenting as a feeling of tightness. Surgical division of the muscle sheath may be required and will generally relieve the symptoms.

HEEL PAIN (SEE CHAPTER 11)

Plantar Fasciitis

Pain on the medial aspect of the fascia origin is the most common presentation. The presence of a spur usually is not the cause of heel pain, despite often impressive radiographic evidence.

The plantar fascia is not intimately attached to the spur, giving rise to the flexor digitorum brevis.

Stretching of the fascia before rehearsing or performing can reduce the incidence of this injury. Also, using a firm rubber ball for rolling into the plantar fascia while weight bearing helps to loosen the fascia and make it more pliable. A silicone heel can also give symptomatic relief in a dancer who has point tenderness in this area.

Plantar Calcaneal Bursitis

Found beneath the calcaneus, this condition usually can be diagnosed clinically; however, ultrasound can confirm the diagnosis.

Baxter's Nerve Neurapraxia

The first branch of the lateral plantar nerve or nerve to abductor digiti minimi may be trapped under the deep fascia of the abductor hallucis.⁴² This is exacerbated when the dancer "rolls in" or pronates. Although the cause is a neurapraxia of the lateral branch of the plantar nerve, the condition is painful on the medial aspect of the heel, adjacent to the medial calcaneal tuberosity. A local anesthetic is the treatment for recalcitrant cases.

LEG PAIN (SEE CHAPTER 23)

The three primary conditions in dancers that predispose to leg pain include shin splints, stress fracture, and compartment syndrome.

Shin Splints (Medial Tibial Stress Syndrome)

Shin splints is a generic term often used to describe both traction periostitis and stress fractures of the tibia. It has gained credence in the general population to describe generalized leg pain. A more useful nomenclature is medial tibial stress syndrome (MTSS). For the purposes of this discussion, MTSS describes a traction periostitis alone. This condition is associated with a diffuse anteromedial or posteromedial tibial pain. Typically, the pain is in the distal one-third of the tibia. It can be differentiated from stress fracture, which has localized point tenderness and

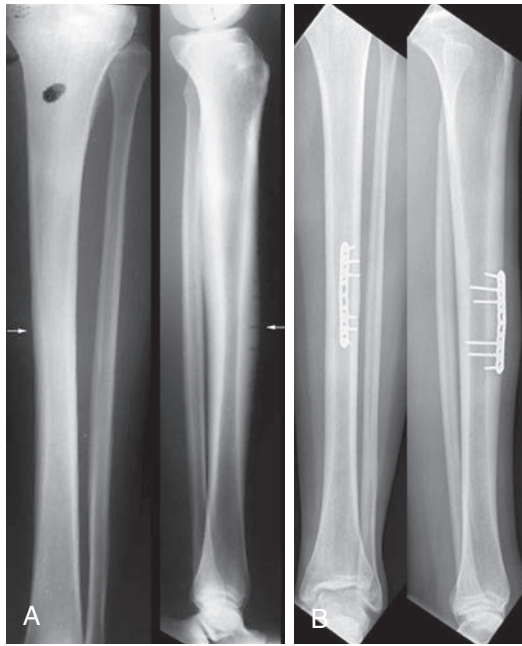


Fig. 24.22 Anterior Tibial Shaft Stress Fractures in Dancers. (A) Pre- and (B) postoperative anterior-posterior and lateral views.

is usually located in the mid-diaphysis of the tibia and distal one-third of the fibula.

Typically, MTSS occurs at the beginning of the season after a prolonged period of inactivity. Stress fractures, on the other hand, happen secondary to repetitive trauma and occur usually in mid to late dance season.

MTSS can be localized anteriorly or posteriorly. Posterior MTSS is the most common in dancers and arises at the origin of the flexor digitorum longus (FDL) muscle, and not from the tibialis posterior, which arises from the interosseous membrane. Anterior MTSS, not as common in dancers, represents a periostitis at the origin of the tibialis anterior muscle.

Stress Fractures

Prolonged biomechanical imbalances and increased repetitive loads beyond the body's reparative capacity typify the causes of stress fractures. Thus these injuries generally occur at the end of the dancer's season, in contradistinction to MTSS, which usually occurs at the beginning. Initially, radiographic findings may be subtle with mild periosteal reaction, and the best method of confirming a clinical suspicion is MRI. As the process progresses, conventional radiographs may reveal the fracture line that is usually located on the anterior aspect of the tibia and better seen in the lateral view (Fig. 24.22). The presence of the line is an indicator of severity and that the fracture will be slow to heal, requiring an extremely long period of 6 to 8 months of protected weight bearing, not acceptable for a competitive dancer. We advocate anterolateral compression plating for those dancers with localized disease, but if there are multiple lines, intramedullary fixation with a locked rod is the treatment of choice. Since the introduction of Balanchine method of dance, which emphasizes fluid

motion, the number of stress fractures has reduced. This is in contrast to the rapid deceleration motion seen in the Bravura technique.

Compartment Syndrome

When the pressure within an enclosed fascial compartment exceeds the pressure required to perfuse the muscle with blood, the muscles and enclosed structures may become compromised. This can lead to pain initially and may reach the point of muscle ischemia in more severe cases. In dance, the blood volume to the exercising muscle can increase up to 20%, thereby exceeding the physiologic pressure within the muscle compartments. Most cases of exertional compartment syndrome involve the anterior compartment or the deep posterior compartment. Normal resting compartment pressures range from 0 to 8 mm Hg. During exercise this can increase to 50 mm Hg. Following exercise, this pressure should fall to 15 mm Hg within 15 minutes. Treatment of exertional compartment syndrome usually is conservative, with antiinflammatory medication and shoe modification and activity modification. Rarely, a fasciotomy is required.

SUMMARY

Classical ballet offers a graceful and beautiful spectacle. This beauty comes at great physical, psychological, and economic cost to the ballet dancer.⁴³ It is estimated that up to 95% of dancers employed for greater than 1 year will suffer a significant injury. Most of these physical injuries occur to the foot and ankle in female ballet dancers.⁴⁴ Many of these injuries are as a result of dancing on the point of the toe. This form of dancing was first performed by Genevieve Gosselin in 1818 at the Paris Opera house. The illusion of weightlessness and the grace implied in *en pointe* dancing was further enhanced by the great dancers Taglioni and the immortal Istomina. Since their time, the beauty, romance, and grace of *en pointe* have been enjoyed by dancers all over the world. Unfortunately, the "cruel little slipper" that is the *en pointe* shoe, as well as the physical demands of the dance itself, has left many dancers with significant injuries and permanent deformities. It must be emphasized that, when the orthopedist examines a ballet dancer, the entire kinetic chain requires close inspection. Isolated injuries to the foot and ankle may precipitate additional injuries farther up the kinetic chain as a compensatory response to the injury or inadequate and improper rehabilitation.⁴⁵ Apart from the physical examination, a careful history and biochemical profile should be investigated in those dancers showing any signs of the aforementioned dancer's triad: anorexia, amenorrhea, and osteoporosis.⁴⁶ In addition to the biomechanical examination and biochemical evaluation, the orthopedist should be cognizant of the psychosocial aspects of a dancer's makeup. Dancers, in general, regard injury and pain as a way of life and are reluctant to present to health care professionals for fear of long-term immobilization and eventual unemployment.⁴³ As an advocate for the dancer as an athlete, the clinician should be aware of these concerns and strive to provide an accurate diagnosis and expeditious treatment strategy.

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Video Legends - <https://www.kollaborate.tv/link?id=5c9d20f756b57>

Video 24.1 Title: About Dance Smart.

Video 24.2 Title: Airplane Test.

Video 24.3 Title: Example of modern dance.

Video 24.4 Title: Example of musical theater dance.

Video 24.5 Title: Example of newer dance form example blend of jazz/ hip.

Video 24.6 Title: Jete.

Video 24.7 Title: Neural Mobility.

Video 24.8 Title: P to TO Yard Stick Test.

Video 24.9 Title: Pirouette.

Video 24.10 Title: Plie Passe.

Video 24.11 Title: Plie.

Video 24.12 Title: ROM–Beighton Scale.

Video 24.13 Title: ROM–hip rotation.

Video 24.14 Title: SauteTest.

Video 24.15 Title: Stability–Hip.

Video 24.16 Title: Stability–Toes.

Video 24.17 Title: Stability Core.

Video 24.18 Title: Tendu.

Video 24.19 Title: Torsion–Hip Knee.

Video 24.20 Title: Triple Hop.

An International Perspective on the Foot and Ankle in Sports

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BRAZILIAN FOOT AND ANKLE INJURIES IN SPORTS

Epidemiological studies on sport injuries occurring at the foot and ankle are not consistent and lack uniformity in published outcomes in Brazil. Current literature on sports injuries in the country indicates that soccer is the most frequently investigated and the first source of injury.¹ Research has shown that soccer players have a 1000-fold increased risk of injury compared to industrial workers.¹ Approximately 15.3 million people in Brazil play soccer, which represents 39.3% of all sports practiced in the country.² Barefoot sports such as beach soccer and beach volleyball have also been gaining participants due to Brazil's advanced placements in recent international tournaments.

Among sport injuries, after the thigh, the foot and ankle are the most common locations for injury.³ In one epidemiological study, severe ankle and foot problems occurred in 33% of the 200 healthy soccer players over 2 years of prospective observation.⁴ Direct player-to-player contact (32%) and overuse (26%) are the most commonly cited mechanisms of injury.⁵ Of these, by far the most common injuries are sprains (80%) (which includes foot sprains such as Lisfranc lesions), followed by bruises (9%–49%) and tendon lesions (2%–23%). Fractures are rare and account for only 1% of all ankle injuries in soccer.⁶ Except for Achilles tendinopathies, which are higher during the preseason, most injuries occur during competition.⁵

Here, we briefly describe some of the most common injuries that are seen in our clinics, including subtle Lisfranc injury, sand-toe, posterior ankle impingement, and loose bodies of the ankle joint.

The Subtle Lisfranc Ligament Lesion

In athletes, Lisfranc lesions are mostly the result of indirect trauma, occurring as plantar flexion or abduction injuries.⁷ Plantar flexion injuries often occur when the player suffers a slide tackle and an axial load is applied to a plantar flexed foot while the toes are dorsiflexed through the metatarsophalangeal (MTP) joints. The ligamentous complex at the tarsometatarsal (TMT) joints usually fails dorsally as rotational forces are applied to the midfoot, with resulting subtle joint subluxation or dislocations. Abduction pattern injuries are also common in Brazil and occur when the forefoot is suddenly abducted relative to a fixed hindfoot, such as in equestrian sports in which riders have their feet stuck in the stirrup and may suddenly be thrown out of the saddle.

Early diagnosis of Lisfranc injuries is critical to allow for appropriate treatment and a speedier return to play. Misdiagnosis or maltreatment of these potentially career-ending injuries may not only prolong return to competitive play but can also lead to posttraumatic degenerative changes and pain that limit activity and quality of life in the future. Patients with subtle Lisfranc complex injuries may present with varied degrees of swelling throughout the midfoot, though they reliably report pain with attempted weight-bearing activity.⁸ Several tests are described, but a high index of suspicion is paramount for diagnosing these injuries. Radiographic studies are critical. If it is too painful for patients to cooperate with adequate standing x-rays, a couple of days of rest or intra-articular local anesthetic can be administered (Fig. 25.1).

Excellent results have been reported with nonoperative treatment when less than 2 mm of diastasis occurs.⁹ However, a low tolerance for displacement should be accepted in elite players, as some recent studies have shown a trend that a surgical approach results in an earlier return to play.^{8,10}

The intraoperative finding of proximal extension of a Lisfranc injury into the intercuneiform area and naviculocuneiform joints is not uncommon. Small intercuneiform diastasis can be misdiagnosed and represents a less stable injury pattern that demands adequate open reduction and internal fixation (Figs. 25.2 and 25.3).



Fig. 25.1 Subsequent weight-bearing x-ray after 1 week of rest demonstrates widening at Lisfranc joint.



Fig. 25.2 A professional goalkeeper (same as Fig. 25.1). Dorsal approach for subtle Lisfranc injury.

Subtle Lisfranc lesions in athletes are not typically associated with a severe degree of swelling. Therefore, in subtle lesion cases swelling would not limit the ability to proceed with surgery as soon as feasible. The authors generally attempt to avoid penetrating cartilage surfaces at internal fixation up to 3 months after injury. In cases where the initial diagnosis was missed, or treatment is delayed for more than 6 weeks, open reduction of the involved joints is required to remove the thickened scar tissue between the joints, preventing a stable closed reduction. Dorsal bridging plates with or without a C1M2 screw may be a better option for high-impact athletes and contact players (Fig. 25.4).



Fig. 25.3 A proximal intermetatarsal lesion is appreciated with manual stress.



Fig. 25.4 Dorsal plating after extensive removal of thickened scar tissue. This patient presented 6 weeks after the trauma.

Our postoperative care consists of a below-knee nonweight-bearing boot used for 6 weeks. Foot and ankle range-of-motion (ROM) exercises are initiated at 3 weeks. At 2 months, patients are allowed to wean out of the boot as tolerated. Plates and screws are routinely removed during the fourth month, and patients are permitted to fully weight bear on the limb.

Beach Toe Lesions

Barefoot sports seem to have a surprisingly low number of injuries, sometimes even lower than the rates for athletes wearing shoes and doing the same or similar activities. For instance, in beach volleyball, injury rates have been found to be fourfold lower during competition on the sand compared to indoors.¹¹

Competing barefoot on a sand interface may pose a unique risk for injury that is mainly exclusive to beach volleyball, known as “sand toe.” The mechanism of this injury is a hyper-plantarflexion sprain of the great toe, which usually occurs when the player steps down unsteadily and shifts forward, catching the big toe in the sand (Fig. 25.5). This stresses the dorsal capsule of the MTP joint. This is essentially the opposite mechanism of injury of turf toe.¹² Clinically, athletes present with tenderness at the dorsum of the MTP joint capsule and pain in the same area with toe plantarflexion, which is worse with passive stretching. Active dorsiflexion is not as painful, and strength is typically intact.

Diagnosis is made clinically, but x-rays should rule out fractures or avulsions. A magnetic resonance image (MRI) should be reserved for severe lesions with likely extensor hallucis longus (EHL) weakness, when dorsiflexion strength does not get better after 6 to 8 weeks.

Treatment starts with taping that is typically continued for the remainder of the competitive season, or until the toe can be plantarflexed passively and fully without pain. On the other hand, rupture of the dorsal hood resulting in EHL subluxation may result in pain that is most pronounced with resisted



Fig. 25.5 Mechanism of the sand toe injury—hallux in hyperflexed into the sand.

dorsiflexion and may need surgical repair. In a series of 12 patients, the average player took 6 months to fully recover from the injury.¹² Beach volleyball players will probably benefit most if they do some portion of their rehabilitation on an uneven sand surface, since this is the surface on which they will be returning to play.

In beach soccer, most injuries occur in the feet and toes (36.4%), followed by the Achilles tendon (18.2%).¹³ A specific clinical syndrome characterized by progressive pain and swelling in the dominant (kicking side) hallux MTP or interphalangeal (IP) joints can lead to an osteochondral injury of the hallux (Fig. 25.6). Hyperextension or hyperflexion with repetitive trauma of the hallux MTP or IP joints is the postulated mechanism of injury. Radiographic evaluation can show a marginal, often sclerotic, bony fragment within the lateral or medial aspect of the symptomatic MTP or IP joints. This fragment usually represents marginal osteonecrosis that can be confirmed with MRI. When a symptomatic bony fragment is identified within the MTP or IP joints of the hallux, simple excision has been reported to predictably relieve symptoms.¹⁴

Posterior Ankle Impingement

Posterior ankle impingement syndrome (PAIS) is a clinical disorder defined as a painful limitation of ankle ROM—more specifically, forced plantarflexion—usually caused by repetitive stress. Causes include neurovascular lesions that involve the Stieda process, malunion fractures of the posterior malleolus or talus, os trigonum syndrome, flexor hallucis longus stenosing tendinitis (Figs. 25.7 and 25.8), osteochondral lesions (OCLs), and retrocalcaneal bursitis.¹⁵

Among soccer players, PAIS symptoms arise more commonly when the soccer player sprints or hits the ball, which results in forced plantarflexion of the ankle. Approximately 60% of conservatively treated athletes show improvement of their

symptoms by changing sports habits and wearing braces to limit plantarflexion.¹⁶

Os trigonum is present in 8% to 13% of the general population and can impinge against the tibia and calcaneus, especially in ballet dancers and soccer players.¹⁷ When symptoms cannot be relieved by conservative measures, athletes tend to compensate the loss of plantarflexion by placing the foot in a suboptimal position in order to diminish symptoms, which leads to further injuries. Therefore, surgical treatment should not be delayed in order to avoid other related lesions (Fig. 25.9).

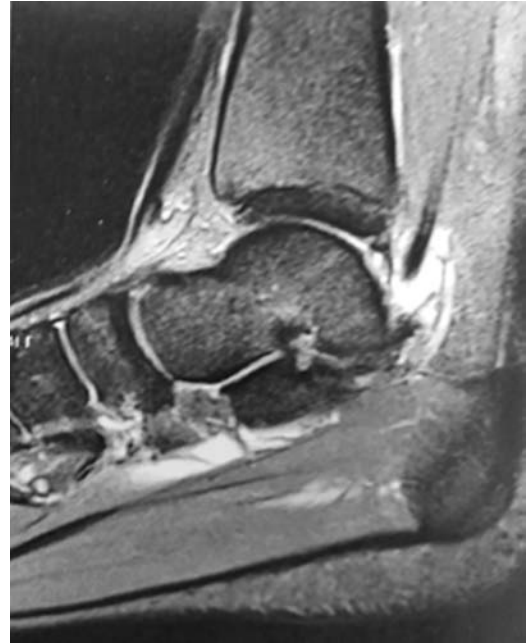


Fig. 25.7 Flexor hallucis longus tendinitis caused by impinging effect from Os Trigonum Syndrome.



Fig. 25.6 Osteochondral injury of the hallux in beach soccer.

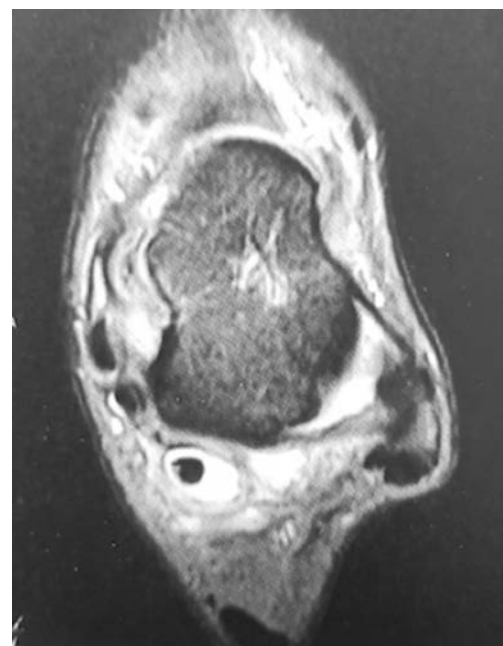


Fig. 25.8 Flexor hallucis longus is involved with extensive tenosynovitis, proximal to its tunnel.

We prefer to endoscopically treat these patients through two para-Achilles portals previously described in the literature,¹⁸ because we believe faster return to play can be achieved. Return to play usually occurs around 40 days. Full weight bearing with crutches is allowed immediately, and after 1 week can be done without crutches or a cane. Sports activity is resumed on their second postoperative week and field training with their respective teams from week 4 thereon.

When resecting the Os Trigonum, one should release all bands involving the flexor hallucis longus (FHL), since a constricting effect may arise from scar tissue there (Figs. 25.10 and 25.11).

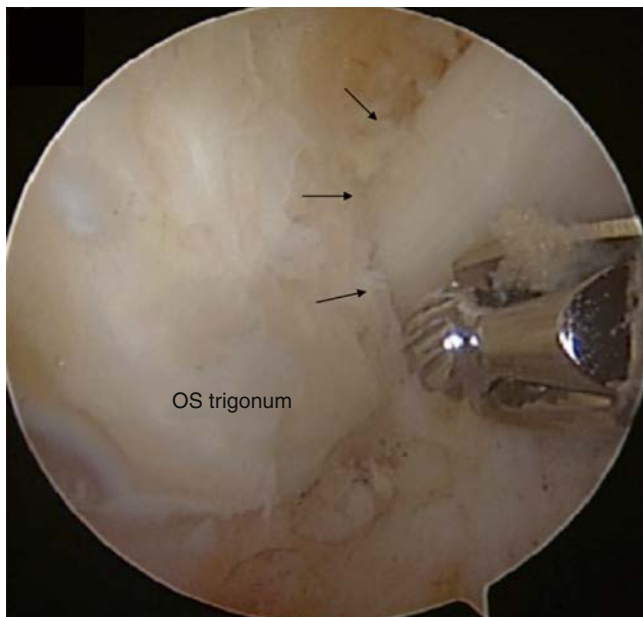


Fig. 25.9 Os Trigonum impinging against posterior subtalar and ankle joints. Note the narrowing of the flexor hallucis longus tunnel.



Fig. 25.10 Os Trigonum (OS) being resected. Fibers bands around flexor hallucis longus are seen (*).

Loose Bodies of the Ankle Joint

Loose fragments of the ankle joint in athletes usually result from anterior bone impingement with fracture of the anterior osteophytes (Fig. 25.12). The mechanism of loose bodies is related to recurrent ball impact or the repetitive hyper-dorsiflexion, which occurs in high-impact sports, such as volleyball and running. The classic presentation is anterior ankle pain (especially with compression of the soft tissues against the anteromedial aspect of the ankle), restriction of dorsiflexion, and swelling. When fracture of the osteophytes occurs, pain may go away and loose bodies may deposit into lateral or medial gutters.

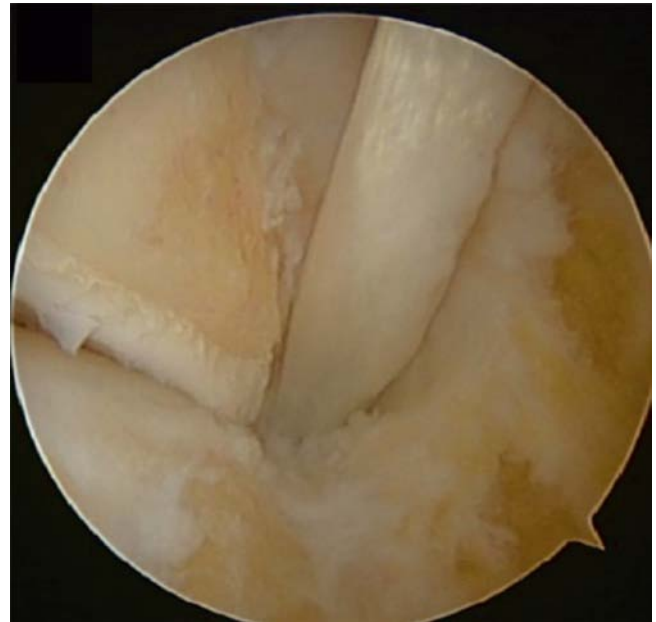


Fig. 25.11 After removal of the Os Trigonum and soft tissues, subtalar surface and flexor hallucis longus tunnel are well delineated. Care must be taken not to over-resect bone tissue and damage healthy cartilage.



Fig. 25.12 Loose body in the anterior compartment of the ankle.

Lateral ankle radiographs reveal osteophytes on the anterior tibia and talar neck (kissing osteophytes). The diagnostic value of an oblique radiograph in addition to a lateral radiograph has been shown. When the lateral radiograph was combined with an oblique anteromedial radiograph, the sensitivity and specificity of the method were high.¹⁹ MRI is useful to rule out any associated soft tissue lesions (Fig. 25.13).

We have a low threshold to maintain athletes in conservative treatment. Arthroscopy is usually indicated when diagnosis is confirmed. Results of ankle arthroscopic soft-tissue debridement and osteophyte resection are good, satisfaction is high (94% to 98%), and the rate of major complications is as low as 1%.²⁰

Postoperatively, the ankle is immobilized with a bandage, and partial weight bearing is applied for 3 to 5 days. Full weight bearing is allowed 5 days after surgery, and postoperative rehabilitation is started. Athletes usually return to complete activity after 4 weeks.

Fifth Metatarsal Stress Fracture

Fifth metatarsal stress fractures account for less than 2% of metatarsal fractures in college football players, but can be a source of significant temporary disability and loss of playing time.²¹

Athletes typically report an acute episode of lateral foot pain that, in some cases, follows a 1- to 2-month prodromal history of a lateral foot “ache.” They also often report mild to moderate pain with ambulation but intense, sharp pain at the base of the fifth metatarsal with attempts to run, jump, cut on the involved foot, roll up on the lateral side, or land on the lateral side of the foot.²²

The mechanism of injury is rarely from a direct blow or crush injury that may occur in actions such as a pileup. Some have described the position of 30 degrees to 50 degrees of foot

supination as the most likely position for maximal strain on the fifth metatarsal base. Whether any prodromal symptoms existed or not, the athletes report acute pain in the lateral foot at the metaphyseal-diaphyseal junction of the fifth metatarsal. Commonly, the athlete is unable or lacks confidence to continue full play.²¹

Fifth metatarsal stress fractures typically involve the proximal to mid diaphysis, characteristically occurring more distal than a traumatic base of fifth metatarsal tuberosity avulsion fracture.

Physical examination confirms mild to moderate swelling localized to the lateral midfoot and sharp tenderness at the base of the fifth metatarsal. It is important to examine the athlete, both sitting and standing, to assess the full lower leg alignment, especially the hindfoot and midfoot alignment. Cavus midfoot or varus hindfoot can be present but is not uniform. More recent studies have demonstrated an everted rearfoot and inverted forefoot alignment were associated with fifth metatarsal stress fractures.²³

Weight-bearing anteroposterior (AP), lateral, and oblique radiographic imaging of the involved foot is mandatory. Additional specialty imaging, such as a bone scan, MRI, or computed tomography (CT), is used in unique circumstances.²⁴

Radiographic and MRI findings of fifth metatarsal stress fractures include features of both diaphyseal (cortical-predominant) and metaphyseal (trabecular-predominant) stress fractures given its location at the junction of the metaphysis and diaphysis. Initial radiographic findings include periosteal reaction, with the degree of intramedullary sclerosis increasing in healing and chronic fractures. Important prognostic findings include the presence of a plantar gap at the fracture site, which is associated with poorer healing. MRI demonstrates periosteal and intramedullary edema, with a cortical fracture line evident in higher-grade injuries.²⁴

Significant medical workup for athletes with Jones fractures is not routinely needed. However, vitamin D deficiency, low calcium intake, and general nutritional evaluation are helpful in athletes at all levels. Vitamin D deficiency has been shown to occur in up to 50% of Division I athletes and is noted in some studies to be higher in African American populations.²⁵

In the female athlete, a history of menstrual cycle irregularity should be investigated, as low estrogen and other hormonal imbalances may lead to a higher risk of delayed unions or non-unions. Dual-energy x-ray absorptiometry (DEXA) scan evaluation is reserved for the athlete with multiple stress fractures or recurring nonunions, as well as the middle-aged and older athlete. Newer-technology DEXA scanning allows for a more detailed evaluation of trabecular bone abnormalities despite normal, generalized bone.²⁶

Fractures of the proximal fifth metatarsal that occur in the proximal diaphysis (greater than 1.5 cm from the tuberosity) have an increased risk of poor healing, which is believed to be due, at least partially, to the relative lack of blood supply of the proximal diaphysis. In contrast to the metaphysis, which is supplied by a rich network of metaphyseal arteries, the proximal diaphysis is supplied by a sole nutrient artery, which may be disrupted in proximal diaphyseal fractures.



Fig. 25.13 Professional volleyball player with loose fragments of kissing osteophytes.

In a recent review of the literature, it is described that operative treatment of fifth metatarsal stress fractures results in a smaller number of delayed unions or nonunions, compared to conservative treatment. Also, the goals of early surgical management are to minimize the risk of nonunion and recurring fracture, and to decrease the time to return to sport. Foot anatomy must be taken into consideration, and additional procedures such as the lateralizing calcaneal osteotomy for a cavovarus foot should be considered.

Internal fixation with a solid stainless-steel IM screw has become the procedure of choice.²⁴ The patient is kept nonweight bearing for 3 weeks; then partial weight bearing is allowed with a removable boot, and stationary bicycle and pool therapy is initiated. Transition to full weight bearing begins at 5 weeks. In general, return to activity is allowed 2 months after surgery, depending on the resolution of pain and radiographic evidence of healing.

SLALOMING THE ANDES MOUNTAINS: SKI AND SNOWBOARD INJURIES OF THE FOOT AND ANKLE IN CHILE

Introduction

The Andes Mountains is the world's longest mountain range extending from north to south for 4498 miles across seven countries: Venezuela, Colombia, Ecuador, Peru, Bolivia, Argentina, and Chile. The chain has an average height of 12,000 feet and a width of 149 miles, giving shape to the landscape and scenery of most of western South America.

Providing a stunning backdrop to almost the entire Chilean territory, the Andes mountains contribute with approximately 3000 miles of natural border running down from the arid Atacama Desert to the Glaciers of the Patagonia. The above, added to the narrowness of Chile's geography, makes the mountains accessible from almost every point of the country and usually can be planned as a day trip from almost every major city. This unique feature facilitated since the 1930s the development of numerous ski areas and world-class resorts throughout the central and south regions, being the firsts of their kind in South America and making Chile nowadays an internationally renowned winter sports destination (Fig. 25.14).

Skiing and snowboarding are the most popular activities during the regular winter season that runs usually from May until October. These sports have been growing in popularity among the Chilean population during the last 20 years, as they are more accessible and are no longer perceived as elite activities. Professional skiing has also seen great advances in the current decade with consistent World Cup and Olympic participation (Fig. 25.15).

Injuries affecting skiers and snowboarders have been widely described.²⁷⁻³² The most common injuries in skiers involve the knee, specifically the anterior cruciate ligament and medial collateral ligament, with a prevalence reported between 27% and 33% of all injuries.^{28,30} On the other hand, snowboarders usually sustain upper extremity injuries especially involving the wrist and shoulder, corresponding to almost 20% of all injuries.^{30,33}



Fig. 25.14 Slalom at La Parva, Chile.



Fig. 25.15 Chilean Olympic skier Henrik Von Appen during downhill competition.

Foot and ankle injuries are far less frequent in these disciplines. Nevertheless, there are several sport-specific injuries in this category presenting unique characteristics that physicians in charge of these winter sports athletes must consider for prevention and adequate treatment.

Equipment and Technical Considerations

The basic modern skiing equipment considers the use of a boot-binding-ski system. This spring-actuated mechanism attaches the boot to the ski in a fixed position until the bindings are released in response to torsional and upward forces (Fig. 25.16). Release is determined by the spring tensions in two adjustable toe and heel pieces.³⁴ A significant decrease in foot and ankle injuries during downhill skiing has been evidenced after the 1970s due mainly to the breakthrough of better-designed and -engineered ski boots and binding systems, transmitting the torque forces more proximally in the lower limb.^{32,35} These

advances also included improved materials for harder boot shells and weight/height-specific bindings that optimize the detaching process following falls and twisting forces.

However, the current popularity of snowboarding has meant an increase in foot and ankle injuries due to the use of softer boots that allow better control and maneuverability (Fig. 25.17). In line with this trend, there are several studies showing that 12% to 38% of snowboarding lesions are ankle-related, in comparison to only 1.7% to 6% of all skiing injuries.^{29,30,36} Several different types of boot-binding combinations exist nowadays, but the most commonly used



Fig. 25.16 Boot-binding-ski system.



Fig. 25.17 Snowboarding soft boot.

is a soft “pack-type” boot, with a nonreleasable strap binding. Although less popular, hybrid and hard ski boots are also used with either releasable or nonreleasable bindings. Kirkpatrick and collaborators compared snowboarding injuries of the foot and ankle in relation to the type of boot used (soft, hybrid, or hard) and found no significant correlation between boot type and overall injury rate. Nevertheless, in their study, hard boots appeared to be protective for ankle fractures not involving the lateral process of the talus (LPT).¹⁰

Ankle Sprains and Fractures

Ankle sprains and fractures are the most prevalent injuries of the foot and ankle segment reported both in skiers and snowboarders.^{28,30,33} Differential diagnosis is crucial in patients sustaining an ankle sprain in this context, especially because there are similar clinical findings on fractures of the LPT or peroneal tendon injuries that can be easily misdiagnosed.

The most frequent mechanism of fractures of the lower leg and ankle usually involves a fall in skiers, while a greater proportion of snowboarders has jump-related injuries.³⁷ Concerning the level of injury, skiers present most commonly with oblique or spiral distal tibial fractures at the edge of the skiing boot, or “Boot-Top-Fractures.” On the other hand, malleolar fractures are more common among snowboarders, with isolated fibula fractures almost exclusively seen in this discipline. Ishimaru and colleagues reported that most ankle fractures in snowboarders were supination-external rotation type II injuries as described by the Lauge and Hansen classification.³⁸

Pilon fractures are common in the context of vertical impact and axial load, especially in skiers landing from a high-altitude jump. Energy tends to dissipate when the athlete lands on a slope and then continues in a downhill direction. In cases where the landing is effected on a flat surface, the energy is not capable of dissipating, and this can result in a tibial plafond injury.³³

Management of these injuries is no different from lesions occurring outside of the ski/snowboard context. Emphasis should be on rehabilitation and complete healing of the fracture site with a period of at least 12 weeks following open reduction and internal fixation before returning to practice.

Snowboarder’s Fracture

Fractures of the LPT are a specific type of injury that was rarely seen before the snowboard era but has become more prevalent mimicking snowboarding popularity, which has contributed to its current name, “Snowboarder’s fracture.”³⁹ Kirkpatrick in his series reported a talar fracture incidence of 17% within all snowboarding injuries around the foot and ankle. LPT fractures were particularly high, accounting for almost 95% of these injuries.³⁶

The LPT is a wedge-shaped prominence that comprises the most lateral aspect of the talar body, being part of the talofibular and subtalar joints and serving for various ligamentous attachments.

Even though the mechanism of injury is controversial, the most common accepted explanation for LPT fractures is an axial impaction in dorsiflexion with the hindfoot in inversion and in an externally rotated position. This axial impaction is typically seen after landing from a height in snowboard jumping.^{39,40}

In 1965, Hawkins described three types of LPT fractures^{41,42}:
Type I, Simple (42%): Extends from the talofibular articular surface and the posterior subtalar facet.

Type II, Comminuted (34%): Involves both articular surfaces and the entire lateral process.

Type III, Chip (24%): Normally compromising only the subtalar joint, variable size.

Clinical presentation resembles an ankle sprain and may result in pain and disability if not recognized and treated, especially because up to 41% of LPT are missed on initial presentation.⁴³ Typically, there will be local ecchymosis, edema, and tenderness to perifibular palpation, with variable weight-bearing ability. ROM can be limited by pain, though deformity is usually not seen in these kind of injuries.

Imaging study includes x-rays to rule out other more prevalent injuries such as ankle fractures. LPT fractures can be identified more clearly on the Mortise view. However, there is a general agreement that the best diagnostic accuracy is obtained with CT-Scan, allowing classification of the type of fracture and assessment of articular involvement, thus guiding the treatment plan.³⁶

Treatment of LPT fractures is guided by the topographical Hawkins classification and the amount of displacement involved. Valderrabano proposed a treatment algorithm based on their series of 20 cases.⁴⁴ Large displaced fragments (type I) should be surgically treated in order to restore articular congruity and diminish long-term morbidity (Fig. 25.18). For nondisplaced fragments, a nonsurgical approach can be made but with a higher risk of future morbidity. For type II and III, treatment depends on the amount of displacement; nonoperative management if no displacement is seen, and surgical debridement if there is any displacement. In general, this approach appears to be reasonable and is accepted by the vast majority of authors.

Outcomes for type I fractures treated operatively, regardless of the method of fixation, are fairly good. Perera and colleagues reported that 88% of their patients presented mild or no symptoms during follow-up. On the other hand, up to 38% of the patients treated conservatively persist with moderate or severe symptoms.⁴² Poor prognosis has to be expected if diagnosis is missed initially, which is a common reason for nonsurgical treatment. For patients with continued pain and morbidity, depending on the type of fracture, excision or articular fusion

may be indicated. Common complications include chronic pain, nonunion, ankle instability, and subtalar arthritis.⁴⁰

Morton's Neuroma

Erroneously classified as a neuroma, this entity is one of the most common causes of metatarsalgia. Morton's neuroma is an entrapment neuropathy causing an interdigital neuralgia secondary to perineural fibrosis.⁴⁵ This condition is frequent in skiers, in relation to narrow hard boots producing increased pressure on the forefoot. This leads to traction of the interdigital nerves, which subsequently produces perineural fibrosis. Other factors influencing the condition is repetitive trauma and stresses over the MTP joints producing swelling of underlying metatarsal bursa and causing greater compression of the interdigital nerve against the intermetatarsal ligament.⁴⁶

Clinical features include pain and tingling at the involved web space. Numbness may be present. Patients often describe a shooting electrical sensation during or after skiing that usually alleviates by taking the boot off. Ultrasound and MRI examination have been described to confirm the diagnosis.⁴⁷ However, clinical findings still have the highest accuracy for Morton's neuroma diagnosis.⁴⁸

Conservative treatment options include ski boot modifications, inserts to reduce pressure beneath the metatarsal heads, or lidocaine/steroid injections. Surgical excision is indicated with persistent symptoms and has been shown to have between 70% to 85% success in literature.⁴⁵

Skier's Toe

Skier's toe or subungual hematoma is produced by excessive compression of the toes against a narrow boot or by a repetitive trauma of the affected toe secondary to a loose boot. The most affected toe is the hallux, and patients usually complain of progressive pain due the increased pressure produced by the hematoma on the nail bed.

Another factor related to skier's toe is improper skiing technique. Sitting back on the skis forces the toes against the roof of the boot, while maintaining forward pressure on the shins diminishes this pressure on the toes. Long toenails also act as a lever, increasing pressure on the respective toes. We recommend keeping toenails short when skiing or snowboarding in order to avoid this complication.

Treatment of the subungual hematoma includes conservative measures such as applying ice and nonsteroid antiinflammatory drugs (NSAIDs). In cases of persistent pain or complete involvement of the nail bed, aseptic fenestration of the nail to drain the hematoma is indicated.

Chilblains

Chilblains, or pernio, is an injury that develops with prolonged exposure (1 to 5 hours) to frigid or near-freezing temperature. This condition appears as erythematous, well-defined, and tender papules typically on the hands or feet of susceptible skiers and snowboarders.

Treatment involves removing wet clothes or constrictive boots, gentle washing and drying of the area, and covering the skin with dry and loose clothing. Nifedipine may decrease pain and accelerate resolutions of symptoms.⁴⁹



Fig. 25.18 Fracture of the lateral process of the talus (black arrow). Hawkins type I.

CHINESE TRADITIONAL CONCEPTS OF FOOT AND ANKLE PROBLEMS AND TRADITIONAL TREATMENTS

There are many traditional sports and performances that are still popular in China at present, such as shuttlecock kicking (Figs. 25.19 and 25.20), flipping in various Chinese local operas (Figs. 25.21 and 25.22), and the Chinese martial art “wushu,” which is also called Kung Fu (Figs. 25.23 and 25.24). Usually, the Chinese do traditional sports for health purposes and



Fig. 25.19 Shuttlecock kicking is a popular sport in China.



Fig. 25.20 Shuttlecock kicking performance is technically demanding.



Fig. 25.21 Flipping is often performed in many kinds of traditional Chinese local operas.



Fig. 25.22 Flipping is a maneuver in Chinese martial art “Wushu.”



Fig. 25.23 Chinese martial art “Wushu.”



Fig. 25.24 Some kinds of “Wushu” are less competitive.

personal fulfillment. Thus there are infrequent opportunities for the traditional Chinese sports to be competitive. However, it is not unusual to see foot and ankle injuries caused by these traditional sports in clinic. Soft-tissue injuries are much more common than a real fracture.

The most common reason for the shuttlecock players suffering a foot or ankle injury is players performing a trick. When there is uneven ground, there is more likely to be an accident. Ankle sprain occurs and may lead to chronic ankle instability several years later. Furthermore, injury may occur as a result of increasing body weight or decreasing strength of the ligament. Flipping is often performed in many kinds of traditional local Chinese operas. This maneuver is more skillful and always performed by professional actors. Since the calf contracts suddenly and strongly when doing this, Achilles tendon injury (partial or complete rupture) may occur. Chronic injury is seen in Chinese wushu players when they continue to practice wushu exercises for decades after a primary injury.

There are many different kinds of foot and ankle injuries caused by these traditional Chinese sports and performances, including ankle sprain, fifth metatarsal avulsion fracture, Achilles tendon rupture, diastasis of syndesmosis, MTP joint capsular injury, instability of the ankle, and medial and lateral malleolus fracture.

Many Chinese believe in traditional treatment and traditional medicine when they have foot and ankle problems. There are some special, traditional treatments for soft-tissue injuries of the ankle joint in China that have a long history. These treatments include acupuncture, Chinese herb ointment, poultices, foot massage, and so on (Figs. 25.25 through 25.28).

Chinese herb ointment and sometimes a semirigid splint made of bark are the usual management modalities for soft-tissue injury in the early stage. Chinese herb ointment can decrease the swelling effectively and quickly. Poultices,

acupuncture, and massage, accompanied by functional exercises, are the treatments for subacute injury.

Acupuncture and massage are important components of restoring balance to the person's vital energy channels, which form the basis behind traditional Chinese medicine. The channels are a system of conduits throughout the body that carry and distribute "Qi," which can be considered as vital energy. Disease is present when the flow of vital energy through the channels is disrupted. This may occur when the integrity of the channels themselves is damaged by a sprain. The Chinese describe this as a disease of "Bi," or pain, caused by a localized disruption to the flow of "Qi."

The traditional Chinese explanation for soft-tissue injury is that the channel running through the damaged tissue has been physically disrupted, resulting in local pain, a disease of "Bi." To treat the pain, the integrity of the channel and the flow of vital energy through the channel must be restored. This can be achieved by the selective use of points on the damaged channel, thereby restoring the flow of "Qi" and relieving the pain.

The foot plantar surface is an important place for the body because there are many points of channels, which represent many internal organs. Therefore foot massage not only treats the injury of foot but also can treat diseases anywhere in the body. In China, foot massage is looked on as a good method for preventing and treating diseases and is popular throughout the whole country.

Foot massage is used to stimulate the points of the channels that can activate the gates of the body, which are opened and closed to adjust circulation in the channels. Foot massage has four functions: it can enhance the blood circulation, so as to accelerate the metabolism of the body¹; it can regulate the nervous system; because there are many nerves endings in the foot, one can stimulate the reflex zone of the foot to regulate the corresponding tissues and organs²; it can mobilize "Qi," moisture, and blood and promote proper function of the muscles, nerves, vessels, glands, and organs³; and it brings the efficacy of release and relaxation.⁴

Chinese traditional treatments for acute foot and ankle injury are similar to modern treatments in some aspects, such as rest, relieving the pain, and diminishing the swelling.

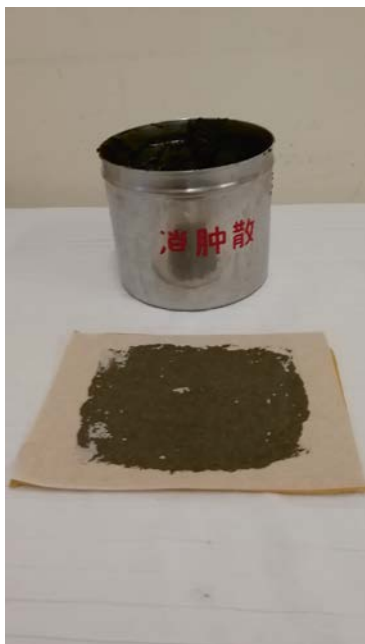


Fig. 25.25 Chinese herb ointment.



Fig. 25.26 Acupuncture treatment at the foot.

CHART OF THE FOOT REFLECTIVE ZONES

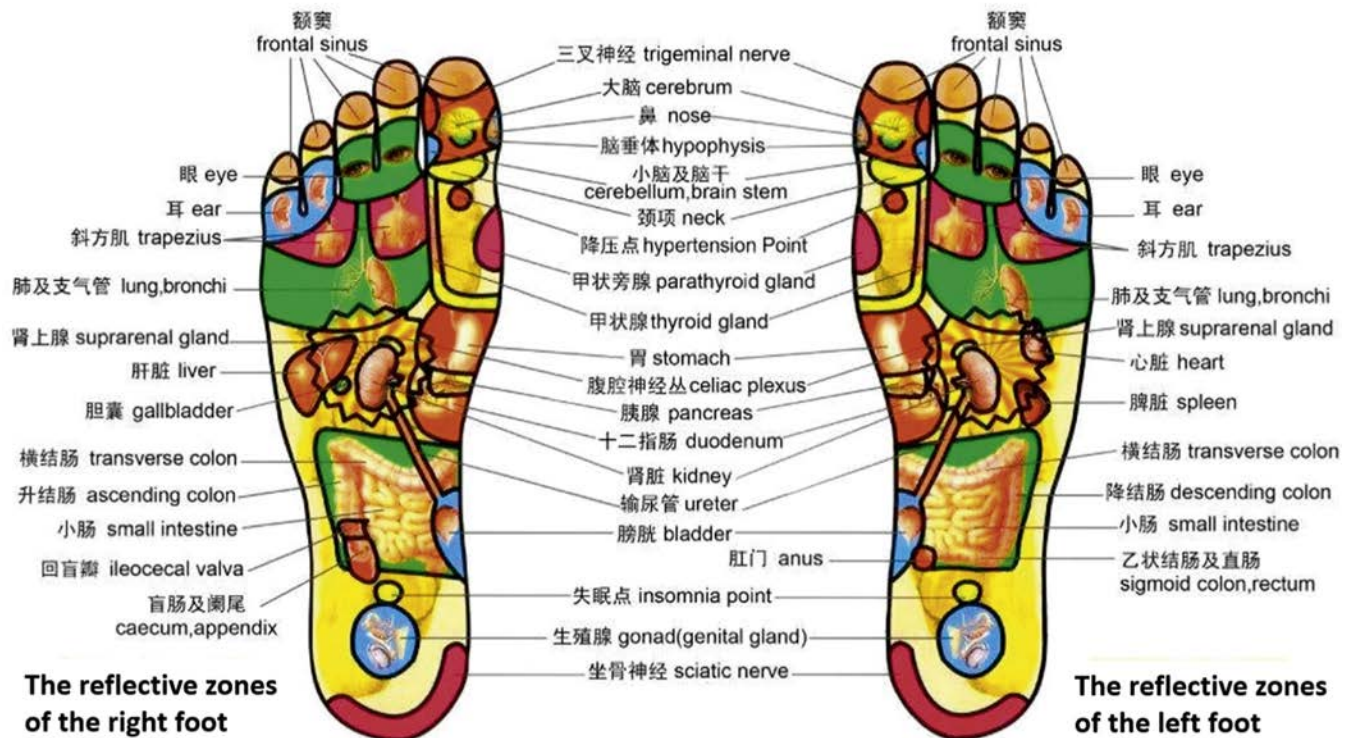


Fig. 25.27 The points on the foot reflect the organs of the body.



Fig. 25.28 Foot massage.

The treatments of acupuncture and foot massage are helpful to the patients during rehabilitation. Nowadays, the modern treatment and rehabilitation methods have been widely accepted and used throughout the country. The traditional method and modern method supplement each other.

COLOMBIAN FOOT AND ANKLE CONDITIONS IN SPORTS

Colombia has a varied geography and a variety of sports and recreational activities. The most popular sport is soccer, which

is a widespread activity throughout the country, and professional dancing has a special place in some regions of the country. Athletic activities including 5, 10, 21, and 42 km racing competitions, and cycling in two modalities (mountain biking and road biking) is also very popular sports sometimes associated with foot and ankle conditions. Since Colombia has no seasons, these sports and recreational activities can be practiced year-round, leading to frequent overuse syndromes.

Related to soccer, the most frequent injuries are ankle sprains and anterior ankle impingement. Soccer is played on turf, which can be soft in the rainy season and hard in the dry season. The uneven grounds are also related to the frequency of ankle sprains. Indoor soccer played on synthetic turf has also a very high frequency of ankle sprains, due to shear forces on ankle and knee joints. Radiological evaluation of regular soccer players shows frequently medial, lateral, and anterior osteophytes associated with the repetitive trauma to the ankle. In recent literature, the origin of the tibial and talar osteophytes is related to ankle instability or repetitive trauma at or near the joint line. Thus, these are called osteophytes and not enthesophytes. The traction theory is no longer valid as cited by Johannes et al.^{50,51}

Ankle Sprains and Ankle Impingement

Patients with a history of frequent soccer games in the past present with multiple sprains and painful episodes after games. They may have had an initial severe sprain that received medical attention with adequate management. However, after



Fig. 25.29 A, Preop x-rays. B, 7 months postop. C, 2 years postop.

ongoing sprains, patients do not search for medical attention until symptoms evolve either with instability or limitation in motion and impingement symptoms. In cases of ankle anterior impingement on physical exam, patients may complain of ankle reduced dorsiflexion compared to the contralateral side. Palpation on the anterior ankle joint can elicit pain, and depending on the size of the osteophyte, these can often be palpated. Stability testing of the ankle is carried out, and sometimes instability findings can be associated. Patients must also be tested for peroneal tendon pathology and subtalar symptoms, which are to be ruled out. Eversion strength and gastrocnemius muscle contracture are also evaluated.

Antero-posterior, lateral, and mortise standing comparative views are taken in routine fashion (Fig. 25.29), and if a small osteophyte is seen, an additional van Dijk's view is taken⁵² (Fig. 25.30). Only if any other instability-generating factor is suspected will a CT or MRI be ordered.

The ankle sprains are managed initially with the rest, ice, compression, elevation (RICE) protocol and after a short

immobilization period sent to physiotherapy. We put great value in a thorough rehabilitation program looking for a total restoration of full eversion strength, Achilles tendon flexibility, and proprioception. Most of these patients get back to regular sports after 6 to 8 weeks, and only those who continue having giving-way symptoms and recurrent pain are treated surgically with the open Bröstrom-Gould procedure.

Other patients who have an instability sensation in more chronic cases can have an anterior impingement syndrome, which affects their proprioceptive condition. In these cases, the surgical procedure is focused on the resection of the anterior osteophytes of the tibia and the dorsum of the talar neck. This procedure is preferably performed either with a 40-mm arthroscope or with a mini-open technique. The joint is inspected first with a thorough complete ankle joint evaluation, with special attention to the medial and lateral gutters and at the horizontal joint line. Temporary distraction of the ankle is performed for inspection. The main procedure is performed without traction, and we proceed to resect the osteophytes



Fig. 25.30 Van Dijk's view.

on both sides (tibia and talus). The extent of the resection is checked under fluoroscopy. Careful resection of the inflamed synovial tissue of the anterior joint space is also performed. These patients are operated as outpatients under peripheral nerve block and sedation. Postoperatively they are immobilized with a soft dressing and 48 hours bed rest and elevation. Weight bearing is allowed after 2 days to tolerance, and sutures are removed after 12 to 15 days. A rehabilitation protocol centered on dorsiflexion recovery, strength, and proprioception is begun at that time.

On follow-up, we have experienced what the literature describes as recurrence of the osteophytes in a large percentage of our cases,^{53,54} but less than a third of those patients have recurring symptoms. If instability persists, the recurrence is greater (see Fig. 25.30).

Our main problems with surgical treatment of ankle instability, are neuropathic pain with superficial peroneal nerve lesion, arthrofibrosis, and medial or residual lateral instability.^{55,56} The multimodal pain management or in combination with nerve blocks with the aid of ultrasound and open neurolysis are the common approaches to this clinical entity. Arthrofibrosis is not frequent after open or arthroscopic ligament reconstruction. If there is a mild to moderate case, physical therapy has been beneficial. Uncommonly there are severe cases of arthrofibrosis that may require open debridement.

The residual instability of ankle is described more often in the recent literature, and the medial component of this pathology has had more focus in the last years. We are focusing on this clinical problem and see it with a more prevalent frequency in our clinical practice. When rupture of the deltoid ligament in an acute setting is present, a repair of the ligament is performed. In acute or chronic spring ligament ruptures, and an MRI with positive findings, our choice is a repair with suture anchors in acute settings or debridement of scar tissue and direct repair in the chronic lesions.⁵⁷



Fig. 25.31 Rupture gap in Achilles tendon.

Achilles Tendon Lesions

Achilles tendon ruptures are increasing and causing morbidity in our young population. These young adults are more involved in indoor and outdoor soccer without a physical training program, which renders more lesions than in the professional players.⁵⁸ High frequency of games throughout the week (3–4 times) generate overuse syndromes that predispose to tendinopathy and tendon ruptures.⁵⁹ These lesions can be caused by either indirect or direct trauma to the tendon. Patients can present with some prodromal symptoms before the acute rupture. Occasionally our patients have had previous visits with paratenonitis or paratendonitis with tendinosis that were not treated adequately.⁶⁰

These patients come in to the emergency room with an acute onset of pain and a sudden snapping or popping sensation at the Achilles tendon during sports activity. On occasions, a previous history of Achilles tendon pain can be obtained from the patient.

On physical exam the patient walks with a limp and variable edema around the rupture site. A positive Thompson test and a palpable gap are typical of a complete rupture (Fig. 25.31). The plantar flexion strength can be diminished. In cases of doubt we order an ultrasound or an MRI, but these are not routinely performed.

If the rupture is a palpable gap within 7 cm of the postero-superior calcaneal tuberosity with a positive Thompson test, we proceed to perform a minimal incision repair within the first 10 days. If the rupture does not have a clear gap, but a positive Thompson test, an ultrasound or an MRI is ordered to define the type of rupture to plan the surgical approach. The mini-invasive technique has a lower complication rate with respect to the soft-tissue complications in simple ruptures (horizontal), but in massive and irregular tears, an open reparative technique is preferred.

Surgical Technique

The patient is positioned prone with both legs prepped for comparison of foot position at final tendon repair (Fig. 25.32).



Fig. 25.32 Positive Thompson test.



Fig. 25.33 Tension after surgical reconstruction of Achilles.

A 4-cm skin incision is performed horizontally 1 cm above the gap, and careful incision of the paratenon is also performed. The stumps are localized and pulled through the incision, and a commercial tendon repair kit is used to perform the repair on both sides of the ruptured tendon. Three strands of reinforced nonabsorbable suture are knotted with the foot in a similar or slightly more plantar-flexed ankle position as the contralateral limb (Fig. 25.33). Suture of the paratenon and skin incision are performed. A bulky dressing with the foot in plantar flexion and a posterior splint is applied for 12 days nonweight bearing. On day 12, the sutures are removed and a walker boot with a 40-mm heel lift is applied with permission for progressive weight bearing. Physiotherapy with the Mendelbaum protocol is started.⁶¹

For all chronic lesions, either partial or complete, with more than three months evolution, we use the FHL transfer deep to

the Achilles insertion, fixed with anchor sutures or biotenodesis screws.⁶² We have used this transfer tendon technique in more than 80 patients with solid results at this time.

Lisfranc Low-Grades Sprains

In other activities performed in Colombia, like dancing, cycling, or horseback riding, trauma to the foot and ankle can involve torsional forces that generate these sprains and have a relative high incidence and are not properly diagnosed in local emergency rooms. Sometimes these lesions are detected in patients in a late form with residual pain and problems at their midfoot. Persistent pain after 4 to 6 weeks of injury, with edema present at the TMT point of first and second rays, with initial ecchymosis described on the first visit at the emergency room, can be indicative of severe ligament or soft-tissue damage.

Clinical findings, AP, lateral, and oblique standing bilateral views can sometimes be sufficient to view subtle differences of both feet to make the diagnosis. The fleck sign, which indicates Lisfranc's avulsion fracture, must also be considered. CT scan or MRI can be indicated in cases of doubt, but they are usually not ordered in a routine fashion. The open reduction and internal solid fixation are mandatory in all displaced fracture-dislocations.⁶³⁻⁶⁷

For low-grades lesions and symptomatic sprains, we use the Nunley and Vertullo⁶⁸ classification, with good and excellent results in the 89% of our patients with surgical treatment in grade II and III lesions with transarticular screws or the endobutton technique.

AN EGYPTIAN PERSPECTIVE ON THE FOOT AND ANKLE IN SPORTS

In Egypt, soccer is the most common sport practiced both on professional and recreational levels. People of all age groups play soccer, and it is currently played on different terrains including turf mostly, but also sand and concrete. An array of foot and ankle injuries has been associated with soccer, including but not limited to ankle sprains, Achilles tendon ruptures, turf toes, FHL tendinitis, Lisfranc injuries, ankle impingement, and OCLs of the talus. Other popular sports include squash, wrestling, and martial arts. Recently, cycling and marathon running have gained popularity and attracted a good proportion of the youth population.

One thing to mention is the delayed presentation of many of our patients. This is partly due to limited access to health care at some areas. Moreover, patients tend to ignore their complaints and try natural remedies and alternative medicine long before they seek medical advice. This reflects on the chronicity of the case and therefore decision-making and available options to treat such problems.

We here report on our experience with management of some of the aforementioned injuries and our preferred surgical techniques.

Osteochondral Lesions of the Talus

It is estimated to occur in 2%–6% of ankle sprains; however, more than 80% of lesions are missed or diagnosed in a delayed

fashion.⁶⁹ Many factors have to be considered when discussing surgical management of these injuries. These can be classified into patient factors (age, body mass index [BMI], activity level, and comorbidities) and lesion factors (size, containment, condition of surrounding cartilage and subchondral bone). Treatment strategies can generally be grouped into two major categories:

- 1) Reconstruction techniques: fixation or reattachment of OCLs and autograft or allograft osteochondral transplantation
- 2) Repair techniques or marrow-stimulation techniques: microfracture and drilling (with or without biologic augmentation), autologous or allogeneic cell implantation

While we have performed many of these options, our results have been inconsistent and not fully satisfactory. This is in line with data showing no superiority of one cartilage repair technique over others.^{70,71} We recommend performing microfracture for lesions <10 mm². Lesions larger than 10 mm² are treated with either microfracture supplemented with biological augmentation (bone marrow aspirate concentrate [BMAC] or platelet-rich plasma [PRP]) or osteochondral autologous transplantation (OATs) with allografts reserved for critically sized lesions that are not amenable to autografts.

Recently, the senior author (AH) reported on the use of culture-expanded bone marrow-derived mesenchymal stem cells (MSCs) in treatment of large OCLs of the femoral condyle. Their preliminary results were promising, and they noted improvement in patient outcomes and MRIs.⁷² We subsequently decided to use the same technique with OCLs of the talus. We started using second-generation bone marrow MSC implantation, which entailed culture-expanded MSCs, implanted on platelet-rich fibrin glue (PR-FG) scaffold. This mixture was injected via all-arthroscopic technique into the base of critical-sized OCLs, and patients were made non-weight bearing for 6 weeks postoperatively (Fig. 25.34). We reported on a small case series of 16 patients treated with this technique with an average size of OCL of $3.74 \text{ cm}^2 \pm 1.12$ and mean follow-up of 18 months. Briefly, autologous bone marrow aspirate was acquired from patients in a first-stage procedure. This was transferred to the Tissue Engineering Unit at the Department of Biochemistry, Cairo University College of Medicine, where MSCs were isolated and culture expanded for a minimum of five passages. The plasma was processed to create PR-FG. In a second-stage all-arthroscopic procedure, the base of the defect was curetted to a stable healthy rim of cartilage and underlying bleeding subchondral bone. Defects deeper than 8 mm were grafted with autologous calcaneal bone graft.⁷³ The culture-expanded MSCs were then mixed with the autologous PR-FG and injected in the base of the defect after establishing dry arthroscopy. The mixture was let to clot and ankle ROM was tested under arthroscopy for stability of the implant. Immobilization for 2 weeks was followed by active and passive ROM exercise while maintaining nonweight-bearing status for 6 weeks, followed by progressive weight bearing. Our study showed improvement in postoperative AOFAS scores compared to preoperatively, but this improvement did not reach statistical significance.⁷⁴

Flexor Hallucis Longus Tendinitis/Posterior Ankle Impingement

FHL tendinitis is a chronic overuse disease common among ballet dancers but can also affect soccer players due to repetitive plantarflexion maneuvers. Symptoms and physical exam can mimic posterior ankle impingement.⁷⁵ Both conditions can coexist and are sometimes considered as spectrums of the same pathology. Conservative management can be successful in up to 64% of cases with immobilization.^{76,77} Surgical treatment consists of surgical release/tenosynovectomy, which can be done both open and arthroscopically. We now prefer doing arthroscopic release to avoid the invasive open approach, which carries higher risks of postoperative adhesions and increased morbidity. As previously described by van Dijk et al,⁷⁸ posterior arthroscopy is performed with the patient in prone position with utilization of a thigh tourniquet and 30-degree 4.0-mm arthroscope. Two main portals are established; a posterolateral and posteromedial portal. The posterolateral portal is established first 1 cm proximal to the tip of the lateral malleolus just lateral (5 mm) to the Achilles tendon. Subsequently the posteromedial portal is performed just medial to the Achilles tendon at the same level as the posterolateral portal. A motorized shaver is used to perform synovectomy and resection of soft tissues between the talus and Achilles tendon, starting laterally until the FHL is visualized. The FHL serves as a “lighthouse” to this approach, and visualizing it is the key to the procedure. The FHL is located typically about 1–2 cm proximal to the subtalar joint. Identifying the tendon can be aided by passive motion of the big toe. Extreme caution is warranted to avoid injury to the neurovascular bundle located just medial to the FHL tendon. In the presence of a symptomatic os trigonum, it can be excised via a combination of shaver and punches. At this point, the tendon is inspected for any pathology. In patients with FHL tenosynovitis (Fig. 25.35A), the tendon can be entrapped underneath its sheath, which is then incised and subsequently excised with a shaver (Fig. 25.35B). The entirety of the tendon is then examined to ensure absence of tears and smooth gliding of the tendon (Fig. 25.35C), including the most distal part within its fibro-osseous tunnel (arrow in Fig. 25.35C). We believe adequate portal placement is a must to be able to perform adequate FHL decompression. Inadvertent distal placement of the portals makes this procedure extremely difficult, especially in the face of an os trigonum that can block access if not approached from a “higher” portal. This higher portal also allows relative ease of access into the FHL fibro-osseous tunnel.

Other Injuries

Management of these injuries usually follows the general techniques practiced worldwide. Ankle sprains are mostly treated conservatively. Patients with ankle instability are treated with Brostrom-Gould repair when sufficient local tissue is available. Insufficient local tissue has prompted us to use anatomic reconstruction with gracilis autograft fixed with biotenodesis screws at the fibula and talus. More recently, we have been using the fibertape construct (Internal Brace, Arthrex, Naples, FL) for augmentation of repair in cases of insufficient tissues. Our

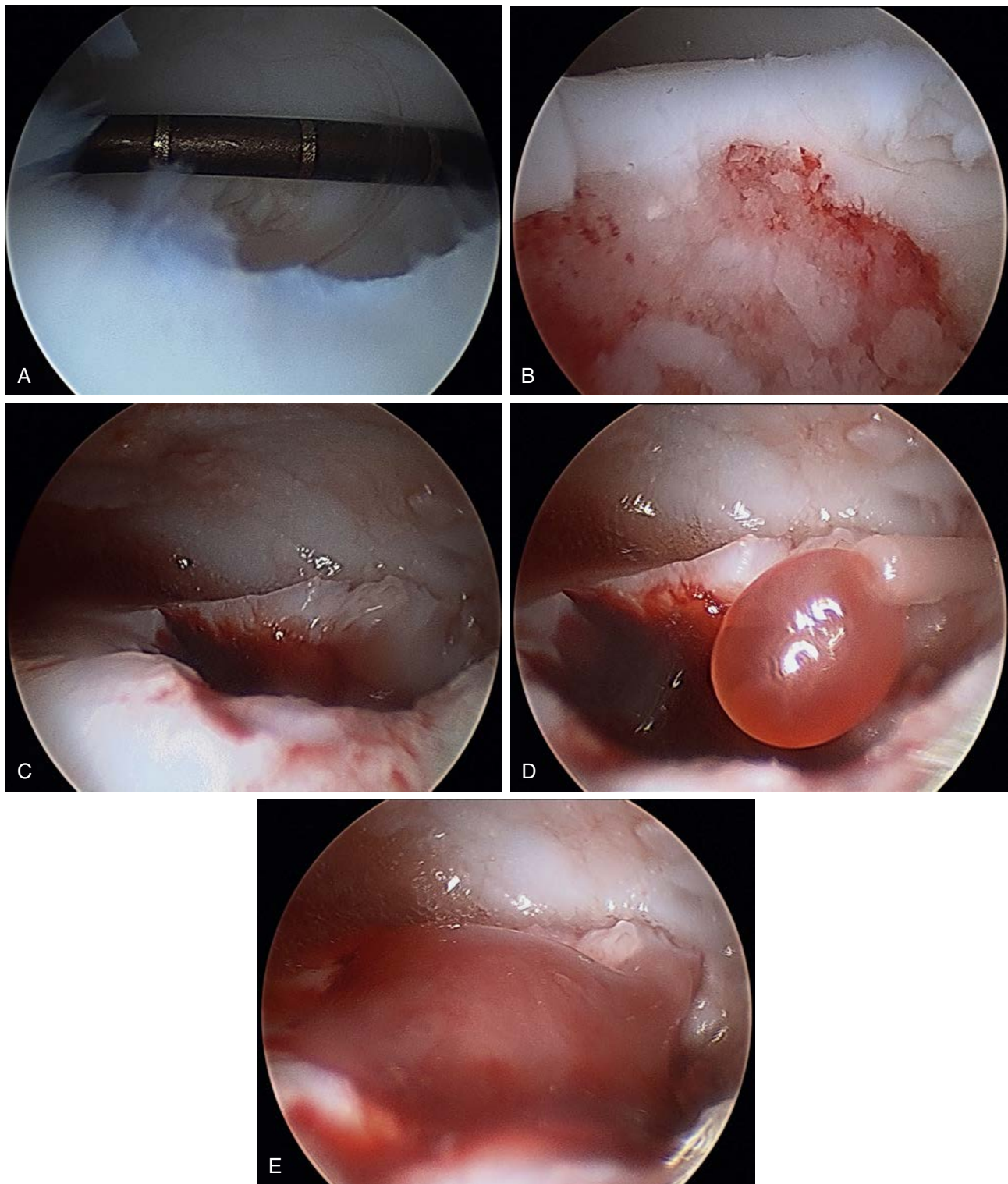


Fig. 25.34 Talar osteochondral defect (OCD) treated with mesenchymal stem cells (MSCs)/platelet-rich fibrin glue (PR-FG) mixture. **A**, Medial talar dome OCD measuring 7 mm x 8 mm. **B**, OCD after debridement to a stable rim and healthy bleeding subchondral bone. **C**, Establishing dry arthroscopy before implantation of the MSCs/Fibrin glue mixture. **D**, Injection of the MSCs/PR-FG from the dual barrel syringe arthroscopically into the base of the defect. **E**, The mixture is set level with the surrounding cartilage and left to dry for 7 minutes after which the construct's stability is verified by ranging the ankle under arthroscopic visualization.

short-term experience has yielded comparable results to the standard Brostrom-Gould reconstruction without augmentation while allowing an accelerated postoperative recovery due to the restraining effect of the fiber tape.

Acute Achilles ruptures are treated via open repair using Krackow technique. We started utilizing percutaneous techniques more often recently but have not noticed a difference in outcomes with the latter. The price of the percutaneous kit is a limiting factor in its wide use in Egypt due to limited resources

provided for the health care sector. Moreover, there has been a shift in paradigm lately in light of recent literature^{79,80} toward nonsurgical treatment with accelerated functional rehabilitation protocols.⁸¹ This is gaining more popularity given the reduced expense and equivalent functional results of such treatment algorithm. Surgical repair, however, remains the preferred method in professional and high-demand athletes due to studies showing slight increased difference in push-off during running and jumping.⁸²

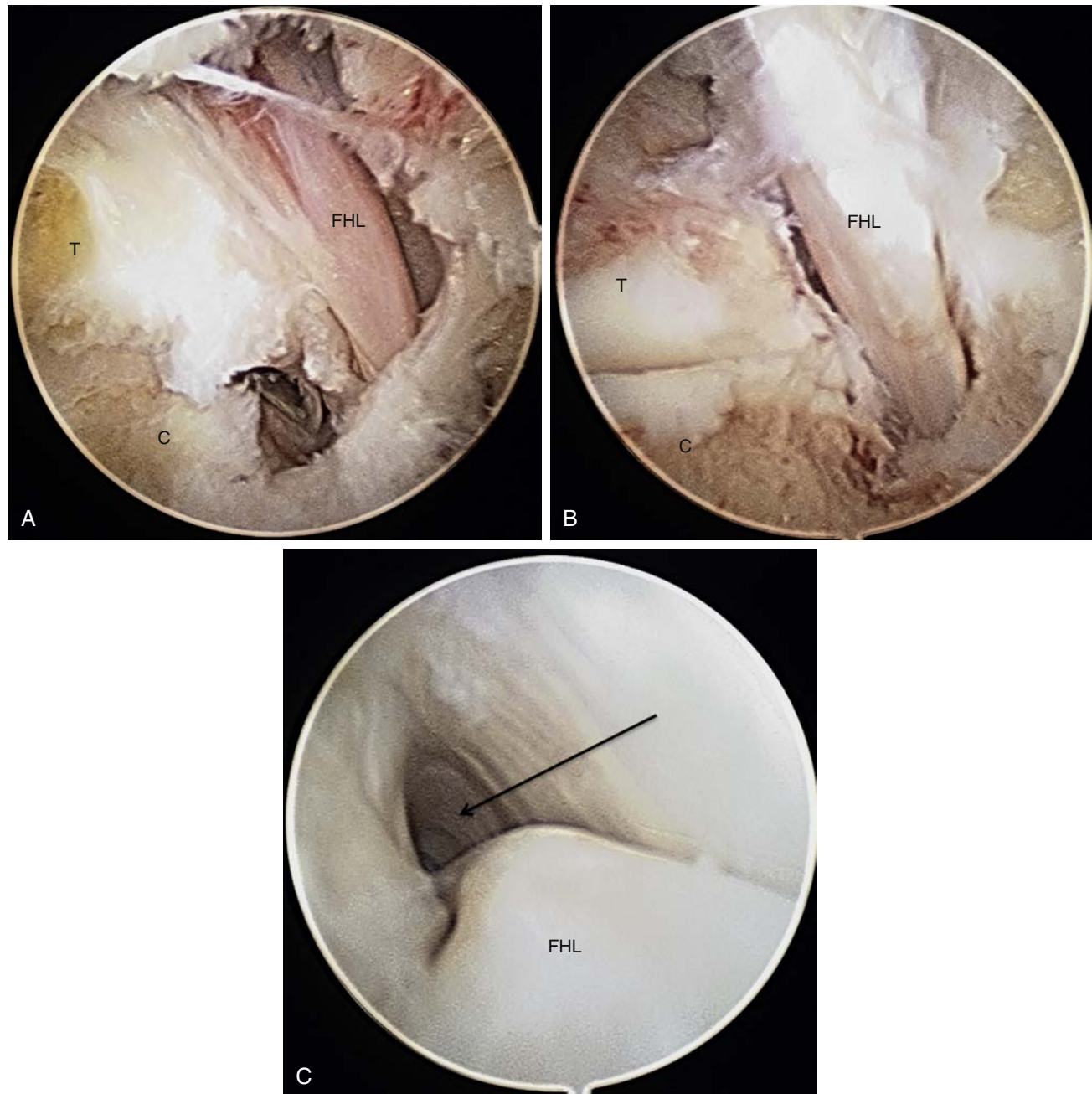


Fig. 25.35 Arthroscopic images of a 23-year-old soccer player who presented with severe posterior ankle pain and exacerbation of symptoms with hallux dorsiflexion. MRI confirmed extensive FHL tenosynovitis (*C* = calcaneus, *T* = talus, *FHL* = flexor hallucis longus). **A**, Arthroscopic image demonstrating significant tenosynovitis of FHL tendon with the tendon enclosed in hypertrophied inflamed synovium. **B**, Image after FHL tenosynovectomy and debridement revealing properly oriented tendon fibers free of tears. **C**, FHL tendon was traced all the way down in its tunnel to ensure complete excision of tenosynovitis and exclusion of tears in the tendon proper (arrow = fibro-osseous tunnel).

AN INTERNATIONAL PERSPECTIVE ON THE FOOT AND ANKLE IN SPORTS: INDIA

India is a big country with a huge, diverse population. Four very commonly played sports in India are cricket, field hockey, kabaddi, and khokho. Though hockey is a national sport, cricket is ‘The religion’ in India and outnumbers every other sport played in India.⁸³ In today’s competitive world, no sports are immune from sporting injuries. The tendency of the body to over perform against its capabilities is the key reason for most of the sporting injuries. External factors like playing environment, playing surfaces, lack of proper training, and poor protective equipment are other important causes of sports injuries.⁸⁴ Internal factors like strength and endurance plus anatomical abnormalities also can be responsible for sports injuries.⁸⁴

In India, varying formats of these key sports are noticed. A large number of children, adolescent, and adults play these sports on streets on a daily basis for recreation. Street sports outnumber competitive sports played on playing grounds. Street sports are characterized by poor playing environment, poor surfaces, lack of training, and lack of protection (Fig. 25.36).⁸⁵ Presumably, the rate of injuries must be very high, but data to support this are lacking. Next in count are school-level sports, again with poor documentation of injury and treatment. Studies have focused mainly on injuries at a competitive level. The main objectives of studies are to analyze reasons for injuries and to suggest preventive actions. No studies have specifically focused on management aspects of foot and ankle injuries. With these inherent odds, we present sports foot and ankle injuries with its management modalities.

Cricket

Cricket is a globally popular sport being played in 105 countries. It is the second most popular spectator sport after football in the world. Cricket is played on an oval ground with a rectangular pitch with 11 players on each team. Activities done by a player involve batting, bowling, fielding, and wicket-keeping. Though cricket is a noncontact sport played with ball and bat, it requires physical fitness, skill, and strategy. Recently many

formats of this game have evolved to attract more and more spectators from every age group. Such aggressive and attractive formats have increased viewership at the cost of increased rate of injuries to players. While the game has limited contact injuries, activities like bowling, fielding, throwing, diving, catching, and running lead to impaction and overuse injuries. Projectile injuries to any part of players’ body can occur by a hard-thrown heavy cricket ball in spite of protective gear.

In 2016, an international consensus statement on injury surveillance in cricket was introduced. Countries like Australia, England, South Africa, the West Indies, and India have all contributed data. Today the subcontinent has become the hub of cricket, but very limited data is published from these countries. With fewer publications on cricketing injuries, very few publications have focused on the foot and ankle.⁸³ Incidences of cricket injuries, in general, are found to be increasing because of a greater number of games being played with decreasing rest periods between games. The act of bowling and fielding (41.3%) accounts for the highest number of injuries, with hamstring sprain being the most common injury. Based on the mode of onset, Dhillon et al. classified cricket injuries as acute (64%–76%), acute on chronic (16%–22.8%), and chronic (8%–22%).⁸³ Younger players under the age of 22 sustain more chronic overuse injuries than do older players. Stretch et al. in his study mentioned that lower limb injuries (48%) rate highest in numbers followed by back injuries (22.8%), upper limb injuries (23.3%), and neck injuries (4.1%). Hamstring and quadriceps sprains form the most common lower limb injuries.⁸⁶

Studies have reported that 11% of injuries affecting fast bowlers involve the foot and ankle.^{83,86} In general, the forefoot is more prone to acute injuries while the hindfoot is more prone to chronic overuse injuries. Acute-onset foot and ankle injuries in cricket are hematomas, contusions, ankle ligament injuries, syndesmotic and midfoot sprains, Lisfranc injuries, and turf-toe injuries. Sudden-onset aggressive impact can lead to foot and ankle fractures. Acute and chronic-onset foot and ankle injuries result from overuse coupled with poor training and preexisting unrecognized anatomical abnormalities. Posterior ankle impingement is one such common overuse injury in fast bowlers. Modern lowcut boots with harder surfaces aggravate this problem. Other problems include plantar fasciitis, tendoachilles tendonitis, medial tibial stress syndrome, flexor hallucis tendonitis, peroneal tenosynovitis, intra-articular loose bodies, ankle synovitis, and os trigonum disorders.

In an unpublished study the authors conducted foot and ankle evaluations of 400 cricket players from district-level cricket associations with interesting observations. We noticed that more than 50% of cricketers had undiagnosed anatomical abnormalities in form of pes planus, accessory bones, and pes cavus. Foot postures were found to be different among fast bowlers (pronated) and spinners (supinated). More than 20% of cricketers had a poor bowling posture resulting in easy fatigue, loss of accuracy, and injuries in turn. In all cases coaches and parents were unaware of such anatomical as well as postural abnormalities in players. We even noticed a lack of knowledge about selection and evaluation of footwear among players and coaches.



Fig. 25.36 Street sport (kabaddi) being played by children in a poor environment without the use of protective gear.

Hockey

Field hockey is the second most popular sport in the world after soccer and is played in more than 132 countries around the world. Though it is the national sport of India, it is more popular in northern Indian states. Prabhu et al. and Dhillon et al. in their studies noted that the overall rate of injuries in field hockey is around 6 per 1000 athlete exposures with lower limbs being the most frequently injured site (51%).^{83,87} Out of all lower limb injuries in hockey, ankle sprains are the most common (14% to 19%). Improper footwear plus rapid rotational movements are the key factors responsible for higher rates of ankle sprains in hockey. Overuse injuries to the ankles and feet are also very common (18% to 32% of all hockey injuries). Typical examples of these injuries in hockey include tibial stress syndrome, Achilles tendonitis, plantar fasciitis, and stress fractures. Females are more prone to overuse injuries than males. Rarely foot and ankle injuries do also result from direct injuries like being struck by the hockey stick or ball. These injuries range from minor contusions to serious compound fractures.

Kabaddi

Kabaddi is the most ancient sport played in urban as well as rural areas of India. It is a type of contact sport that is very simple, inexpensive, and easy to organize. A player has to raid the opponent's court to tag members of the opposite team while continuously chanting the word 'Kabaddi' and holding his breath, while on the side of the opposition, they act as a team, to prevent any kind of tagging of their team member (Fig. 25.37). Because of its inherent nature of heavy contact in the form of tackling each other with aggression, kabaddi is an injury-prone game. Large pulling and pushing forces to the lower body result in injuries like ligament and tendon sprains and tears in the ankle and foot. Direct collision with other players also results in ankle and foot injuries. Injuries also occur due to falls on the hard or uneven ground. Hell and Schonle mentioned in their study that ankle injuries form 55% of all injuries due to collision with opponents.⁸⁵ Ankle sprains are the most common injury and occur due to sudden twisting while running or jumping.

Inflammation of Achilles tendon due to overuse results in ten-donitis. One of the rare injuries documented by the corresponding author was habitual medial dislocation of the first MTP joint (Fig. 25.38).

Kho Kho

Kho kho is one of the most ancient and traditional games and is a modification of a "Run Chase." In the game, the player runs around two poles chasing the opponent to touch him or her and send the opponent out of the game (Fig. 25.39). It requires not only fast running but also sudden strategic stopping to interchange runners. Thus, it is a game of strength, speed, stamina, and skill. The game develops endurance and intellectual capabilities. The game also develops sportsmanship, team building, and obedience among players. Participation is highest among schoolchildren (69%) followed by college (50%) and university (34%) students. Thus, in India the participation level decreases as the education qualification increases. In a study of 503 kho kho injury cases, Jayati Sen reported that knee (14%) and back (13%) injuries were the most common form of injuries.⁸⁸ Ankle (11%) and foot (10%) injuries were next in line, with males being more prone to ankle and foot injuries than their female counterparts. Ankle injuries comprise ankle sprains due to distortion followed by a concussion. Toe injuries are also common due to twisting and falls. Fractures of the foot and ankle do occur due



Fig. 25.37 Game of kabaddi being played at a competitive level.

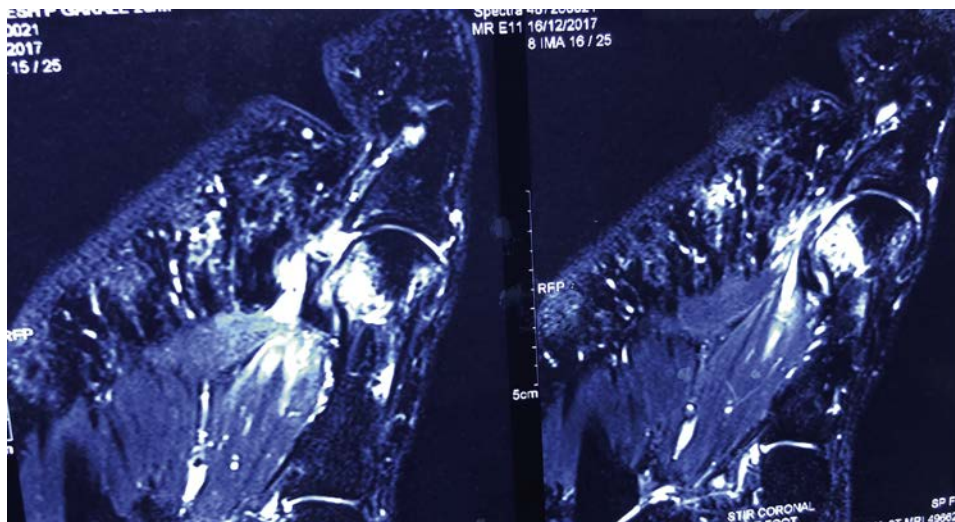


Fig. 25.38 MRI pictures of a professional kabaddi player who injured lateral capsule-ligamentous structures resulting in recurrent dislocation of the first metatarsophalangeal joint.

to falling to the ground or due to a violent collision. Common reasons for injuries in kho kho are lack of use of protective gear, poor playing grounds, and poor training.

Management

Foot and ankle orthopedics has yet to develop as a specialty in India. Sports medicine is slightly better positioned. Available data on foot and ankle sporting injuries is only from injuries happening at competitive level. Interestingly in India, the number of games played at competitive level is much lower than games played at street level for recreation. There are no studies focused on injuries happening in street-level sports. School- and college-level sports do have data of injuries but are very scanty because of lack of precise record keeping. No registry of sports injuries has yet been created for any sport to date. Naturally, the data we get represent just the tip of the iceberg. Street-level injuries presumably are being managed by home remedies like local applications or by bonesetters with remedies in the form of manipulation plus local applications. Complications like cellulitis, abscess, and stiffness follow such unscientific management and are ultimately dealt with by qualified orthopedic surgeons. Many of the street injury cases are also managed by family physicians. Neglected cases are quite common due to being ignored by player or coach in order to play out the season. At competitive level, due to a handful of foot and ankle and sports medicine specialists in India, foot and ankle sports injuries are being treated either by coaches and physiotherapists or by generalists.

Acute ankle sprains are treated with modalities like RICE supported with NSAIDs. Long-term plaster immobilization is also practiced, unlike the recent trends noticed in the western world. There is no trend to acutely repair torn ligaments. Pain and persistent disablement are very common after ankle sprains. A huge number of unresolved ankle sprain cases, in fact, are missed injuries like peripheral talar process fractures, high ankle sprains, osteochondral injuries of talus, etc. In a study of 482 foot and ankle injuries, the corresponding author reported missed injuries with ankle sprains being second highest after Lisfranc injuries.⁸⁹ Chronic lateral ligament injuries are being managed with Brostrom Gould reconstruction of the lateral ligament. At some centers in India, arthroscopy-assisted lateral ligament reconstruction is also practiced.

Lisfranc injuries are often misdiagnosed as midfoot sprains. Documented Lisfranc injuries are managed commonly with

open reduction internal fixation (ORIF) rather than primary fusion. Use of k-wires over screws for Lisfranc injuries is still common, while spanning plates are used at a few centers. Fractures, hematomas, and contusions are managed on their merits as per standard AO (Arbeitsgemeinschaft für Osteosynthesefragen – German for Association for the Study of Internal Fixation) principles like elsewhere in the world.

Overuse injuries like tendonitis and fasciitis are primarily treated by team physiotherapists with rest, NSAIDs, and physiotherapy, failing which, qualified sports medicine specialists get involved. The most common diagnostic modality is MRI. Treatment is in the form of rest, activity modifications, orthotics, and bracing. Modern therapies like extracorporeal shock wave therapy, radiofrequency ablation, and PRP injections are utilized by a few centers without any published data on results. Stress fractures are quite commonly missed. Once diagnosed, they are treated with rest, orthotics, and physiotherapy. Posterior ankle impingement is managed with open surgery at most of the centers as hindfoot endoscopy is evolving. Ankle joint issues like synovitis and impingement are treated with ankle arthroscopy at many centers.

Improved record keeping and precise documentation of sports injuries at every level are the need of the day in developing countries like India.

THE ATHLETIC FOOT AND ANKLE IN IRAN

Iran is a vast country in the Middle East with high mountains, green areas, dense jungles, deserts, and seashores. Four full seasons are experienced in Iran with all types of weather in any time of year. There are different ethnic groups living in this country with different habits and sports preferences.

All categories of sport are available in Iran, but ball games like football and volleyball are considered the most popular sports. Wrestling, rock climbing, and martial arts are other popular activities. Most uniquely, Zurkhaneh (a form of martial arts and gymnastics) and Chovgan (a horse-riding team sport) are traditional Iranian sports.

Mountain Tracking and Rock Climbing

These are popular sports in Iran and recently have changed with modern techniques (Fig. 25.40). This sport is part of real living in some western mountain areas.

Rock climbing can be potentially risky for acute foot and ankle injuries (Fig. 25.41). Although there are several reports, emphasizing safety of the legs, ankles, and feet as long as guidelines are followed, up to 50% of acute injuries occur in lower extremities. These injuries increase in mountain tracking, especially in the form of an acute ankle sprain and fractures of the calcaneus and talus. The most common injuries occur in nonprofessionals during the winter. Some of the injuries are related to flexible shoes that may lead to hallux valgus or hallux rigidus over time. Most climbers prefer to use rigid boots with better support in foot and ankle on uneven ground. Still they may induce some overuse injuries like subungual hematoma. Professional rock climbers use specific shoes that are asymmetric and have forefoot turn-downs. These shoes reduce pressure and injury on toes, although they are not suitable for long-distance walking.⁹⁰⁻⁹²



Fig. 25.39 Game of kho kho being played at a competitive level.

Chovgan

Chovgan is a horse-riding team sport. Historically it has been considered an aristocratic game done by kings in the first millennium CE, and most injuries occur to the elbow and head, yet there are a few reports of leg and ankle injuries.

Wrestling

Wrestling is considered the most popular traditional sport of Iran especially in northern areas, with few variations (Figs. 25.42 and 25.43). In this sport there are lots of pushing, cutting,



Fig. 25.40 Mountain tracking has always been associated with foot injuries; people usually use stiff boots that help them with long-distance journeys but are not ideal for rock climbing.



Fig. 25.41 Rock climbing as a growing sport; note the specific turn-down shoes that reduce pressure on forefoot and toenails.

and twisting motions, making it very strenuous on the foot. Although there is also trauma from direct contact, overall injury is not common in this sport. Most injuries including ankle sprain, Achilles tendinosis, and peritendinitis, which occur during competition season.^{93,94}

Pahlevaniv

Pahlevaniv (bastani or zurkhganeh) is a unique traditional martial art of Iran, which is infused with ethical and moral rules. All parts of this sport are rigorous and include wrestling, whirling, club exercises, and calisthenics synchronized with a special style of music incorporating bells, drums, and chanting. This sport is performed barefoot in a ceremony with a unique engraved suit. Part of the activity consists of different kinds of running, hopping, leaping, short jumping, and fast spinning.^{95,96} (Figs. 25.44 and 25.45)

FOOT AND ANKLE INJURIES CAUSED BY TRADITIONAL JAPANESE MARTIAL ARTS

In Japan, there are many forms of traditional martial arts that are still actively practiced today. This chapter explains in detail



Fig. 25.42 Wrestling with chookhe is one of popular traditional kinds of wrestling. It was played in special ceremonies.



Fig. 25.43 Wrestling with chookhe is associated with increased risk of foot and ankle sprains.



Fig. 25.44 Zurkhaneh federation with Pahlevani. The picture shows a special form of spinning. This activity can produce acute and chronic high ankle sprains.

foot and ankle injuries associated with the three most popular martial arts: judo, sumo, and kendo. Although the origin of these martial arts is not known, the earliest known mention of their basic forms is found in Japanese documents written during the eighth century. In the last half of the nineteenth century, the modern rules for these martial arts were established, and people began to practice them as sports. Because these martial arts are practiced barefoot, there is a high incidence of ankle and foot injuries among their practitioners. However, because playing surfaces and styles of competition differ markedly among these three martial arts, they are associated with different foot injuries.

Judo

Because judo is an Olympic sport, the number of people who practice judo is increasing worldwide. A judo contest is a fight

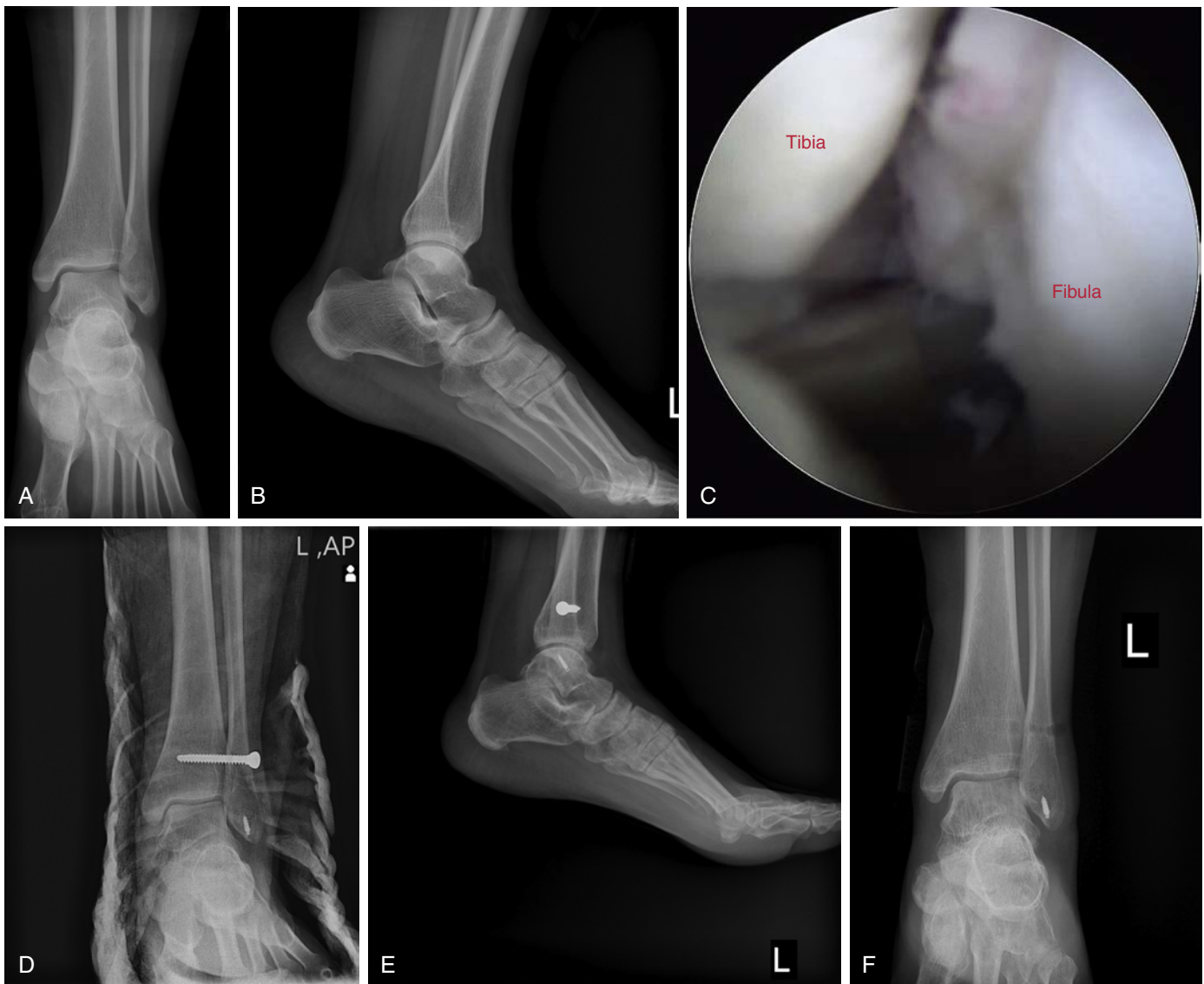


Fig. 25.45 High ankle sprain in an athlete from repetitive twisting activity. A and B, preoperative radiograph shows no problem. C, Arthroscopy was performed because of persistent pain on anterior inferior tibiofibular ligament (AITFL) and syndesmosis. There was a positive arthroscopic cotton test with marked synovitis. The 3-mm shaver is easily passed into syndesmosis space, indicative of instability. D and E, early postoperative. F, Four months after operation the syndesmotic screw was removed.

between two contestants who wear judo suits and fight on tatami (straw) mats. The first contestant to score a full point ("ippon") wins. A contestant can score a full point by throwing the opponent on his or her back, holding the opponent for 30 seconds, or making the opponent concede. Injuries almost always are caused by throwing moves. Many judo injuries occur in the lower extremities, particularly at the knees, ankles, and feet. Because mild foot injuries are so common, those who sustain them rarely seek treatment at a medical institution.

The most common foot injury is ankle sprain; about half of all judo practitioners suffer an ankle sprain at some point (Fig. 25.46). Severe inversion sprains typically are accompanied by osteochondral fracture of the talar dome. Also, ankle instability persists in many cases, and many people who practice judo for a long period develop osteoarthritis of the ankle. Also, when a strong external force is applied, a malleolar fracture occurs, but plafond fractures and talar fractures are rare.

The incidence of toe injury is high among judo practitioners. Turf-toe is a well-known injury associated with sports played on turf, such as American football. Most cases of turf-toe are caused by excessive dorsiflexion of the great toe. When a foot sweep is attempted in judo, the sweeping foot is in the equinovarus position and is swung horizontally. If the sweeping foot gets caught in the seams of the tatami mats or on the opponent's foot, the MTP joint of the great toe is excessively plantarflexed (Fig. 25.47). Although this generally causes sprain without a fracture, severe bending can cause a chip fracture. This type of toe injury is sufficiently unique to judo to merit its own name (perhaps "tatami toe"). If accompanied by osteochondral damage to the MTP joint of the great toe, osteoarthritis can lead to hallux rigidus. Although toe injuries most often affect the great toe, sometimes they can affect the lesser toe.



Fig. 25.46 Many judo injuries occur in the lower extremities. During "Ohsoto-gari," a throwing technique, the foot and ankle assume an equinovarus position. Inversion sprain can occur in the foot when defensing (arrow).

Sumo

Sumo is a sport in which two wrestlers fight on a round ring that is made of packed earth and has a diameter of about 4 m. Sumo wrestlers wear nothing but a loincloth belt. In each bout, two wrestlers initially face each other from behind two parallel lines at the center of the ring. Once the bout begins, they collide violently, like guards and tackles in American football. The loser is the first wrestler to touch the ring with any part of the body other than the bottom of the feet or the first wrestler to go out of the ring. Sumo wrestlers try to push each other out of the ring, and heavy body weight confers an advantage in this pushing. Consequently, sumo wrestlers intentionally try to achieve and maintain a heavy body weight. Although the most common clinical problem associated with sumo is lumbar pain, injuries in the lower extremities account for more than half of all injuries associated with sumo.

Ankle and foot injuries account for about 15% of all sumo-related injuries. It might seem that this is a low percentage for a sport that is practiced barefoot. The reason for this low percentage is the manner in which sumo wrestlers move, by shuffling their feet instead of lifting their feet off the ground (Fig. 25.48). In sumo, the friction between the ground and the soles of the feet is important in keeping a wrestler in position. If either foot comes off the ground for even a short time, the wrestler easily can be pushed out of the ring. Thus, shuffling helps to prevent a wrestler from being pushed out of the ring. During shuffling, the knees are bent in the valgus position, the lower legs are abducted, and the feet are pronated. As a result, sumo training strengthens the peroneal muscles, thus lowering the incidence of inversion sprain. Furthermore, even if a sprain occurs, it usually does not cause persistent ankle instability.

Foot shuffling and squatting with knees spread apart are the basic movements of sumo, and during these movements the ankles are dorsally flexed. Thus, in competition, the ankle often



Fig. 25.47 The great toe is easily plantarflexed (arrow) ("tatami toe"). Tatami (straw) mats typically are used as a floor covering in judo.

is dorsally flexed (Fig. 25.49). Furthermore, when a wrestler braces against being pushed out of the ring, the ankles are in excessive dorsiflexion. On the anterior surface of the ankle, the tibia often collides with the neck of the talus, causing impingement exostosis. Because this condition exists in most sumo wrestlers, and not many sumo wrestlers have ankle instability, its onset must involve collision.

Because sumo wrestlers are heavy and collisions are violent, there is a high incidence of bone fracture around the ankle. Pronation-external rotation-type malleolar fracture is common because the lower leg is abducted and the foot is pronated, unlike the case in sports that are played with a ball. However, despite their severity, rehabilitation of such injuries is faster than for soft-tissue injury.

Severe toe injuries are less common than severe ankle injuries. Unlike judo, sumo does not involve many moves in which



Fig. 25.48 Exercise of shuffling. Keeping the feet on the ground improves stability in this sport, which revolves around collisions and pushing. The knees are bent in the valgus position, the lower legs are abducted, and the feet are pronated. The playing surface is packed earth. Sumo wrestlers tape their toes and wear “tabi” to prevent lacerations on the soles of their feet.



Fig. 25.49 The ankles often are dorsally flexed in a bout. The incidence of anterior ankle impingement exostoses is high in sumo wrestlers.

a foot in the equinovarus position is swept sideways. However, lacerations of the skin on the plantar side of the first MTP joint are very common. Some sumo wrestlers prevent such lacerations by taping their toes or wearing Japanese thick-soled socks (“tabi”) (see Fig. 25.48).

Kendo

Japanese swords are the symbol of the Samurai culture. Unlike Western swords, Japanese swords are held using both hands. Kendo is a sport modeled after samurai sword fighting, using bamboo swords resembling Samurai swords. Practitioners wear protective pads on the face (“men”), belly (“do”), and forearm (“kote”). A point is scored when a bamboo sword cleanly hits one of the protective pads. A kendo practitioner holds a bamboo sword using both hands, with the right hand in front of the left hand, somewhat like a right-handed baseball player holding a bat. The two competitors face each other so that the tips of their bamboo swords are lightly touching (Fig. 25.50). Right- and left-handed practitioners take the same stance. The right foot is placed in front, while the left foot stays back. Competitors put their weight on the front half of each foot and slightly lift the heels so that they can move very quickly.

Kendo is generally a safe sport, with a low incidence of fracture, but mild toe injuries are quite common. Beginners often complain of heel pain. Because kendo is practiced barefoot on a wooden floor, there is great impact on the feet during kendo moves. About 40% of kendo practitioners develop hemoglobinuria because red blood cells in the skin and subcutaneous tissue of the sole are destroyed by the impact of the heel hitting the floor. Some kendo practitioners develop a condition called “black heel,” which is characterized by ecchymosis on the sole of the feet. Usually, heel pads are used to treat this condition.



Fig. 25.50 A starting posture of kendo is demonstrated. Note that the sport is practiced barefoot on a wooden floor. The right leg is in front of the left. Weight is kept on the forefoot.

In kendo, the most common severe injury is rupture of the Achilles tendon. This injury almost always occurs in the left leg, because of the positions of the legs in the kendo stance (Fig. 25.51). During kendo moves, a great amount of force is applied to the left leg. When the body pushes forward, the triceps muscle of the calf is tensed, and the Achilles tendon can rupture if there is a delay in plantarflexion of the ankle. In most sports, rupture of the Achilles tendon is rare among young people, but among kendo practitioners, this injury is somewhat common in high school students. This supports the theory that a great amount of force is applied to the Achilles tendon in the left leg when the body pushes forward in kendo. Rupture of the Achilles tendon is rare among beginners but is more common among skilled practitioners. Most of those who sustain this injury chose to undergo surgery, and rehabilitation takes 6 to 12 months.

FOOT AND ANKLE INJURIES OF RACETRACK JOCKEYS IN MEXICO CITY

Introduction

Throughout the world since the time of the ancient Greeks, horse races have been popular.⁹⁷ Since the opening of the Mexico City Racetrack, *Hipódromo de las Americas*, in 1943, this sport has drawn public interest in Mexico. Although much is known about the sport nationally, little has been written on foot and ankle injuries of race track jockeys. This is a retrospective review of 30 years of foot and ankle injuries as chronicled by their designated trauma orthopaedic surgeon.

Every season (from January to October every year) the active jockey population is 24 to 30. These athletes typically have a petite physique with a weight between 42 kg (92.5lb) and 54 kg (119.05 lb) and a height that ranges from 1.31 m (4.2ft) to 1.65 m (5.4 ft), while the thoroughbred horses they ride have a

height between 1.62–1.64 m (5.3 ft) and can reach a speed of 60 to 70 km/hour (37–43 mph).⁹⁷ Jockeys have special equipment to protect them from trauma (riding helmet and special vests for impact absorption), but there are still some parts of their body exposed, such as their pelvis and lower limbs. Jockeys' injuries during horse races have been published in the United States and Australia.^{98,99} These injuries include jockeys' head or neck (18.8%), the leg (15.5%), foot/ankle (10.7%), back (10.7%), arm/hand (11.0%), and shoulder (9.6%).

Jockeys' lifestyle and nutrition status are a very important factor of vulnerability for this kind of injuries. Apart from the risk of riding a high-speed racing horse in competition, these patients typically have an unbalanced diet. Occasionally they ingest alcohol, use diuretics, and have sauna sessions in order to maintain a low weight before races. All of this has been reported to increase fracture risk.¹⁰⁰

There are lots of minor to moderate injuries that are attended on the site of the accident or the racetrack emergency room, such as minor head and limbs contusions and minor skin abrasions. Mild to moderate injuries that are treated at the hospital emergency room include lacerations and trauma involving the neck, wrist, knee, and ankle. Severe trauma is always treated on an inpatient basis in the hospital.

Medical records of jockeys treated for orthopedics and trauma purposes since 1978 were reviewed identifying 1333 cases that had musculoskeletal treatment.

The top cause of hospital admittance was upper extremity injury (52.36%) (Table 25.1). Of this, clavicle and wrist fractures were the most frequent.

Lower limb was second with 31.58% and a total of 421 patients.

Starting in July 1978 until December 2014 (with a pause between 1996 and 2001 while the racetrack was closed, 399 patients were admitted to the hospital for surgical treatment because of foot and ankle injuries. (Table 25.2)

Mechanism of Injury (Box 25.1)

During preparation for the race, the horse is taken for saddle placement, placing the jockeys at risk for a crush injury due to being stepped on by the horse. Usually the jockey or the trainer are vulnerable to a soft-tissue crush injury or a metatarsal fracture. We had 18 feet with one to four metatarsal fractures that needed reduction with internal fixation with K-wires or plates/screws.



Fig. 25.51 An offense hit on a face guard ("men"). The right leg goes forward during a lunge. Excessive force is loaded on the left Achilles tendon during the sport. The incidence of Achilles tendon injuries is high relative to the other martial arts.

TABLE 25.1 Injuries That Required Hospital Admittance

Injuries that required hospital admittance		Cases
Upper Limb	52.36%	698
Lower Limb	31.58%	421
Pelvis	0.60%	8
Head Trauma	4.80%	64
Cervical/Lumbar Spine	9.52%	127
Multiple Costal Fractures	0.30%	4
Abdominal Trauma	0.80%	11
Total		1333

A very specific area where foot and ankle injuries presented was the departure gate. This happens because whenever the gates are suddenly opened, the horse jumps forward and the jockey’s foot in the saddle stirrup locks within the gate, experiencing a violent external rotation inducing a bimalleolar fracture. This occurred in 22 cases. In our review, the most common mechanism was the jockey falling from or with the horse during a race. When a horse falls while running, limb fractures are sustained, but due to the speed and weight of the animal, sudden death may occur.⁹⁹ An uneven surface on the racetrack could lead to a horse stumbling, a front limb fracture, and rear limbs collapsing. Since the jockey is projected forward, the upper limbs sustain direct trauma.

This is why clavicle and wrist fractures were the most frequent fractures in the 1333 patients in our study. Whenever a jockey is down, other running horses can trample the athlete, resulting in spine, pelvis, and/or abdominal trauma.

Open Fractures

There were 23 open fractures in this review. All of these were treated with surgical irrigation and debridement before open reduction internal fixation (ORIF). These cases are treated with triple antibiotics: metronidazole, penicillin, and gentamycin, given the contamination from the dirt and fecal matter.

Recovery Time on Bimalleolar Fractures

Jockeys, as with other elite athletes, have the determination to quickly return to work.

All 201 patients were treated with regular ORIF with medial screws, lateral plate, and screws as well as syndesmotic fixation as needed. Typically, following the surgery, there was no weight bearing until the sixth week. Syndesmotic screws were removed between weeks 3 and 4. After this they started aggressive physical therapy (Figs. 25.52 through 25.55).

All of these patients returned to horse racing between week 9 and 12 postoperatively.

Of course, the youth, low BMI, and nonchronic condition play a very important role in this recovery time.

Protective Gear

Apart from riding helmet, goggles, and rigid vest, further improvement in this and new equipment has been advised in order to lower the incidence of fractures in jockeys.⁹⁷ Foot and ankle protection is controversial. While racing, the jockey’s ankle must have 110–115 grades of dorsiflexion, so a more rigid or complex boot that could provide more protection definitely would diminish this ROM and potentially the performance.



Fig. 25.52 Antero-posterior view of a preoperative distal tibial and fibular malleolus fracture.



Fig. 25.53 Lateral view of a preoperative distal tibial and fibular malleolus fracture.

TABLE 25.2 Type of Foot and Ankle Injury		
Type of Foot and Ankle Injury	Feet/Ankles	
Bimalleolar Ankle Fracture	201	50%
Unimalleolar Ankle Fracture	101	25.3%
Trimalleolar Ankle Fracture	25	6.2%
Distal Tibia Fracture	22	5.5%
Metatarsal/Phalanx Fractures	18	4.5%
Pilon Fracture	15	3.7%
Lisfranc Fracture/dislocation	13	3.2%
Syndesmotic Injury w/o Fracture	4	1%
Total	399	

BOX 25.1 Mechanism of Injury

1. Falling from the horse while racing
2. Foot/ankle locking in the departure gate
3. Standing horse stepping on foot
4. Running horse stepping on foot



Fig. 25.54 Antero-posterior view of an open reduction internal fixation (ORIF) of distal tibial and fibular malleolus fracture treated with plate and screws to distal tibia and fibular malleolus.



Fig. 25.55 Lateral view of an open reduction internal fixation (ORIF) of distal tibial and fibular malleolus fracture treated with plate and screws to distal tibia and fibular malleolus.

Summary

Injuries to the musculoskeletal system are frequent in jockeys. They range from contusions and simple trauma to limb fractures, head trauma, spine injuries, or death. Bimalleolar ankle fractures, Lisfranc injuries, and metatarsal fractures are common. Open fractures are also frequent, requiring immediate treatment with triple antibiotics and aggressive debridement and stabilization.

SPORTS INJURIES IN SOUTH AFRICA

Overload Injuries to the Second Metatarsal

South Africa is a country blessed with wonderful weather, so most South Africans love their sporting and outdoor activities. Golf, tennis, rugby, football, watersports, cycling, and running are very popular, and sport-related injuries are thus very common. These include ankle sprains, Achilles' tendon problems, stress fractures, Lisfranc injuries, impingement syndromes, and so forth.

People of all ages participate in running, and we are famous for our ultra-marathon races, especially the Two Oceans marathon (56 km) and the Comrades marathon (89 km). Thousands of people line up at the start of the Comrades marathon every year (23,818 participants in 2018). Few sportsmen are as fanatic and passionate about their sport as the ultra-distance runner. The mean age of participants in this grueling event is around 40 with the age range varying from 20 years old to octogenarians.

This presents a special group of injuries to the sports physicians and foot and ankle surgeons, namely injuries associated with forefoot pain due to overloading of the second metatarsal. Stress fracture of the second metatarsal is a common diagnosis, but we want to highlight overload injuries of the second MTP joint in this selected group of patients.

As in nonathletes, it is more common in women and usually unilateral. It initially presents with forefoot pain and swelling only when running and then later progresses to pain with daily activities. Deformities in the sagittal and coronal plane develop later in the progression of the disease. Often it is possible to find a biomechanical reason for the development of this overload other than a long rigid second metatarsal (Morton foot), which should be addressed in treatment. Surgical treatment of the symptoms and not the cause will end in recurrence of the injury, including instability and associated deformities.

Patho-anatomy that causes overloading of the forefoot includes the following:

- Hypermobility first ray
- Abnormally long second metatarsal/Short first metatarsal
- Metatarsal primus elevates
- Hallux valgus
- Short gastro-soleus complex

This results in synovitis that causes the capsuloligamentous structures of the MTP joint to become stretched, leading to instability. This can progress to attenuation of plantar plate and extension of the MTP joint (sagittal component) and an associated crossover deformity (coronal component). Progression of instability can lead to dorsal dislocation of MTP joint, predisposing to hammer toe deformity.

Overload injury can also present with different patho-anatomy including cartilage injury and synovitis.

Case report

Thirty-nine-year-old healthy female marathon runner with a 15-month history of right forefoot pain and swelling. Diagnosed with a stress fracture of the second metatarsal and treated with orthotics, twice with cortisone infiltration, a boot



Fig. 25.56 Cartilage was deficient centrally and was debrided and drilled.



Fig. 25.57 X-ray showing a dysplastic first metatarsophalangeal joint with a short proximal phalanx.

brace, and ultimately 7 months of cessation of running. On physical exam there was swelling, crepitus, and tenderness of the second MTP. The joint was stable ([Fig. 25.56](#)). Evaluation of the alignment and mobility of the rest of the foot and ankle was normal. There was no gastrocnemius tightness. X-rays demonstrated a shorter right first metatarsal. MRI revealed synovitis of the second MTP ([Fig. 25.57](#)). Patient was treated with a Weil osteotomy to shorten the second metatarsal 2 mm. A double cut was made to elevate the metatarsal. The cartilage was deficient centrally and was debrided and drilled ([Fig. 25.58](#)).

Mechanical pain associated with running will be the main initial complaint. A sudden increase in distance could be the initiating factor in this overuse injury. In obtaining a history from these patients, it is important to inquire about previous surgery, i.e., previous Bunion surgery, with resultant shortening or elevation of the first ray. Complaints of swelling of the MTP joint indicating synovitis of the MTP joint are common. Most patients would have visited a sports physician and received cortisone injections into the MTP joint with the possible worsening of any deformities. A change of footwear or worn-out shoes might also contribute to the injury.



Fig. 25.58 Swelling, crepitus, and tenderness of the second metatarsophalangeal. The joint is stable.

On examination, it is easy to localize the pain to the second MP joint, and there is usually obvious swelling present on the dorsal aspect of this joint. The joint must be tested for instability with the drawer test and compared to the other foot. Tightness of the Achilles' tendon should be excluded and the Silfverskiöld test performed. We always examine the first ray for instability and alignment and exclude other reasons for pain in the area like sesamoid pain, stress fracture, and Morton's neuroma.

Investigations

Plain standing radiographs are routinely performed. With the high accuracy of clinical examination for most diagnoses including plantar plate injury, MRI is reserved for cases when the diagnosis and treatment plan cannot be established from the clinical findings or to exclude other pathology, e.g., OCLs.

Treatment should include:

- Adaptation of footwear as necessary (running and daily shoes)
- Orthotics—Medial arch supports with metatarsal pads
- Modification of mileage and cross training
- NSAIDs

Case report

A 44-year-old female marathon runner had gradual onset of pain in the left forefoot with running. Her usual distance for training was 100 km/week. She was treated elsewhere for a neuroma with cortisone infiltration and physiotherapy. On physical exam she was noted to have a tight gastrocnemius, moderate hallux valgus, synovitis second metatarsal phalangeal joint, and slight valgus deviation of the first and second toes. X-rays revealed a dysplastic first MTP joint with a short proximal phalanx ([Fig. 25.59](#)). Treatment began with footwear modifications, rest, and orthotics. Once symptoms subsided, she gradually built up her distances but stayed limited to training distances of 20–30 km/week. She also began swimming for alternative exercise.



Fig. 25.59 MRI showing synovitis of the second metatarsophalangeal.

We do not support injecting the joint with cortisone, as we have seen disastrous results with it, especially if it allows the patient to continue with running and participating in races. This includes deterioration of the stabilizing structures around the joint and progress of intra-articular pathology. As this problem is sometimes a critical injury preventing the athlete from continuing with running ultra-distance races, a change in approach and even sporting activity should be considered.

Surgical treatment options differ from patient to patient. It is important to treat not only the joint problem (synovitis/cartilage injury/instability) but also the underlying mechanical overload of the MTP joint. This includes different metatarsal osteotomies, stabilizing and aligning the first ray and gastrocnemius slides. As mentioned above, these (e.g., gastrocnemius slide) may affect the patient's ability to participate in running sports. When there is MTP instability, a plantar plate repair is performed through a dorsal approach. A Weil osteotomy is recommended for access to the plate and to alter the length and height slightly. Finally, any IP deformity is addressed as indicated. When the plantar plate is irreparable owing to insufficient tissue, a flexor-to-extensor transfer is performed. More commonly a Cobb II type procedure is performed. In this technique the extensor digitorum longus (EDL) is released distally and routed through a drill hole in the neck of the metatarsal or sutured to the plantar plate.

Postoperatively patients are allowed weight bearing in a rigid shank postoperative shoe for 6 weeks.

The operated toe is strapped in a plantarflexed position for 6/52. Active and passive ROM exercises begin at 6 weeks. Patients may advance to normal footwear at this stage.

Above-discussed patient group can be very demanding, as they are extremely passionate about their sport. Diagnosis is usually relatively straightforward. Unfortunately conservative and surgical treatment can be challenging for the patient and the surgeon.

A PERSPECTIVE ON THE FOOT AND ANKLE IN SPORTS FROM THAILAND

In Thailand, there are different kinds of unique cultural games and sports that have implications in foot and ankle injury. Muay Thai and Sepak Takraw are two that will be the focus of this section.

Muay Thai

Muay Thai, an ancient traditional martial art of Thailand, has been developed with the concept of using the body as a weapon in close-range person-to-person combat. It is also referred to the “Art of the Eight Limbs” because of its use of punches, kicks, and elbow and knee strikes as opposed to other martial arts such as Judo and Taekwondo in which only punches and kicks are used. This is the reason why Muay Thai is widely regarded as a devastating martial art, with a knockout rate ranging from 28% to 56% in all professional fights.

More than half (55.4%) of Muay Thai boxers reported an injury in their most recent fight. The lower extremities (51%) were the most commonly injured body region during fights.¹⁰¹ Most of the reported injuries were soft-tissue injuries contributing to at least 80–90% of all injuries. The severity of these soft-tissue injuries were usually lower on the injury severity scale.¹⁰² Injury prevalence is also associated with fight experience level, use of protective equipment, level of competition, and previous injury history.^{101–103}

Even though Muay Thai is a type of kickboxing, it has a distinctive kicking style. The kicks in Muay Thai have been divided into two types. The first is the front kick (Fig. 25.60) using the forefoot, especially the first metatarsal head, to hit the target. The second is the side kick (see Fig. 25.60), in which the shin is used as a weapon to attack the target. Because of these distinctive kicking styles, the most common injury in Muay Thai is contusion of the shin. However, because Muay Thai boxers can use their elbows and knees to protect themselves from attack, if the kicks miss the target, there is a chance of hyperextension ankle injury or soft-tissue injury of the dorsal portion of the foot. Thus, the rate of foot and ankle injuries in the well-trained Muay Thai boxers is fewer when compared to other types of kickboxing. Regarding the prevalence of shin contusion, use of shin guards is recommended (Fig. 25.61) for all Muay Thai beginners.

There are unique types of attacks called Kon Muay, advanced tactics to take down the opponent. Some of these Kon Muay tactics (Fig. 25.62) can result in improper landings, causing sprains. Moreover, Muay Thai allows the boxers to sweep under the legs of their opponents by using the dorsum of the foot and anterior aspect of ankle. With these types of attacks, twisted ankles and Achilles tendon injuries are common.

Vaseenon et al.¹⁰⁴ found that the most common foot and ankle problem in Muay Thai boxers was callosity over high contact zones (77.5% forefoot, 16.3% malleoli, 3.1% midfoot, and 3.1% heel), followed by gastrocnemius contracture, toe deformities, and heel pain. The causes of these common problems are barefoot training with tiptoe dancing and use of the forefoot as a pivot point when kicking.

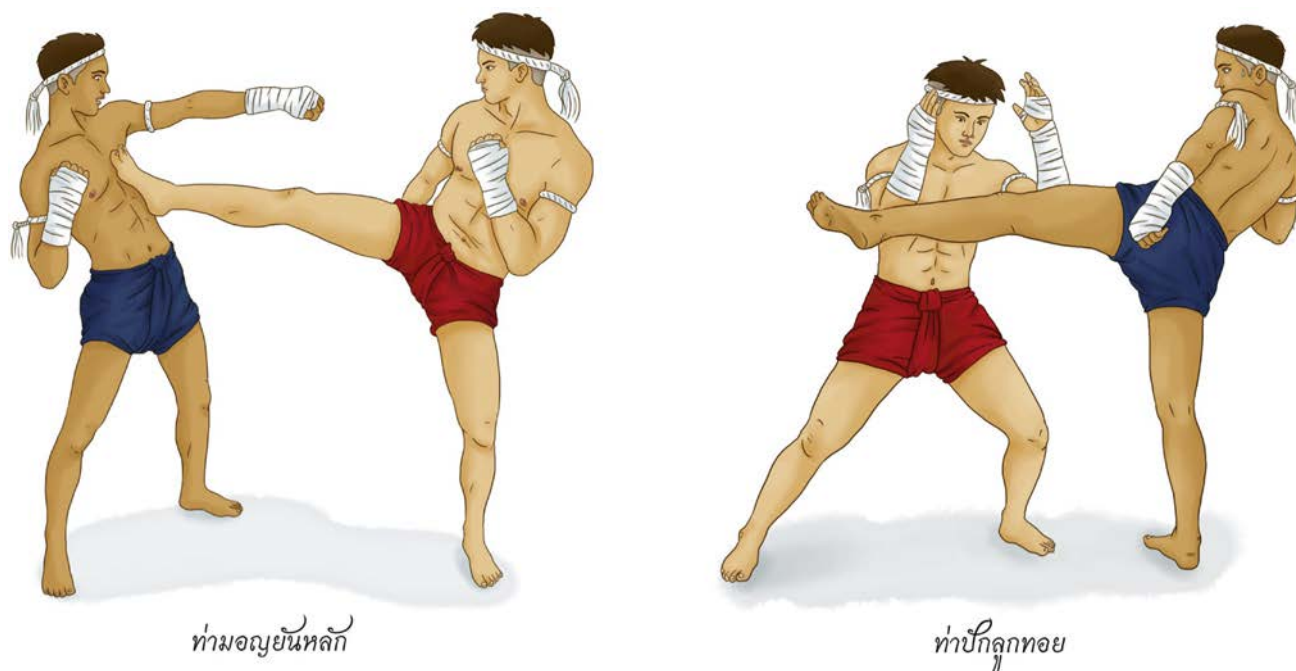


Fig. 25.60 The front kick (left) and the side kick (right).



Fig. 25.61 Shin guards.

Sepak Takraw

Sepak Takraw is a game originating from Southeast Asia. The word “Sepak” is from the Malaysian language that means “kick” and “Takraw” originates from the Thai language, meaning “rattan ball.” In the fifteenth century, the game rules varied slightly in different parts of Southeast Asia. Traditionally, Thai people formerly played this game by standing in a circle and used their feet to pass the ball back and forth between them, which was called “Circle Sepak Takraw.”

In 1833, the Thai sport association introduced the volleyball-like style net and held the first contest. By the 1940s, the first set of formalized rules were introduced, and the game took the official name of Sepak Takraw with the International Sepak Takraw Federation (ISTAF) governing the sport worldwide. Like the volleyball game, the Sepak Takraw players are allowed a maximum of three contacts with the ball to get it over the net. Unlike volleyball, the players are allowed to use their legs, head, and trunk to contact the ball but are prohibited from having any contact with their arms or hands.

There are three basic player positions in this sport. These consist of the server, Tekong (Back), feeder (setter), and kicker (striker). Each particular position carries a particular risk for a specific type of injury. The kicker position carries the highest risk of foot and ankle injury, as they also function as a blocker and striker near the net, their kicks consisting of a somersault-like kick (Fig. 25.63). The server and the feeder have a lower chance of injury, but their injuries are specific to their positions. The feeder needs to reach the ball before it contacts the floor and try to feed the ball back to the kicker to make the score, therefore they have a higher chance of tripping while chasing the ball. For the server, when they serve by hitting the ball hard and they miss, they can end up landing badly and may experience a foot or ankle sprain.

In the past, the grapefruit-sized balls were handwoven from bamboo or rattan, but most modern ones are made of rattan or synthetic material with weights between 170–180 grams under ISTAF regulations (Fig. 25.64). These balls are different from the balls used in volleyball, soccer, and rugby. There is no air in the balls, making them stiffer with less bounce. As a result, it requires more energy from the players in order to propel the ball across the court. When more energy is used, especially if the player is not properly trained in technique, there is a higher risk of uncontrolled landing when kicking.

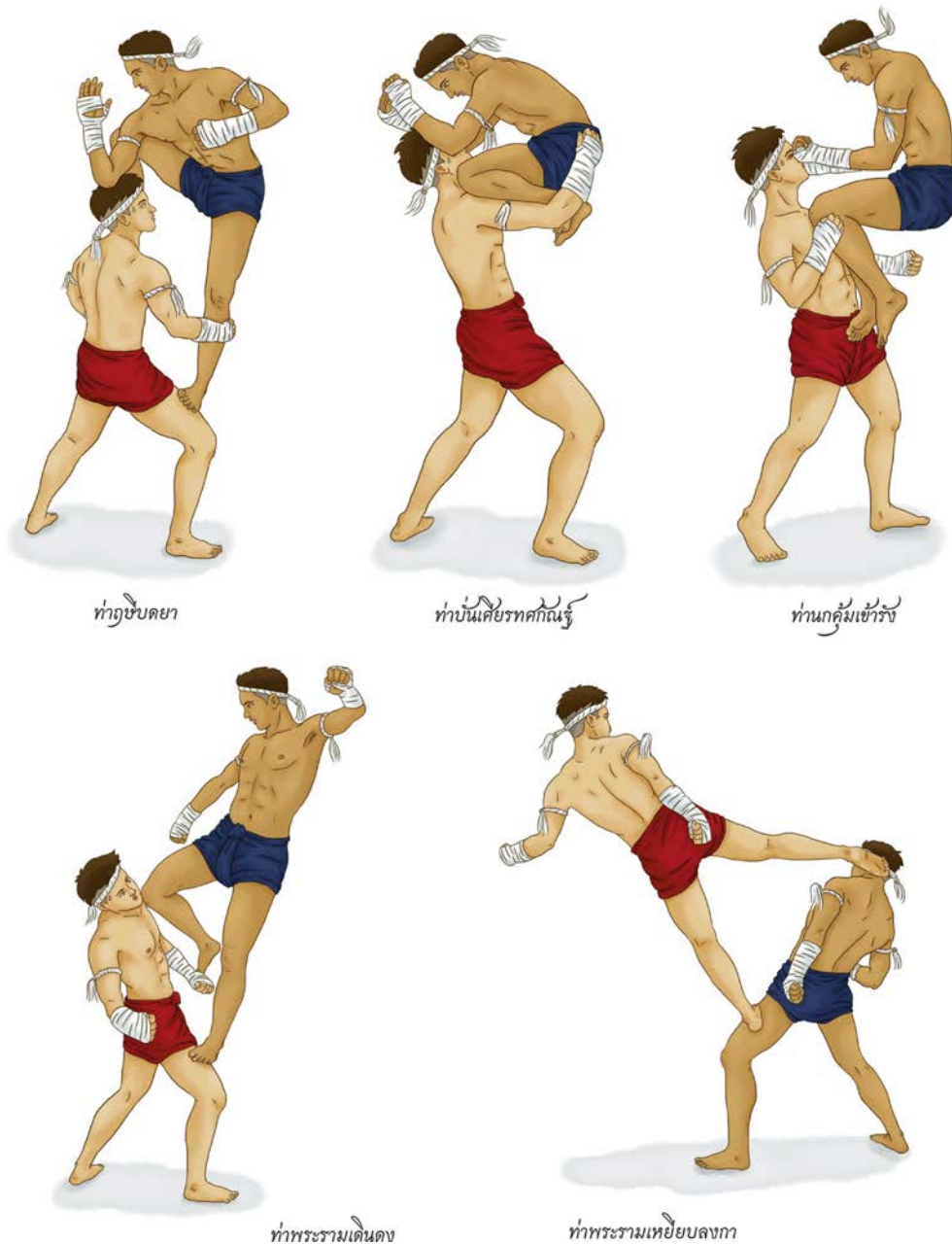


Fig. 25.62 Kon Muay tactics.



Fig. 25.63 Summersault-like kick.

As the ball does not recoil from the player's foot, when kicking, it is necessary to have a large contact area with it in order to have more control. This has led to the requirement of special shoes. Most Sepak Takraw players choose to use a local brand of canvas shoes (Fig. 25.65) that have minimal outer sole in order to maximize the contact surface to the longitudinal arch of the foot during kicks. These canvas shoes are made of a thin cloth so the ball can be felt while kicking, with a low, smooth outer sole that is crafted from a particular type of rubber with high friction and proper weight. With proper weight on these shoes, they can create more momentum as well as increased control, which is necessary because the ball is small and difficult to control.

The disadvantages to these types of shoes do exist. As the insole is a flat surface, there is no support to the arch of the foot, which can lead to plantar fasciitis. The lack of support from the insole will also result in excessive pressure in certain parts



Fig. 25.64 Ball.



Fig. 25.65 A local-brand canvas shoe.

of the foot that are not accommodated, resulting in increased callus formation in the forefoot. Moreover, with a certain type of stiffer rubber, a great deal of force will transfer through the first MTP joint and hallucal-sesamoid complex making these structures vulnerable to injury during landing and pivoting on the forefoot. These shoes also have no ankle support and lack a flared heel, so the ankle will be easily twisted and sprained.

In 58 professional Sepak Takraw national team players, we found the most common injury in this sport is ankle sprain

(42/58, 72%), but only 15% of the time do the players seek medical attention for their sprains. The players are treated conservatively and take less than 2 weeks off to recover. Residual laxity in the ankle is found in 20% (20/58) of the players, but usually it does not affect the player's ability to play as their muscles in the lower leg area are well trained and they are able to compensate.

We also noted common problems including callus formation in 64% (37/58), gastrocnemius tightness (20/58, 34%), hamstring tightness (16/58, 28%), plantar fasciitis (8/58, 14%), Achilles tendinitis (3/58, 5%), stress fracture on the tibia (1/58), and turf-toes (1/58). If improper stretching techniques are implemented, strains and tightness can easily occur at the Achilles tendon, hamstrings, adductor muscles, and iliotibial (IT) band. All this can be corrected by proper stretching, which is emphasized in this sport. If proper time is allocated for stretching, strengthening, and reconditioning then injuries are rare.

FOOT AND ANKLE INJURIES IN UNITED ARAB EMIRATES SPORTS

The United Arab Emirates (UAE) has a desert climate and is situated directly on the Arabian Gulf. This unique geography lends itself to a truly wide variety of sporting activities among the residents. Water sports such as water skiing, wakeboarding, and kite surfing are hugely popular. Other sports such as soccer, rugby, tennis, and squash are commonplace. In the desert,



Fig. 25.66 A unique sport in the United Arab Emirates is hunting prey with falcons. The sandals keep the foot high off the ground to prevent entry of pebbles. They are discarded when running in soft sand.

sand boarding and motor sports command the winter months. Most of the common injuries seen elsewhere are encountered but with some unique scenarios.

Certain niche sports are found in the UAE. For example, falcons are trained to hunt prey. This involves long periods of bonding and progressive conditioning of the predator bird. To accomplish this, the owner often has to run rapidly to tend to his falcon, and in the soft sand this is better accomplished barefoot. Sandals typically are worn (Fig. 25.66) to keep the foot high off the ground to prevent entry of pebbles but are discarded when running in soft sand. Still, minor stub and barb injuries are common. Because the terrain underfoot is soft, the barbs or other objects have little force for any penetration. When training the falcons on the harder desert plain, one wears enclosed shoes (Figs. 25.67 and 25.68) to prevent the entry of foreign objects. Also, ankle sprains (mostly lateral) tend to be relatively minor on the sand because the surface is not firm and thus is very forgiving during inversion.

Arabs have a long tradition with horses. In the UAE, horse racing (speed and marathon) and polo are two sports in which injuries are relatively common. Most tend to be in the upper extremities from falls, but foot and ankle injuries also occur. The prolonged “heel down” position in the saddle can lead to impingement syndromes of the anterior chamber of the ankle, requiring removal of any kissing osteophytes or soft tissues. Because these are mostly symptomatic early in their development, arthroscopic debridement is very successful, with arthrotomy rarely being required. Minor crush injuries of the foot and ankle occur as the horses collide during play, but serious crush injuries from hooves are surprisingly uncommon in the UAE. This could possibly be from the combination of a high standard of horsemanship and well-trained thoroughbreds that are used in the sport.

Motocross has its share of injuries because dirt bikes are ridden in the sand at high speeds. Riders are required to wear body armor and protective footwear (Fig. 25.69). However, unlike hard dirt terrain that causes a violent plantarflexion force of short duration, sand produces a more moderate force of longer duration. This leads to sprains of the anterior structures, with relatively frequent anterior capsular tears. Conservative care

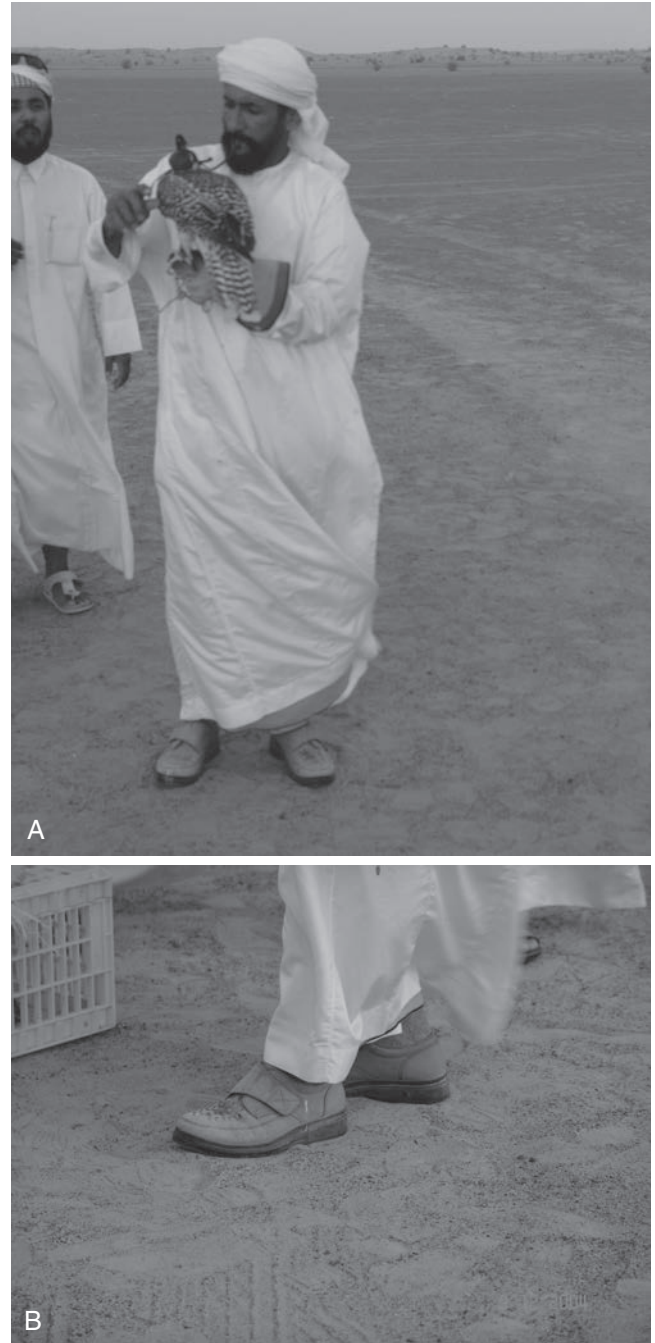


Fig. 25.67 On the harder desert plain, A, the hunter seen here with the falcon uses B, enclosed shoes.

with walker-type removable braces allows rapid return to riding. Occasionally, more severe problems such as Lisfranc injuries and subluxations or dislocations of the talocrural joint occur. When surgical reconstruction is warranted, rigid internal fixation is used, possibly including the repair of a deltoid ligament avulsion and concomitant syndesmosis stabilization (Fig. 25.70).

Long-distance bike riding is now becoming popular in the UAE. A relatively common problem seen in these cyclists is Morton's neuroma. This happens despite appropriate footwear and possibly results from a combination of the high heat and humidity causing edema of the foot. Conservative care with metatarsal pads and a wider toe box to accommodate the forefoot is very successful.



Fig. 25.68 Another example of A, the falcon trainer with B, typical footwear used for C, hard desert terrain.

Tendinopathies in the UAE are now increasingly diagnosed by ultrasound and treated with biologics. Platelet-rich plasma, mostly leukocyte-poor PRP, is the most common medium employed. Success rates are similar to those in the published literature to date. Needle electrical stimulation (EIN or EPTE) is also used but less frequently. For Achilles tendon ruptures, the trend is toward functional brace treatment instead of surgery. The decision on the ground here is guided by dynamic diagnostic ultrasound, looking for approach of the torn tendon ends with passive ankle plantar flexion. In the UAE, we emphasize rapid rehabilitation and return to sports. Fast-track programs and hydrotherapy for management of foot and ankle injuries provide an early start to the recovery process, with weight bearing as soon as safely possible. Rapid progression to strengthening and proprioceptive feedback exercises has been beneficial to returning the player quickly to his or her sport.

PLANTAR PLATE RUPTURE AND DRUM-BEAT DANCE IN VENEZUELA

Drum-beat dance has become a very popular musical genre in Venezuela for the last 200 years. It is strongly related to veneration of saints Peter and John, and part of the African slaves' musical heritage. This dance is mainly from the country's central coast, and depending on the area, it may differ in dance

rituals, rhythm, songs, and instruments. There are usually seven different types of drums playing the beat.

To celebrate Saint John on June 24, parishioners perform their rituals and dance the drum-beat for 3 days in many coastal town central squares or beach areas, although the dance has become so popular that it is performed all year long and all around the country.

The dance is performed by a male and female couple surrounded by drummers, singers, and spectators in a circle who sing and cheer at the same time. The male dances around the female pretending to trap her with sudden movements, and the female pretends to ignore the male dancer turning her back to him while he tries to face her again; it can become very fast, a bit aggressive and sensual at the same time. The male is replaced after a short time by another male dancer who jumps suddenly between the couple; the female remains longer and can dance with many different males in the same song.

The main characteristic of the Venezuelan drum-beat dance is that the dancer's rear foot has to remain in a forced MTP joint dorsiflexion position for long periods, exerting pressure in this area when the body position suddenly changes during the dance, which is mostly performed barefoot in sandy areas. This rear foot stance makes the dancer susceptible to forefoot problems, mainly metatarsalgia, toes deformities, and acute or chronic plantar plate rupture (Fig. 25.71).

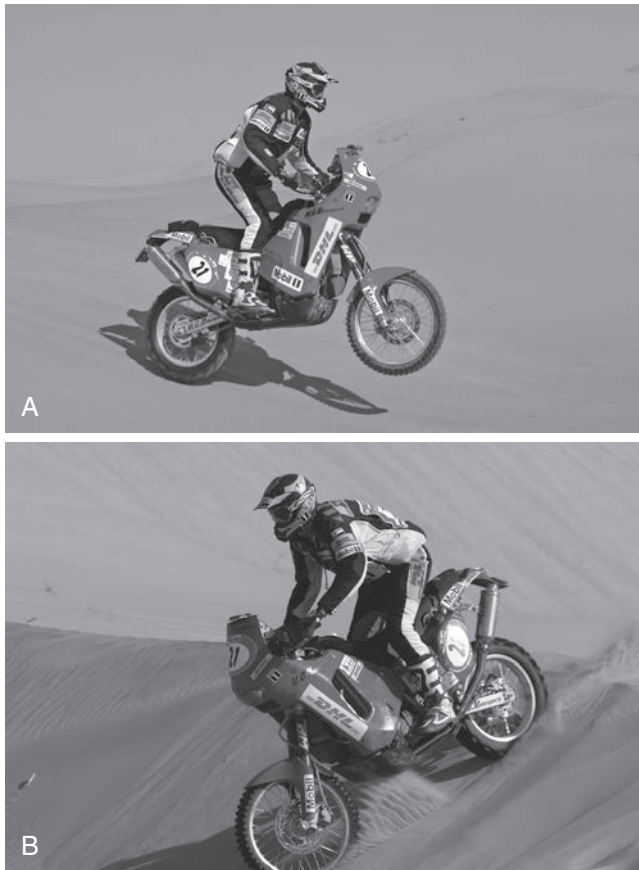


Fig. 25.69 A popular winter sport is motocross. The rider wears body armor and protective footwear to minimize injury. **A**, The rider here is preparing to land on a dune following a jump. **B**, The rider cuts across the soft and at times unstable sand and is susceptible to ankle and leg trauma.

Plantar Plate Rupture and Venezuelan Drum-Beat Dance

Metatarsophalangeal plantar plate rupture can occur in an acute or chronic setting.

Acute plantar plate rupture is an infrequent condition. The patient reports having felt a sudden crepitation and pain in the plantar plate area during the event, progressive second MTP



Fig. 25.71 There are mainly seven different types of drums playing the beat.

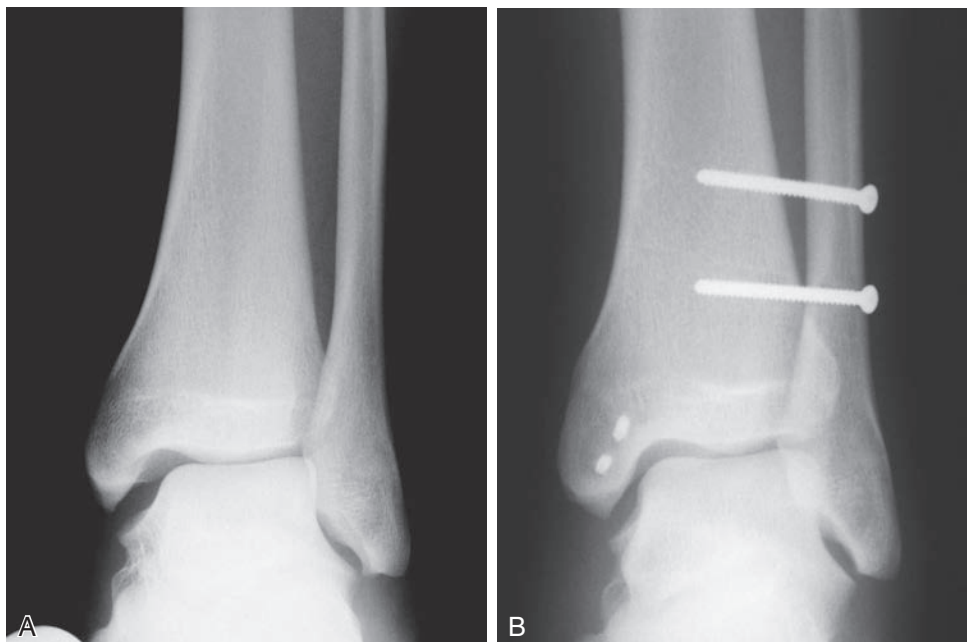


Fig. 25.70 **A**, This syndesmotomous injury was noted on a stress view of the ankle. **B**, Two syndesmotomous screws close the tibiofibular space and suture anchors stabilize the deltoid tear.

joint dorsiflexion days after the injury with swelling and even hematoma around the area.

Metatarsophalangeal instability and chronic plantar plate rupture can be present in drum-beat dancers, mainly in more frequent dancers. Most patients with this condition, refer plantar or dorsal gradual pain onset in the effected joint and progressive long-standing MTP dorsiflexion, with coronal or sagittal deviation.¹⁰⁵⁻¹⁰⁸ Depending on the clinical progression, patients may have positive drawer test or discomfort with the exam. Drum-beat dancers normally have clinical deformities of grades 0, I, or II and very rarely grades III or IV according to the Nery et al. staging system (Table 25.3).¹⁰⁹

Our Treatment Choice for Plantar Plate Rupture

Acute Plantar Plate Rupture

In acute plantar plate rupture we suggest repairing the plantar plate acutely, by a plantar incision.¹¹⁰ The MTP joint is approached directly after the flexor digitorum longus is retracted laterally (as shown in the image), and the plantar plate is reinserted to the base of the first phalanx with 2.0-mm suture anchors. Postoperatively the injured joint in held in slight plantar flexion by strapping for 2 weeks and active or passive joint dorsiflexion is avoided for the next 2 weeks, protecting the foot in a postop shoe for the first 4 postop weeks (Figs. 25.72 through 25.75).

Since these are normally acute injuries in balanced forefoot, there is no need to alter the forefoot biomechanics with metatarsal osteotomies or invade healthy joints to access and repair the acutely ruptured plantar plate.

We operated on seven patients with acute plantar plate rupture using the previously described technique, three were



Fig. 25.72 In acute plantar plate rupture, progressive second metatarsophalangeal joint dorsiflexion is observed days after the injury.

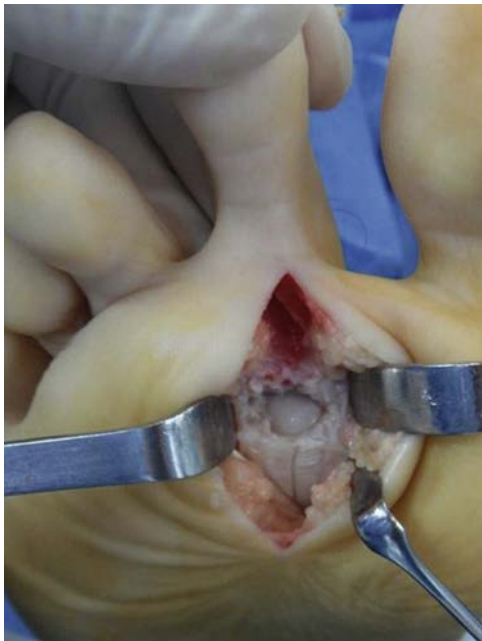


Fig. 25.73 In acute plantar plate rupture we perform the plate repair through a plantar incision, the metatarsophalangeal joint is approached directly after the flexor digitorum longus is retracted laterally.

TABLE 25.3 Clinical Staging System for Lesser Toes MTP Joints Instability		
Grade	Alignment	Physical Exam
0	Prodromal phase: no deformity MTP joint alignment	Thickening or swelling of the MTP joint. Reduction of the toe purchase Negative drawer test
I	Mild deformity: toe elevation + web space widening + medial deviation	MTP joint pain, swelling of the MTP joint. Loss of toe purchase Mild positive drawer less than 50% subluxable
II	Moderate deformity: toe elevation + medial or dorsomedial deviation	MTP joint pain, reduction of swelling. No toe purchase Moderate positive drawer: more than 50% subluxable
III	Severe deformity: toe elevation + medial deviation toe overlap. Flexible hammertoe	Joint and toe pain, little swelling. No toe purchase Severe positive drawer: dislocatable MTP joint
IV	Very severe deformity: dorsomedial or dorsal Cross over toe. Fixed hammertoe.	Joint and toe pain, little or no swelling. No toe purchase Dislocated MTP

From Nery C, Coughlin MJ, Baumfeld D, Mann T. Lesser metatarsophalangeal joint instability: prospective evaluation and repair of plantar plate and capsular insufficiency. *Foot Ankle Int.* 2012;33(4):301–311.



Fig. 25.74 Plantar plate is reinserted to the base of the first phalanx with 2.0-mm suture anchors.



Fig. 25.75 Plantar plate is refreshed and reinserted to the base of the first phalanx with 2.0-mm suture anchors.



Fig. 25.76 Plantar plate is reinserted to the base of the first phalanx with 2.0-mm suture anchors forcing slight plantar flexion.

recreational male drum-beat dancers after a prolonged dance round in sandy areas. The other four patients, two were injured after playing beach racquet, one sliding into second base while playing baseball, and the last one after a strong golf swing.

After surgery three patients reported slight discomfort and edema in the plantar plate area that lasted over 3 months and eventually disappeared. One patient had transient plantar neuritis for 2 months. No patient had any skin issues or complications from the plantar approach.

Instability and Chronic Plantar Plate Rupture

In clinical grade 0, with no deformity (or slight dorsiflexion) and moderate joint pain increased with drawer test, treatment consisted of strapping the MTP joint in slight plantar flexion for 4 to 6 weeks.¹¹¹ Twelve of 16 patients with grade 0 improved their symptoms and did not develop deformity after 1 year (4 patients were drum-beat dancers). The other four patients did not improve and toe dorsiflexion increased—they were treated as grade I.¹⁰⁷

Grade I patients presented with mild toe elevation and medial deviation, grade II patients have a moderate deformity with toe elevation and medial or dorsomedial deviation, both grades with discomfort and positive drawer test. We performed surgery in 24 patients grades I and II (five patients were drum-beat dancers). In 17 of 24 patients, surgery consisted of plantar plate reconstruction using a dorsal approach,¹¹²⁻¹¹⁴ Weil osteotomy and lateral capsule reefing or extensor digitorum brevis (EDB) transfer. In 8 of the 17 patients, Girdlestone Taylor flexor to extensor transfer was added to address flexible hammer toe.¹¹⁵ We found that 9 of



Fig. 25.77 Chronic plantar plate reconstruction is performed through a dorsal approach and Weil osteotomy.

the 17 patients had edema and discomfort in the plantar plate area that lasted over a year in most of the patients, and 7 of 17 patients ended with mild to moderate floating toes. In the other group, 7 of 24 patients, treated only with Girdlestone Taylor flexor to extensor transfer and lateral capsule reefing or EDB transfer, there were no plantar plate discomfort after 3 months and no floating toes (Figs. 25.76 through 25.78).

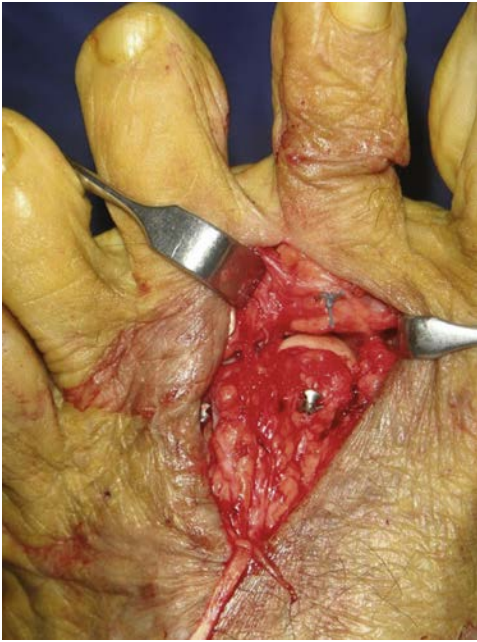


Fig. 25.78 After chronic plantar plate reconstruction is performed, Weil osteotomy is fixed in position.



Fig. 25.80 We consider plantar plate repair and Weil osteotomy in patients diagnosed of plantar plate rupture grade I or II, with long central metatarsals, second and third toes divergence, plantar hyperkeratosis, and metatarsalgia.



Fig. 25.79 Light to moderate floating toe is a common complication after Weil osteotomy.



Fig. 25.81 According to Nery's Clinical staging system for lesser toes metatarsophalangeal joints instability (Nery et al, FAJ 2012, vol 33-4), right foot is grade III and left foot grade II.

For these reasons, our treatment choice is the Girdlestone Taylor flexor to extensor transfer with lateral capsule reefing¹¹⁶ or EDB transfer to treat grade I and II plantar plate rupture. We consider performing the plantar plate repair and Weil osteotomy in patients diagnosed with plantar plate rupture grade I or II, with long central metatarsals, second and third toes divergence, plantar hyperkeratosis, and metatarsalgia (Figs. 25.79 and 25.80).

Our treatment choice for grade III and IV is similar, but without plantar plate reconstruction, since the tissue quality is not optimal to heal at this grade. We may also add in some of the patients a DuVries first proximal interphalangeal joint fusion for fixed hammer toes and EDB transfer (Figs. 25.81 and 25.82).



Fig. 25.82 In some patients Extensor Digitorum Brevis transfer is needed.

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The Military Athlete

Tobin Eckel Scott Shawen

OUTLINE

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INTRODUCTION

While the military generally represents a young, athletic population, it is distinct in many ways. Functional outcomes are different when compared to the civilian population, largely because the definition of success and the functional demands of the soldier are unique. To better understand this, one must first recognize what the military does. Simply stated, the purpose of the military is to protect the nation and win wars.¹ However, with a military that is spread across the world and has been at war for the past 16 years, the demands placed on soldiers, sailors, marines, and airmen are unparalleled in the civilian population. The US military is an all-volunteer force that only comprises 0.4% of the American population, yet we place great demands on these select few, which can inherently lead to increased physical strain and eventual musculoskeletal injury.²

The armed forces represent a highly active population with strenuous occupational demands. The routine physical fitness and rigorous combat training mirrors that of an athletic population.³ Musculoskeletal injuries are exceedingly common and often present as either traumatic in nature or the result of overuse injuries. Overuse injuries are seen most commonly during initial entry training into the military, as well as in the later stages of a military career. In fact, it is estimated that 25% of males and 50% of females sustain a physical training injury during basic training, with the vast majority being overuse injuries.⁴⁻⁶

Musculoskeletal injuries continue to place a huge burden on our military force. They are the leading cause of lost work time and disability leading to medical separation.⁷ The reason these injuries are so prevalent is multifactorial. There has been much speculation regarding the effect of the combat load that soldiers carry and its effect on overuse injuries. Over the past two decades, the average external load carried by a soldier has increased to roughly 100 pounds (Fig. 26.1). This is often carried in austere environments with uneven

terrain, and potentially while maneuvering under enemy fire. Biomechanical changes in gait patterns have been documented with these increased loads, to include increased step rate, decreased stride length, and forward trunk lean. With a concomitant rise in lower extremity overuse injuries, it is reasonable to infer a detrimental effect of these increased external loads.⁸ However, roughly 75% of musculoskeletal injuries can be attributed to physical training and sports-related activities.^{9,10} These nonbattle injuries remain the leading cause of evacuation from Iraq and Afghanistan.¹¹

The other unique aspect of the military athlete is that of functional outcome. Historically, the outcomes within military populations do not mirror those of the civilian population. In general, the military athlete is required to do more in less time. There are strict physical fitness requirements tested on an annual or biannual basis. Each of the services (Army, Air Force, Navy, Marines) has different requirements and frequency, all testing strength, endurance, and agility. The Air Force and Navy require a passing score in a 1.5-mile timed run, timed push-ups, and timed sit-ups. The Navy can select a 500-meter swim as a test of endurance instead of the 1.5-mile run. The Army requires a passing score in a 2-mile timed run, timed push-ups, and timed sit-ups. The most strenuous, the Marine Corps requires a passing score in a 3-mile timed run, timed crunches, and pull-ups/dead arm hang for males/females. The Marines also employ a combat fitness test (CFT) that evaluates simulated combat conditions with sprinting, lifting, and agility testing. Furthermore, soldiers in combat arms branches, as well as elite units and special forces operators from each of the units, have even greater physical demands and requirements. Most units require daily physical training, usually in small groups. If a service member is injured, they typically have a year to recover before the medical separation process begins.¹² Military outcomes focus predominantly on the ability to return to full duty in this finite period of time.¹²

The focus of this chapter will be injuries sustained about the foot and ankle of our military service members. This will include



Fig. 26.1 Example of the external load carried by a soldier.

the prevalence of these injuries and their outcomes. Ultimately, the goal will be to have a better understanding of the impact of these injuries on our military forces and how they differ from a young, active population.

ANKLE SPRAINS AND INSTABILITY

Ankle sprains are one of the most common athletic injuries and account for nearly half of all sport-related injuries.^{13,14} The incidence rates (IR) of ankle sprains in the general population range between 5–7 per 1000 person-years. Yet, the IR in the military is much higher. One study reported the IR of ankle sprains among service members at 34.95 per 1000 person-years, while a second study examining cadets at the United States Military Academy reported an IR of 58.4 per 1000 person-years.^{15,16}

These disparities are likely explained in part by the fact that the military represents an athletic population that has an increased exposure to at-risk activities. In fact, cadets with higher levels of fitness and those that participated in intercollegiate athletics were more likely to sustain an ankle sprain.¹⁶ While there is no correlation between length of service and an isolated ankle sprain, those with recurrent ankle sprains and instability tend to have shorter service times when compared to those who do not sustain an ankle sprain.¹⁵

While 95% of these service members are able to return to sports and physical training within 6 weeks of injury, nearly half will still have residual pain at 6 months postinjury. Furthermore, the average rehabilitative period following an ankle sprain is 40 days, creating a significant burden on force readiness.¹⁷ Even with appropriate functional rehabilitation, 10% to 30% will develop chronic ankle instability and possibly require surgery.³ Because of the prevalence of these ankle injuries, the Department of Defense recommends the use of a semirigid ankle brace when participating in high-risk physical

activity. However, outside of parachuting, there is little evidence to suggest a protective benefit of prophylactic bracing in the military population.¹⁰

Those who develop chronic instability refractory to physical therapy undergo a lateral ligament reconstruction with concomitant procedures performed as necessary. In our practice, we have found that the Broström-Gould-type ankle reconstruction (advancement of the ankle capsule and contained ligaments, augmenting with the inferior peroneal retinaculum) provides adequate stability without sacrificing sub-talar joint mobility and range of motion.^{18,19} In selected cases where there is inadequate soft tissue for reconstruction, or the patient has failed a prior Broström-Gould reconstruction, nonanatomic reconstructions are utilized. We favor the modified Broström-Evans described by Anderson for revision cases, as the ankle capsule and soft tissues can be repaired again and augmented with good outcomes.^{20,21} In cases where there is not sufficient ankle capsule or soft-tissue we recommend augmentation with an allograft tendon (semitendinosus) with a modified Chrisman-Snook.²² One study on the long-term outcomes of athletes undergoing ligament reconstruction demonstrated that 58% were able to return to their pre-injury level of sport, 16% were able to compete at a lower level, and the other 26% discontinued sport but were still able to remain physically active.²³

FRACTURES

Foot and ankle fractures are common in the military, second only to hand fractures. These present as either acute fractures or chronic stress fractures. Within the military, fractures account for 40% of injury-associated hospitalizations and 26% of combat injuries. Orr et al. demonstrated that 83% of service members undergoing operative fixation for an ankle fracture were able to remain on active duty.²⁴ However, at 3 years postsurgery, 36% were unable to return to the required level of running and 17%

were medically separated. This seems to coincide with civilian outcomes, which demonstrate roughly 50% of patients have pain, stiffness, and swelling at 1-year postsurgery. Additionally, only 25% of patients were able to return to sports at 1 year following surgery.²⁴

Stress fractures, on the other hand, continue to be one of the leading causes of injury in new military recruits.⁴ The incidence of lower extremity stress fracture for initial-entry military training is 0.8% to 6.9% for males and 3.4% to 21% for females.⁵ Prevention of overtraining, particularly in new recruits with lower baseline fitness levels, has proven effective in reducing the incidence of stress fractures. In fact, the military has decreased the number of stress fractures by 40% in the past 15 years by implementing modified physical training plans aimed at gradual progression of activity and appropriate cross-training to reduce repetitive stress.⁴ Nonetheless, stress fractures continue to result in significant morbidity and often require long recovery periods. Adequate nutrition is critical to ensure balance between energy intake and expenditure. Calcium and vitamin D supplementation can be both protective and therapeutic. Female Navy recruits placed on calcium and vitamin D therapy had a 20% lower incidence of stress fractures.²⁵

Metatarsal stress fractures account for 16% of all stress fractures. First described in Prussian soldiers in 1855, they were initially named march fractures.²⁵ Increased marching and running in minimalist footwear without a gradual transition are both risk factors for metatarsal stress fractures. The majority of the fractures can be managed with modified activity and weight bearing in a fracture boot. If pain persists, then a period of nonweight bearing may be necessary. The one exception is the fifth metatarsal stress fracture. Given the high nonunion rate, fifth metatarsal stress fractures are often treated with intramedullary screw fixation, with or without supplemental bone grafting.²⁵

ACHILLES TENDINOPATHY AND RUPTURE

Achilles tendinopathy in older military service members is consistent with this disorder in older, active civilian populations. However, there is a disproportionate incidence of tendinopathy in the military. Again, this may be due to the consequences of years of physical training and the toll of multiple deployments.²⁶ Another distinction is the similar incidence in men and women in the military, whereas in the general population, Achilles tendinopathy occurs more frequently in men. Both populations demonstrate a correlation between increasing body mass index (BMI) and Achilles tendinopathy.²⁶

Achilles tendon ruptures present similarly in both military members and civilians. Sporting activity accounts for nearly 70% of all ruptures, with basketball being the most common.²⁷ The increase in Achilles ruptures has been credited to the expanding number of older individuals participating in high-impact sports.²⁸ There is a higher incidence of Achilles rupture among African Americans in the military.²⁹⁻³¹ A biomechanical study comparing the viscoelastic properties of the gastrocnemius complex between black and white athletes

demonstrated a higher stiffness in the black athletes.³² While this difference can improve muscle performance, it may also result in a higher rate of catastrophic failure. Another possible explanation is the trend of higher BMI in black adults in the military compared to white adults, and the increased weight may predispose to tendinopathy and ultimately an increased risk of rupture.³⁰

Recent literature has demonstrated equivalent outcomes with operative and nonoperative treatment when using a functional rehabilitation protocol. This has led to a greater trend toward nonoperative management of Achilles ruptures.³³ However, within a military population, operative intervention may have the advantage of earlier return to duty. Renninger et al. demonstrated that service members undergoing operative management of their Achilles tendon rupture were able to return to duty 6 weeks faster than those managed nonoperatively.³³

LISFRANC INJURIES

Lisfranc injuries encompass a broad spectrum of midfoot injuries, ranging from isolated ligamentous injuries to fracture-dislocations. They can result from high-energy trauma, but low-energy, ligamentous injuries are becoming increasingly recognized in athletic populations.³⁴ Surgical intervention to achieve anatomic reduction is associated with improved outcomes; however, there is still considerable debate between open reduction and internal fixation (ORIF) versus primary arthrodesis (PA) for these injuries.¹² While similar outcomes have been reported with both techniques, there is a higher rate of secondary hardware removal and the development of posttraumatic arthritis in 20%–50% of patients undergoing ORIF, which may necessitate salvage arthrodesis (SA).³⁴

The civilian literature demonstrates favorable results with PA in an athletic population, with 75% being able to return to play at the same level in one study.³⁵ Similar results have been published in military studies as well. Hawkinson et al. demonstrated similar results in low-energy Lisfranc injuries treated with ORIF and PA, with just over two-thirds being able to return to full duty. More importantly, those who failed ORIF and required SA had worse outcomes, with only 29% able to return to full duty.³⁴ Evidence also suggests a more rapid and complete recovery with PA in a military population. Cochran et al. demonstrated that patients treated with PA completed their fitness run 9 seconds slower per mile than pre-injury time, compared to 39 seconds slower per mile for the ORIF group. Additionally, the PA group returned to full duty at an average of 4.5 months compared to 6.7 months in the ORIF group.³⁶

OSTEOCHONDRAL LESIONS OF THE TALUS

Osteochondral lesions of the talus (OLT) commonly affect athletic populations, although the incidence among active populations has not been fully elucidated. They are typically caused by repetitive microtrauma, as seen in chronic

ankle instability previously discussed. They can also result from an acute traumatic event, such as a severe ankle sprain or fracture.³⁷ Osteonecrosis, inflammatory and degenerative joint arthropathy, and genetic predisposition are other potential etiologic factors. Recent evidence suggests that these lesions are more common than previously expected. The incidence of OLT in patients undergoing diagnostic arthroscopy at the time of lateral ligament reconstruction is greater than 20%.³⁸

In a retrospective review of a large military database spanning a decade, Orr et al. demonstrated an OLT IR of 27 per 100,000 person-years.³⁹ Additionally, predictors of OLT included female gender, white race, junior and senior enlisted, increasing age, and service in the Army and Marine Corps.³⁹ Females in the military have demonstrated nearly double the incidence of ankle sprains compared to males, and this increased ligamentous laxity may account for the increase in OLT.¹⁶ The increase in junior enlisted, as well as Army and Marines, is likely a product of at-risk behaviors, as these populations constitute the bulk of our land-based combat force, operating on uneven terrain with heavy combat loads. This would seem to be substantiated by the annual increase in OLT from 2002–2008, corresponding to major combat operations in Iraq and Afghanistan.³⁹ Increasing age as a risk factor may represent physiologic cartilage degradation coupled with increased microtrauma from the physical demands of a military career. Nonetheless, these lesions are likely more common in both athletic and military populations than previously reported.⁴⁰

Our experience with OLT has varied depending on age of the patient at presentation as well as the acuity of the injury, location of the OLT, and size of the defect. In acute injuries, simple removal of the osteochondral fragment, if not repairable, is performed with stabilization of the cartilage edge at the defect. With later presentation, which is not uncommon, marrow stimulation techniques with stabilization of the cartilage rim are typically performed. In those with failed marrow stimulation, augmentation with either particulated juvenile cartilage (Zimmer Biomet, Warsaw, IN) or micronized cartilage (Arthrex, Naples, FL) have been utilized with varying success. Return to activity depends on the modality employed. With simple removal of the fragment, return to running and full activity depends mainly on other injuries or surgeries performed, such as ligament reconstruction or ankle fracture repair. For patients undergoing marrow stimulation, we recommend that they be immobilized for 2 weeks nonweight bearing followed by protected weight bearing in a CAM boot for an additional 4 weeks. Impact activity begins at the earliest at 6 months. For particulated juvenile or micronized cartilage grafts, we maintain nonweight bearing for 6 weeks followed by another 4 weeks of protected weight bearing in a CAM boot. Impact activity begins at the earliest at 9 months. In our experience, over 80% of patients return to activity without limitations. Regarding larger lesions requiring bulk allografts, our experience is similar to that of Orr et al.,⁴⁰ with no return to full military duty without restrictions (all remained on

limited-duty status and were not able to run or fully participate in all military requirements).

PLANTAR FASCIITIS

Plantar fasciitis is the most common cause of heel pain, affecting up to 10% of the population. It is considered an overuse or degenerative condition, and often presents in active adults. It is characterized by sharp pain with the first steps upon waking, or after periods of rest. Risk factors include prolonged weight-bearing, excessive running or walking, improper shoe wear, obesity, and equinus contracture.⁴¹ Plantar fasciitis is not just confined to the work place but also occurs frequently in running athletes.⁴²

Within the military, the IR of plantar fasciitis was reported at 10.5 per 1000 person-years. The incidence was greater in females, African Americans, and those over the age of 40. Additionally, Army service was a risk factor for developing plantar fasciitis when compared to the other branches of the armed services.⁴¹ Similarly, the incidence of plantar fasciitis increases significantly during military deployments. This would further support the overuse model, as service members are forced to walk on uneven terrain with heavy combat loads. Furthermore, it is common for soldiers to increase their normal running regimens while deployed, again placing further stress on the plantar fascia.²⁶ This can have a markedly negative impact on force readiness, as symptoms commonly persist for months.⁴²

Successful treatment of plantar fasciitis remains elusive. Many different modalities are possible, but none are universally successful. In our practice, we do not routinely provide injections with corticosteroid into the origin of the plantar fascia, as it has not proven to provide long-term relief,^{43,44} and may affect outcomes if surgical intervention is provided in the future.⁴⁵ Multiple other regimens have been recommended, but we have had the greatest success with Extracorporeal Shockwave Therapy.^{46,47} Our success mirrors that of others, with over 80% success rates in over 1200 patients treated.

BRACING OPTIONS

Recent advances in bracing technology combining the use of prosthetic as well as orthotic principles has led to the development of the Intrepid Dynamic Exoskeletal Orthosis (IDEO, US Army and Hanger Prosthetics, Austin, TX).^{48,49,50} This brace transfers forces around the ankle to the proximal tibia and flexes on impact, providing push-off power due to the carbon-fiber construct. It has been used extensively in the military to return patients to impact activity following a large number of foot and ankle injuries, from soft tissue injuries to pan-talar fusions. Its use outside of the military has been somewhat limited due to manufacturing expenses, but has become more prevalent with use of off-the-shelf components and modular construction. (Fig. 26.2)



Fig. 26.2 Ankle brace.

SUMMARY

The military represents a unique, physically active population with high occupational demands. Service members maintain required fitness levels through routine physical training and recreational sporting activities. Additionally, military service is a physically demanding profession, heightened by a shrinking force that has been at war for

nearly two decades. For these reasons, there will continue to be a significant number of overuse musculoskeletal injuries in our service men and women, with recovery adequate to resume full military duties uncertain.

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Pediatric Problems and Rehabilitation Geared to the Young Athlete

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INTRODUCTION

The number of young athletes participating in organized sports continues to expand in the United States, leading to an increasing number of both acute and overuse injuries of the foot and ankle in this age group. The recent trend toward early specialization in one sport poses additional risk for those children engaging in high-level activities. Foot and ankle pain in young athletes requires special consideration, as injuries often involve part of their immature skeleton such as the apophysis or the physis, and long-term function and growth must be considered. In this chapter we focus on the major foot and ankle problems most applicable to the health of younger athletes, including congenital problems, osteochondroses, fractures, and ankle sprains.

CONGENITAL PROBLEMS IN YOUNG ATHLETES

Coalitions

A *coalition* is the term used to describe the adjoining of two or more adjacent bones whose mesenchymal tissue fails to separate fully during normal segmentation in utero. For many people, this coalition may be asymptomatic, and they will never even be aware that they have it. For others, however, the coalition can lead to considerable pain and disability, and they may even require surgical resection with or without realignment—or, rarely, fusion.

Tarsal coalitions may be found in anywhere from 2% to 13% of the population, half of whom will have them bilaterally. They are believed to be heritable in an autosomal dominant pattern.^{1,2} Coalitions can be fibrous, cartilaginous, or osseous.³ They most

often become symptomatic during childhood, when the coalition starts to ossify.

The most common types of coalitions are calcaneonavicular coalitions (CNCs) and talocalcaneal coalitions (TCCs), which each make up about 45% of all coalitions, though the former may be slightly more common.⁴ Other, less common coalitions that have been observed include calcaneocuboid, cuneiform-metatarsal, naviculocuneiform, and talonavicular.⁵ Although the true gender distribution of coalitions is not known, given their overwhelming lack of symptoms, recent studies have indicated a possible male predominance, with men receiving 50% more surgeries for tarsal coalitions.⁶

Calcaneonavicular Coalitions

CNCs, the most common type, are usually congenital, although they may also arise iatrogenically, posttraumatically, or from other causes.⁴

CNCs present between ages 8 and 12, during which time the foot is observed to stiffen.⁷ Patients may present with refractory foot symptoms after an injury, or with frequently recurring ankle sprains.³ On exam they will likely have diffuse pain, though it may be localizable to the sinus tarsi. Spasming of the peroneal muscles along with a rigid everted foot, called “peroneal spastic flat foot,” may also be seen, though it is not highly specific for CNC.^{3,4}

Anterior-posterior (AP), lateral, and oblique radiography should all be obtained for a suspected CNC, but it will be best seen on an internal oblique view, with an osseous-type coalition being most visible. When viewed laterally, this osseous bridge

is called the “anteater nose sign” (Fig. 27.1). Other findings that suggest a CNC in the lateral view are dorsal beaking of the talar head and widening of the talar lateral process. Computed tomography (CT) is not necessary to diagnose CNC, but it has a much higher sensitivity than x-ray and can rule out an accompanying talocalcaneal coalition.⁵ MRI may also offer some utility for evaluating nonosseous CNCs.⁴ Occasionally the magnetic resonance imaging (MRI) will reveal a stress fracture of the calcaneus or navicular in association with the coalition.

First-line treatment of CNCs should be activity modification and orthoses for medial arch support, escalating to immobilization with 6 weeks of bracing or short-leg casting if needed. Given the inflammatory nature of the pain, nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen will also offer some relief. However, CNCs are more likely to be refractory to conservative treatment than are other types of coalitions.⁴ If surgery is required, resection of the coalition is usually successful, with up to 90% of adolescents treated having good outcomes.⁵

Talocalcaneal Coalitions

TCCs are the other most common type of tarsal coalition. They will present similarly to CNCs, with activity-related pain midfoot occurring at the age of 8 or older. Like CNCs, they may present after an ankle injury, are frequently bilateral, and should be suspected if a patient has recurring or persistently painful sprains.⁸ However, TCCs may present slightly later than CNCs, continuing to manifest beyond the age of 12 and into the mid-teens.⁹ On physical exam, the oversized medial talocalcaneal facet may be felt distal to the medial malleolus, and pain may localize to the sustentaculum tali.

Imaging of a suspected TCC will also include radiographs, but it is typically best seen on a Harris heel view, which is taken



Fig. 27.1 Lateral weight-bearing radiograph of the right ankle in a 9-year-old girl with a calcaneonavicular coalition, demonstrating the “anteater nose sign” (arrow). When her pain persisted after casting, she underwent excision with tendon interposition and had complete resolution of symptoms.

posteriorly at 45 degrees below horizontal, aiming down the calcaneus, and offers the clearest view of a TCC.⁹ On the lateral view, a TCC will often show a C-shaped line connecting the talar dome and sustentaculum tali, called the “C sign,” which is present in up to 83% of cases (Fig. 27.2); however, this sign is also present in nearly half of flexible flat feet without TCCs.¹⁰ CT will confirm the diagnosis and determine what portion of the subtalar joint is involved. An MRI will be helpful in cartilaginous or fibrous coalitions. Typically, excision is the first-line treatment, offering perhaps the best return to full activity, while arthrodesis is considered once more than 50% of the subtalar joint is involved.^{8,11–13} Occasionally the foot deformity that remains after coalition resection requires calcaneal or midfoot osteotomies for realignment.

Pes Planus

Pes planus (or flatfoot) is typically an asymptomatic deformity in which the medial longitudinal arch of the foot is noted to be low or absent altogether, with associated valgus of the hindfoot and forefoot abduction. The mechanism of flatfoot is thought to occur from either reduced strength of the longitudinal arch musculature or altered intrinsic structure of the bone-ligament complex.¹⁴ Flatfoot presents in two general types: (1) flexible flatfoot, which represents a normal anatomic variant and almost always responds to conservative management, and (2) rigid flatfoot, a less common and more painful condition that may require correctional osteotomy.¹⁴ Congenital calcaneovalgus and congenital vertical talus may also cause flat feet in children, but both of these conditions present long before children reach the age of sports participation.

Flatfoot has a much greater prevalence in children than in adults, as nearly 100% of infants lack a medial arch from the ages of 0–2.¹⁵ However, the condition decreases throughout development, with only 9.1% of children still having flat feet

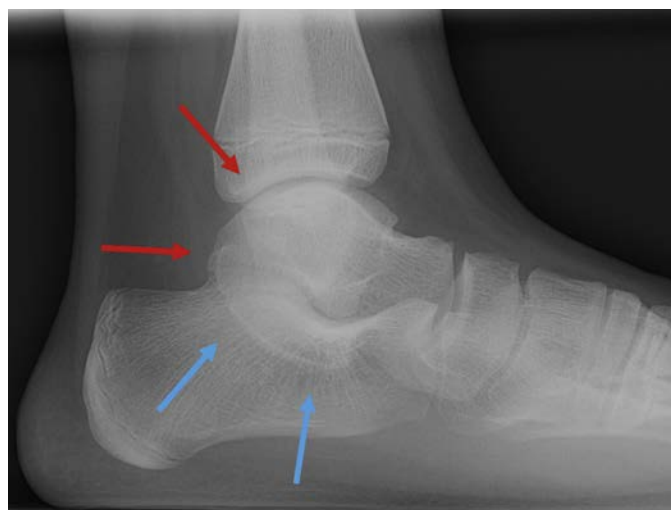


Fig. 27.2 Lateral weight-bearing radiograph of the left ankle in a 14-year-old male baseball player with hindfoot valgus and bilateral talocalcaneal coalitions, demonstrating the “C sign” formed by the posterior border of the sustentaculum tali (blue arrows) and the medial border of the talar dome (red arrows). Symptoms resolved with coalition resection with fat interposition and calcaneal slide osteotomy.

at age 7. The direct role of sports participation on flatfoot is unknown. Children with obesity have been found to have a threefold greater incidence of persistent flatfoot. Generalized joint hypermobility, which is frequently observed in dancers, gymnasts, and divers, has also been associated with flatfoot.¹⁶

A number of physical examination maneuvers can help to identify the specific type of flatfoot. First, when the child is sitting with the legs dangling or when the hallux is dorsiflexed, the flexible flatfoot should show restoration of a medial arch, whereas rigid flatfoot will look no different from how it does while weight-bearing. Second, a flexible flatfoot will have arch elevation and realignment of the hindfoot valgus when the patient stands on tiptoes, whereas a rigid flatfoot will remain flat and valgus. The Silfverskiöld test can also help to identify an Achilles contracture, which is more likely to cause pain in the flexible flatfoot. Passive dorsiflexion of <10 degrees with a neutral hindfoot in both knee flexion and extension suggests tightness of the entire Achilles tendon, whereas an increase in dorsiflexion with knee flexion suggests tightness of the gastrocnemius alone.¹⁴ Imaging is not indicated for the asymptomatic flexible flatfoot, but weight-bearing AP and lateral radiographs may help to distinguish symptomatic flatfoot from other causes of foot pain such as coalitions and accessory naviculars.

Treatment for flexible flatfoot should typically center on education and reassurance. The evidence for orthoses or shoe modifications in flatfoot is lacking, and it may actually exacerbate pain in a rigid flatfoot or tight Achilles tendon by increasing pressure on the medial midfoot.^{14,16} Thus, painless pes planus should merely be observed. However, athletes with persistent pain from flexible flat feet can start with over-the-counter orthotics prior to custom orthoses, for arch support. Flatfoot can also cause asymmetric wearing of the medial sole of the shoe, and so worn-out shoes should regularly be replaced in order to assure appropriate footwear. Athletes with flexible flatfoot and tight Achilles tendons may also benefit from regular heel cord stretching.¹⁴

Surgical treatment of symptomatic flatfoot should proceed only when significant pain and disability persists after prolonged conservative therapy. Osteotomies are the treatment of choice when indicated, as tendon transfers and lengthenings in isolation alone are rarely successful, and arthrodesis introduces undesirable complications.¹⁴ Calcaneal lengthening osteotomy corrects the eversion deformity and can be performed concurrently with Achilles lengthening, which may be informed by an intraoperative Silfverskiöld test. It has satisfaction rates over 90%.^{15,17} Alternately, the combination of calcaneo-cuboid-cuneiform osteotomy, via closing wedge osteotomies of the calcaneus and cuneiform and an opening wedge osteotomy of the cuboid, creates compensating deformities for the hindfoot valgus and has been shown to have comparable results.¹²

DEVELOPMENTAL PROBLEMS IN YOUNG ATHLETES

Hallux Valgus

The pediatric form of hallux valgus is distinguished from the adult type by the presence or absence of an open physis.

However, up to half of all adult bunions are believed to have begun during adolescence and not become symptomatic until later on.¹⁸

Hallux valgus typically presents with a prominence over the medial first metatarsal phalangeal joint, with pain being a secondary complaint. It is often bilateral. In children there is a strong female predominance, with a ratio of 4:1 and a known matrilineal component to the disease. Pediatric bunions tend to skew toward younger children, with about 50% presenting younger than 10.¹⁹

Examination of pediatric hallux valgus should include evaluation of other causes of foot pain, associated foot pathologies, and examination of footwear. A great toe deformity may be called a hallux valgus if the angle between the axes of the first metatarsal and proximal phalanx is 15 degrees or more in the AP view. Medial skin thickening or callus formation may also be seen over this area.⁵ Imaging should include weight-bearing AP and lateral radiographs and a nonweight-bearing (NWB) oblique radiograph. Hallux valgus angles much larger than 15 degrees may be seen, but the angle size does not always correlate with severity of symptoms, and a steep angle itself is not an indication for surgery.¹⁹ A common underlying mechanism is varus of the first tarsometatarsal joint or first metatarsal itself, which then leads to the observed metatarsophalangeal (MTP) protrusion. Metatarsus primus varus is defined as an intermetatarsal angle between the first and second rays of >8 degrees. Potential factors such as ligamentous laxity, pes planus, disproportionate mobility, or length of the first ray may predispose a child for metatarsus primus varus.¹⁹

Young athletes presenting with painful hallux valgus should first undergo conservative management. Tight-fitting shoes are often the cause of pain, and thus trialing wide box shoes that accommodate the shape of the foot can help to alleviate symptoms. Ibuprofen and activity modification may also provide relief. There is limited evidence for the use of orthotics or bracing, outside of one series that found foot exercises and nighttime splinting were effective in half of those treated and had a low recurrence rate.²⁰ The authors prefer conservative treatment with footwear modifications. Children, adolescents, and parents who are bothered by the appearance alone should be counseled that surgery for bunions has a high complication rate and is not recommended solely for cosmetic treatment. For those young athletes who do require operative intervention, it is recommended that intervention be postponed until skeletal maturity, as recurrent deformities are higher with earlier intervention.

Accessory Navicular

An accessory ossicle is a common finding that represents a normal variant of skeletal anatomy. Accessory ossicles, found in more than 20% of the population, are small secondary ossification centers that lie separate from adjacent bones and are typically smooth and well defined.²¹ They may occur throughout the body, but they are most associated with the foot, given their higher prevalence and the wide range of ossicles that may develop there. Most accessory bones have no clinical significance, but some, like an accessory navicular bone, may cause symptoms, particularly in a young athlete.

Accessory naviculars, the most common foot ossicles, may be found in 2%–14% of people.²² When present, they are found on the medial arch of the foot, posteromedial to the navicular tuberosity. An accessory navicular is observed to be more common in girls and is often bilateral. Most people who have them are unaware of it and unbothered by them, but an accessory navicular may become painful in late childhood or adolescence. This will present as pain over the medial mid-foot, with or without redness and swelling, that is worse with weight-bearing.²³

Evaluation of a suspected accessory navicular should include three-view radiographs, including an external oblique foot x-ray. However, providers must take care not to overdiagnose an accessory navicular as the source of an athlete's symptoms when they are seen on imaging, given their widespread prevalence and typical dormancy, and the many other diagnoses that may cause midfoot pain. Accessory naviculars are classified in three types. Type I is a small, ovoid sesamoid that is completely contained within the posterior tibialis and has no attachment to the true navicular. This type makes up 30% of accessory naviculars and is usually asymptomatic. Type II is larger and triangular and may be misinterpreted as a tuberosity fracture. It is the type most likely to cause pain. Type III is attached to the navicular by an osseous bar. Although thought to represent the end-result of Type II, it is also infrequently symptomatic. Type II and type III together make up 70% of all accessory naviculars.²³ Radiographic studies have shown that in children with accessory naviculars, the native navicular itself is also wider and more prominent.^{24–27}

Initial treatment of a symptomatic accessory navicular focuses on nonoperative management, such as activity modification, shoe modification, and use of orthoses for comfort. If these initial steps are not effective, a period of rest with a stirrup brace or boot brace should be helpful. If not, a below-knee cast may provide relief by preventing the regular traction of the posterior tibialis.²³ Surgery should be pursued only after failure of a long period of conservative treatment; however, recent evidence has shown that athletes are more likely to require surgery than nonathletes, and that for those who do progress to surgery, athletes (mean 16 years) tended to be younger than nonathletes (mean 24 years).²² Surgical treatment may involve simple excision with a tendon split (authors' preferred method) or excision with simultaneous tendon advancement (Kidner).²³ As with most rehab and recovery from lower extremity issues, continued conditioning of the upper body and core are encouraged in the young athlete. After surgical treatment, 6 weeks of toe-touch weight bearing in a short-leg cast with a well-molded arch is initiated for soft-tissue and tendon healing. Afterwards, the patient is transitioned to weight bearing in a CAM boot with orthotic inserts, and physical therapy is initiated for gait training and range of motion (ROM) exercises. At 10–12 weeks the CAM boot is weaned, while strengthening, gait training, and proprioception are initiated with physical therapy. Once strength and function in a sports-specific rehab are symmetric to the contralateral side, the young athlete is released to gradual, symptom-free participation in practice and unrestricted sports.

OSTEOCHONDROSES

Sever's Disease

Sever's disease is a traction calcaneal apophysitis that was first described by Dr. James Warren Sever as chronic heel pain in growing children. At the time, it was thought to be an inflammatory injury, but since then it has been clarified to be more so due to a combination of overuse microtrauma and traction injury to the posterior calcaneal apophysis.²⁸

Sever's disease is most common during or just before a child's peak growth velocity, which will typically occur from the ages of 8–15.²⁹ The true incidence of Sever's disease is not known, though it is believed to be a fairly common occurrence. It may often be bilateral, and it is observed to have a slight male predominance.^{30–32} It is commonly seen in young athletes who participate in sports involving high-energy plantarflexion and push-off, such as soccer. However, Sever's disease is not exclusive to active children and may actually be more symptomatic in heavier children.^{31,33} Children with Sever's disease will present with pain along the postero-inferior heel that is worse with activity, but they will not usually have associated redness or swelling.

In an active 8- to 15-year-old, subacute or chronic pain of the hindfoot with activity or palpation, in the absence of recent injury or other exam findings, strongly suggests the diagnosis of Sever's disease. There will be pain over the calcaneal apophysis, which may be exacerbated by stretching of the Achilles. Achilles contracture can also be associated with Sever's. Radiographs are not regularly required to make the diagnosis, unless the diagnosis is in question. A recent study has shown that perhaps 5% of patients diagnosed with Sever's disease may have other radiographic findings—such as a calcaneal unicameral bone cyst, calcaneal stress fracture, or tibial nonossifying fibroma—that x-ray may help to exclude.³⁰ In the lateral view, diagnosis of Sever's disease has been strongly associated with an apophysis that has not yet extended to within 2 mm of the plantar edge; if complete coverage or fusion of the plantar surface is seen, other diagnoses should be considered.³⁴

Sever's is a benign, self-limiting disorder that should resolve with apophyseal fusion at the latest. The largest impact of Sever's disease is on a young athlete's quality of life, as children with Sever's report having lower levels of physical function, happiness, and satisfaction.³² There are isolated reports of Sever's disease resulting in avulsion fracture of the calcaneal apophysis, but such occurrences are exceedingly rare and should not concern providers and patients.³⁵

Treatment of Sever's disease centers on alleviating its symptoms. Activity modification should be considered. Heel-cup orthoses and medial arch supports relieve pain better than heel lifts for those participating in sports.³³ If there is associated tightness of the Achilles, stretching would be appropriate. If pain is severe, immobilization in a short-leg cast, or a CAM boot with a removable heel lift, can be tried. With time and conservative treatment, the “growing pains” felt in Sever's disease will eventually abate, and young athletes should anticipate full return to their prior levels of activity.

Iselin's Disease

Iselin's disease, a traction apophysitis of the tuberosity of the proximal fifth metatarsal, was described by Dr. Hans Iselin. Like Sever's disease, it is a benign osteochondrosis that causes overuse-related foot pain in adolescents through repetitive microtraumas.

The overall incidence of Iselin's disease is not known, likely because of both scarceness and underreporting. It classically presents in girls aged 8–11 and boys aged 11–14, who will present with chronic or subacute pain over the lateral midfoot, with or without mild swelling, which is worse with weight bearing. Pain can also start acutely after a foot inversion injury. Iselin's occurs because of the traction placed on the fifth metatarsal by the peroneus brevis with active eversion or passive inversion of the foot.³⁶

As in Sever's disease, patients with suspected Iselin's disease may benefit from radiography to rule out other causes. On AP view, providers may see widening and fragmenting at the base of the fifth metatarsal, though absence of these does not rule out the diagnosis. Also, given the spectrum of radiographic appearances, the acute presentation of Iselin's is difficult to differentiate from an avulsion at the base of the fifth metatarsal. In its radiographic findings, Iselin's has more in common with Osgood-Schlatter than with Sever's, as the deformity is visible by imaging and the tuberosity can remain enlarged after skeletal maturation.

Iselin's disease is almost always self-limiting and resolves with conservative management, such as shoe inserts and activity modification.³⁷ There are rare reports of Iselin's avulsion.³⁶ With more severe symptoms, or if there is suspicion of a concomitant avulsion, immobilization in a CAM boot for 4 weeks is appropriate. As with all osteochondroses, however, no single blueprint works for every patient, and so the provider should consider the athlete's specific circumstances.

Kohler's Disease

Kohler's disease is an osteochondrosis of the navicular that was described by Dr. Alban Kohler in 1908. It is an infrequent, self-limiting condition that presents as midfoot pain with deformation or sclerosis of the navicular. The proposed pathophysiology of the disease is an idiopathic osteonecrosis of childhood, similar to a diagnosis such as Legg-Calve-Perthes disease.³⁸ It may occur unilaterally or bilaterally and has a male predominance. A genetic component to the disease has been suggested.³⁹

Kohler's presents slightly younger than the previously discussed osteochondroses, being most common in boys from the ages of 2 to 9. Children with Kohler's have pain over the midfoot and tend to walk on the outsides of affected feet in order to avoid putting increased pressure on the navicular, though their ROM is not affected. Erythema and swelling may or may not be seen over the area.⁴⁰

Although Kohler's is a clinical diagnosis, radiography can confirm it by showing narrowing or flattening of the navicular bone. This will also help to distinguish Kohler's from a navicular stress fracture. Radiography alone cannot make the diagnosis, as flattened naviculars may be incidentally observed in asymptomatic children. Imaging of both feet may be appropriate, either to demonstrate bilaterality of the disease or to provide a normal foot radiograph for comparison. More advanced imaging is not needed. Infection in this area is uncommon, but

complete blood cell count (CBC) and C-reactive protein (CRP) tests may be obtained if needed to help rule it out.⁵

Kohler's has a favorable prognosis and should eventually resolve with ice, rest, and NSAID medications. In more severe cases, casting may be used. Although a history of Kohler's might still be discernable by radiography in adults who had the disease as children, this finding is typically asymptomatic.

Freiberg's Disease

Freiberg's disease is osteochondrosis of the metatarsal head. It was described by Dr. Albert Freiberg and, like Kohler's disease, is believed to occur secondary to osteonecrosis, although mechanical and systematic mechanisms have also been proposed.⁴¹ It classically refers to deformation of the second metatarsal, but involvement of the third and other metatarsals has also been reported.⁴² Freiberg originally called it an "infracion" because of his theory (now tenuous) that the disease occurs as a result of trauma.

Among osteochondroses, Freiberg's is unique in that it is the only one known to be more common in girls, with a 5:1 female predominance.⁴³ Freiberg's also onsets slightly later than other foot and ankle osteochondroses, with a typical age range of 11–17 years old.⁴² It is typically not bilateral.

The most-used classification system for Freiberg's disease is the radiographic classification proposed by Smillie. Stage I shows the development of a narrow fissure and subchondral sclerosis after the epiphysis becomes ischemic. Stage II sees collapse of the lesion with bony resorption and subchondral bone collapse. In Stage III, the collapse progresses and the lateral portions of the epiphysis project above the lesion on either side. Stage IV is marked by loose bodies and an epiphysis that has likely closed. Finally, Stage V will show a flattened top edge, a thickened and dense metatarsal shaft, and resorption of the loose bodies.⁴⁴

Children with Freiberg's disease will present with a tender and swollen MTP joint in the affected foot, with or without a preceding trauma. Ambulation that increases pressure on the joint, such as when using a heel lift, high-heeled shoes, or barefoot, tends to aggravate the pain. In later stages, the disease may resemble a crossed-over or clawed toe.⁴⁵

Workup should include AP, lateral, and oblique radiographs of the foot while weight bearing. Bilateral images are unlikely to identify disease in the opposite foot but may be useful for comparison. In early stages, x-ray may be inconclusive, showing only a subtle widening of the joint space or sclerosis of the metatarsal head. As it progresses, subchondral sclerosis may be more evident, followed by lucency and collapse.

For Freiberg's disease judged to be stages I through III, conservative treatment may be possible. This management should include NSAIDs such as ibuprofen, activity modification to reduce running and jumping, and shoe modification focused on reducing pressure on the affected metatarsal, such as with stiff shoes, custom orthotics, or casting.⁴⁶ Some authors believe that nonoperative management should be the first line of treatment for Freiberg's regardless of Smillie stage.⁴⁵

Surgery is necessary for patients who fail conservative treatment and/or present at later stages of the disease. Multiple surgical techniques may be undertaken, including both joint-sparing (for stages I–III) and joint-reconstructing (for stages IV and V) approaches.⁴⁵

Osteochondral Lesions of the Talus

Osteochondral lesions of the talus, i.e., OCDs, are an important cause of joint pain in adolescent athletes. They are believed to occur from a combination of repetitive microtrauma of the articular surface, which may lead to local ischemia, and a component of vascular insufficiency.

The talus, with its highly cartilaginous surface and retrograde blood supply, is the bone in the foot most commonly affected by OCD. Talar OCD most commonly develops over the talar dome, although it has been observed in case reports to occur in the talar head, as well.⁴⁷ The total incidence of ankle OCD is low, at only about 6 per 100,000 children aged 6–19, but it is seven times more common in teenagers than in younger children. It is most likely to occur in the medial talus (72% of lesions), followed by the lateral (22%) and middle talus (4%) (Fig 27.3). Race and gender have both been postulated to be independent risk factors for ankle OCD, with girls having 1.5 times as many cases as boys, and with the disease being most common among non-Hispanic whites and least common among African Americans. The largest epidemiological survey of OCD to date has even suggested that, in girls, ankle OCD may be more common than knee OCD.⁴⁸

Ankle OCD usually does not have a history of recent trauma, but may present after a history of recurrent sprains. Once symptomatic, OCD will present in young athletes who complain of sporadic ankle pain with joint-loading activities. Symptoms will escalate when a fragment detaches from the osteochondral surface, leading to severe pain, swelling, instability, and possible catching of the joint.⁴⁹

Diagnostic workup of ankle OCD should begin with radiographs, which may miss early stage lesions. Mortise views will provide the best view of the lateral talar dome, while

plantarflexion radiographs will bring the posterior talar dome into view. Advanced imaging is useful for characterization and for surgical planning. An MRI best visualizes early lesions and is also best for determining the stability of lesions. Detached osteochondral fragments will have a hyperintense outline in T2-weighted images on MRI. CT offers better fidelity for evaluating depth of lesions, but this advantage must be weighed against the risk of additional radiation.⁴⁹

Talar OCDs that are diagnosed early and are found to be stable or only have mild signs of instability are treated conservatively with immobilization and activity modification in children. Lesions that fail a course of 6 months of conservative treatment or are unstable would benefit from surgical intervention. As with adult OCD, retrograde and transarticular drilling, excision and marrow stimulation, or osteochondral grafting are all options.⁵⁰ For those that have not yet reached skeletal maturity, lesions are often still accessible through open soft-tissue approaches, in place of medial malleolar osteotomies. The reoperation rates for surgery in young athletes can be high, with female sex and high body mass index (BMI) being independent risk factors for poorer outcomes.⁵¹ In a long-term follow-up study of debridement and bone marrow stimulation for ankle OCD, 76% of patients were able to return to sports, though no athletes reported returning to their pre-injury level of function.⁵²

ANKLE FRACTURES

Distal Tibial Fractures

Ankle fractures are the second most common fracture in children, representing 5% of all pediatric fractures. In the skeletally immature athlete, the physes of the ankle require special attention. On one hand, open physes and significant growth potential

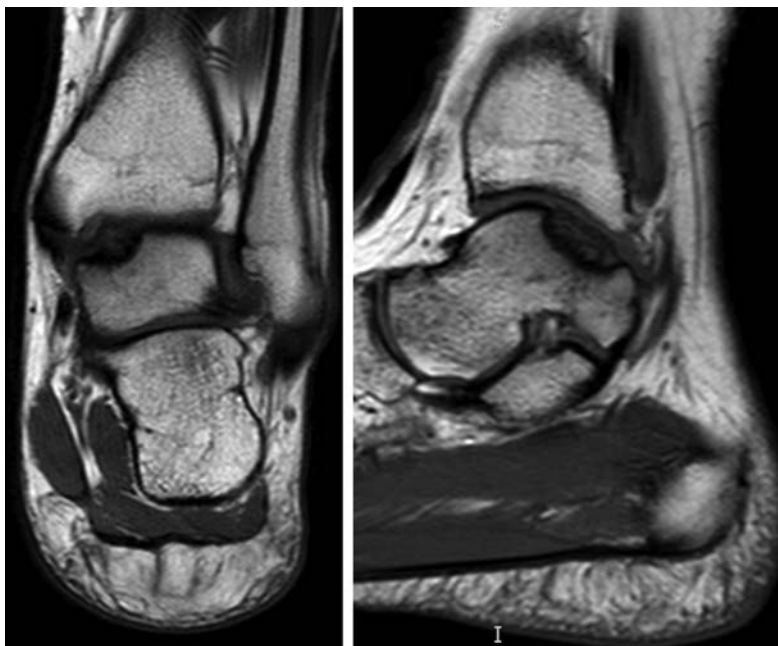


Fig. 27.3 Sagittal and coronal T1-weighted MRI of the left ankle in a 14-year-old female soccer and field hockey player. She had a history of bilateral pes planus and frequent ankle sprains and was subsequently found to have osteochondral lesions over the superomedial talar dome in both ankles, as well as vitamin D deficiency.

allows for fracture remodeling. On the other hand, injury to the physes can also put children at risk for growth arrest and subsequent leg length discrepancy or angular deformity, both complications that are best treated when recognized early.

The distal tibial ossification center appears around 6 months of age and grows an average of 4.5 mm per year axially during preadolescence, contributing about 18% of the lower extremity's ultimate length.² The distal tibial physis closes first centrally, then medially, and finally laterally, a sequence that influences the unique fracture patterns that appear closer to skeletal maturity. The distal tibial physis closes around age 14 in girls and age 16 in boys. Distinguishing normal developmental anatomy from a fracture fragment is an important part of assessing pediatric sports injuries. Secondary ossification centers of the medial malleolus are seen in up to 15% of girls aged 6–9 and boys aged 8–11.⁵³ Clinical exam is important to distinguish these from medial malleolus avulsions.

The Salter-Harris classification system, introduced in 1963, is the most widely used method to evaluate physeal injuries. The numbering of the classification is thought to be predictive of the risk of growth disturbance; however, this tends to be less true around the ankle, where the physeal anatomy is more complex and compressive forces may influence growth. Routine follow-up after fracture healing, with radiographs at 6 or 12 months, should be considered to monitor for growth arrest.

A Salter-Harris I fracture is a fracture straight through the physis, while II is a physeal fracture with a metaphyseal component, and III is a physeal fracture with an epiphyseal component. Salter-Harris IV fractures involve both a metaphyseal and epiphyseal component. Salter-Harris V fractures are a crush injury to the physis. Mechanisms that may lead to Salter-Harris fracture of the distal tibia include twisting on the foot during play, or sports that involve high-velocity jumping or pushing off.

Salter-Harris I tibial fractures are not especially common. While displaced fractures are evident on initial radiographs, nondisplaced fractures may be diagnosed by careful clinical exam and palpation. If a nondisplaced fracture is suspected, follow-up radiographs 7–10 days after injury may confirm the diagnosis, showing periosteal reaction or early callous formation around the physis. Displaced Salter-Harris I fractures should be promptly treated with manual reduction followed by 4–6 weeks of casting. With a closed reduction that results in an unstable reduction or a large persistent gap, open reduction would be indicated to remove any entrapped tendon or periosteal tissue.⁵⁴ However, a closed reduction that is nonanatomic but stable, with minimal coronal and sagittal plane displacement and minimal fracture gapping, is acceptable.

Salter-Harris II tibial fractures are the most common distal tibial physeal fracture, comprising 40% of such fractures. These fractures extend through both the physis and metaphysis of the tibia. Their presentation and treatment are similar to Salter-Harris I fractures, with radiographs helping to make the distinction. The characteristic metaphyseal fragment has a typical triangular shape, often called a Thurstan-Holland fragment. Again, nondisplaced fractures may require a follow-up radiograph to confirm the diagnosis. Physeal fractures that are displaced and present more than 7 days from injury should not be

reduced, in order to avoid a second physeal injury and further risk of physeal arrest.⁵⁴ As with Salter-Harris I fractures, these fractures should be reduced, with minimal coronal and sagittal plane displacement and minimal fracture gapping, and casted for 4–6 weeks. Open reduction is considered for any widely displaced, gapped, or unstable fracture with interposed soft tissue.

Salter-Harris III fractures make up 25% of distal tibial fractures and involve a fracture through the physis and epiphysis. As these fractures typically extend to the articular surface, they have greater potential long-term consequences, since inadequate or delayed correction of the articular incongruity may lead to early arthritis. Although x-ray is the first-line imaging modality, for displaced fractures that potentially would require surgical intervention a CT or MRI is useful to determine amount of displacement, to identify the fracture pattern, and to begin surgical planning. Anatomically reduced and nondisplaced fractures may be treated in a NWB cast for 4–6 weeks, with early radiographic follow-up between 1 and 2 weeks to monitor for loss of reduction. Fractures with more than 2 mm of persistent articular-side incongruity should be treated surgically. Once surgical anatomic reduction is achieved, epiphyseal screws may be used to avoid further physeal injury. However, smooth Kirschner wires crossing the physis can also be used for temporary fixation if it is required by the fracture pattern. Tillaux fractures are a unique type of Salter-Harris III.

Salter-Harris IV fractures account for another 25% of distal tibial physeal fractures and include disruption of the physis, epiphysis, and metaphysis. They may occur either along the medial malleolus or in a special pattern along the lateral physis called a triplane fracture. As with Salter-Harris III fractures, the most important aspect of fracture treatment is an anatomic articular-side reduction. When nondisplaced, Salter-Harris IV distal tibial fractures may also be treated with 4–6 weeks of NWB immobilization. However, more than 2 mm of displacement must be surgically reduced. Fixation with metaphyseal screws, epiphyseal screws, or a combination may be used to avoid further physeal injury. Again, smooth Kirschner wires crossing the physis are useful for temporary fixation if needed.

Salter-Harris V of the distal tibia is rare, representing about 1% of distal tibial physeal fractures, and involves a compression injury to the physis. An adolescent athlete who landed on his or her foot after falling from a height, or otherwise sustained a large axial load to the tibia, may be at risk for this. Salter-Harris V is likely to have little or no displacement, and it may thus be easy to miss by x-ray, but it also poses the greatest risk of growth arrest and poor prognosis, given the subsequent interruption of the physeal matrix and vascular supply. MRI will help to make the initial diagnosis, though these injuries often go months or years before being recognized. In the early period, focal areas of growth arrest may be treated with surgical resection of the physeal bar and placement of a spacer. If the majority of the physis is involved, the patient may benefit from epiphysiodesis of the bilateral distal tibia to prevent future limb length discrepancy.

Leg length deformity and angular deformity are the foremost complications to be avoided in pediatric physeal fractures. Clinically, up to 1 cm of leg length discrepancy, 5 degrees of varus/valgus deformity in the coronal plane, or 10 degrees of

flexion/extension in the sagittal plane are likely to be asymptomatic and considered acceptable. If the deformity exceeds these thresholds, then corrective surgery, such as physeal bar resection, guided growth through hemiepiphysiodesis, epiphysiodesis, or limb-lengthening surgery, may prove necessary.⁵⁵ Compartment syndrome of the anterior tibia has also been observed both after the fracture and after surgery. Pediatric compartment syndrome may be challenging to diagnose, and thus vigilance is needed.

Tillaux Fractures

Tillaux fractures are a subgroup of Salter-Harris III fractures. They are transitional fractures that occur during the active closure of the distal tibial growth plate and thus are unique to adolescents. They constitute 3%–5% of all pediatric ankle fractures.⁵⁶

The shaft of the distal tibia fuses with the distal tibial epiphysis over a period of about 18 months. This closure begins in the center of the physis, followed by fusion at the anteromedial, posteromedial, and finally lateral border of the physis.⁵⁴ This usually lasts from about age 12–14 in girls and 14–16 in boys. During this period, the unfused portions of the physis remain vulnerable to injury, particularly on the lateral side.

Tillaux fractures occur by avulsion of the unfused antero-lateral quadrant of the tibial epiphysis from the already fused medial and central tibial epiphysis. This happens due to traction from the anterior inferior tibiofibular ligament. Forceful external rotation with supination, such as twisting on a planted foot, is the usual mechanism.

Radiographs are the best first-line imaging for suspected Tillaux fractures, with the fracture lines best seen on mortise view. However, if the provider is concerned that there may be more than 2 mm of displacement, a CT can be considered for confirmation, evaluation, and surgical planning.⁵⁴

Nondisplaced Tillaux fractures may be treated with 4–6 weeks in a NWB cast, followed by transition to a CAM boot. Close follow-up is recommended for the first several weeks to ensure maintenance of alignment, as insufficiently reduced Tillaux fractures may lead to early ankle arthritis.⁵⁷ Tillaux fractures with more than 2 mm of displacement should be treated with reduction and internal fixation.⁵⁸ These fractures are less likely to result in growth deformity, since they occur near the end of vertical tibial growth.⁵⁴

Triplane Fractures

Triplane fractures, like Tillaux fractures, are a subgroup of distal tibial fractures that occur during the transitional phase. However, because they occur both above and below the partially fused growth plate, which may be at varying stages of fusion, they fit less neatly into the Salter-Harris classification system. At minimum, triplane fractures consist of Salter-Harris III fracture of the lateral tibial epiphysis (i.e., a Tillaux fragment) accompanied by Salter-Harris II fracture of the posterior tibial metaphysis, making them most like a Salter-Harris IV.⁵⁹ They make up 5%–15% of all pediatric ankle fractures.⁵⁶

Triplane fractures are so called because the fracture line consists of three distinct planes: a sagittal plane through the

epiphysis, an axial plane through the physis, and an oblique coronal plane through the metaphysis (Fig. 27.4). The mechanism of triplane fracture is similar to a Tillaux, involving traction on the epiphysis during forceful external rotation of a supinated foot.⁵⁴

Triplane fractures have multiple types and may present with anywhere from two to four fragments.⁶⁰ In a 2004 study of 51 triplane fractures by Brown et al. (the largest such series), the median age was 12 for girls (range 10–15) and 14 for boys (range 12–16), with older children significantly likelier to have epiphyseal separation.⁶¹ As in Tillaux fractures, radiographs are needed to make the diagnosis, but CT is useful to determine amount of displacement and to guide surgical planning.

Conservative treatment, consisting of 4–6 weeks of casting and a transition to a CAM boot, may be pursued if the patient has <2 mm of displacement. For fractures with >2 mm of displacement, open reduction with fixation by internal screws is the preferred treatment, again with little growth arrest given the relative skeletal maturity.⁵⁴

Distal Fibular Fractures

The pediatric fibula develops in parallel with the tibia, and so many of the same approaches used for the tibia—such as workup and Salter-Harris classification—may also be applied to the fibula. Due to the fibula's smaller size and structural load, pediatric fibular fractures have less of an effect on the child's ultimate limb length and mechanical axis. However, the fibula may also be more easily fractured, and thus it should not be overlooked in the young athlete.

The distal fibular ossification center appears slightly later than the tibia, at roughly 9–12 months of age, and it also closes slightly later, around about 1–2 years after the distal tibia fuses.^{2,62} Like the tibia, the distal fibula may also develop a secondary ossification center, which is more likely to be medial than lateral and must be distinguished from an avulsion fracture.⁵⁵ Mid-shaft fibular fractures may accompany a tibial physeal fracture, but the pediatric fibular shaft tends not to require anatomic reduction and should mend satisfactorily with reduction fixation of the tibia.

Isolated fractures of the distal fibular physis are typically Salter-Harris I or II. They may occur from a lateral blow to the athlete's ankle, such as from another player's foot, or from an inversion injury that might cause only a sprain in an adult.⁵⁵ Salter-Harris I and II fractures of the distal fibula can typically be treated with 4 weeks in a short-leg, weight-bearing cast or CAM boot. Recent evidence has suggested that these injuries may be overdiagnosed, as a series of MRIs on 18 diagnosed Salter-Harris I fractures of the distal fibula ultimately revealed 14 sprains, 11 contusions, and no growth plate injuries.⁶² Salter-Harris III and IV injuries of the distal fibula are uncommon but should respond well to 4 weeks of weight-bearing immobilization.

Young athletes are at risk for early arthritis with malunion or growth arrest associated with fibular fractures. Over time, shortening or displacement of the fibula may cause disruption of the tibiofibular syndesmosis and alter forces about the ankle. Shortening of the fibula will best be observed by a radiograph

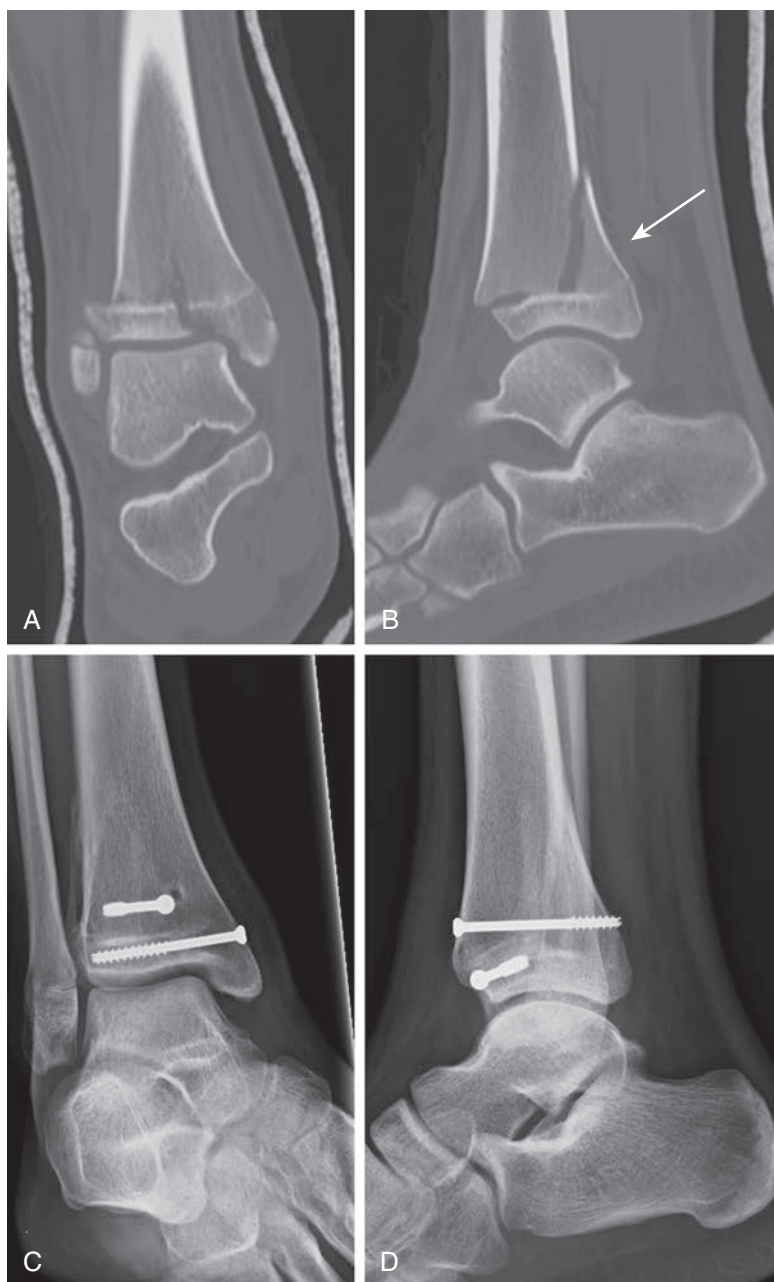


Fig. 27.4 (A) Coronal and (B) sagittal CT of a 14-year-old boy who sustained a triplane fracture of the right ankle after falling off his bicycle. The unique shape of the triplane fracture's Thurstan-Holland fragment (white arrow) is discernable in the sagittal view. (C) Oblique and (D) lateral radiographs of the same patient 3 months later, after open reduction and internal fixation with two screws.

taken on the mortise view, whereas absolute displacement is best observed on a lateral radiograph.⁶³ These malunions are unlikely to be symptomatic while the athlete is young, but they may become symptomatic in mid-adulthood.

FOOT FRACTURES

Foot fractures are an important consideration in children, given that they can make up anywhere from 5% to 13% of all pediatric fractures.^{64,65} In contrast to long-bone injuries, pediatric foot fractures are less likely to result in clinically significant length discrepancies from growth disturbance. A young athlete's

remaining growth potential and remodeling may make most foot fractures amenable to nonoperative treatment.

Metatarsal and Jones Fractures

Metatarsal fractures are a common pediatric fracture, representing nearly two-thirds of pediatric foot fractures and up to 5% of all pediatric fractures of any type.^{66,67} The first metatarsal is the most commonly fractured metatarsal in children 5 and younger, whereas the fifth metatarsal is the most commonly fractured metatarsal in children 6 and older. Both these are likeliest to occur as isolated fractures, whereas fracture of the second, third, and fourth metatarsals tend to occur in groups and

should encourage providers to screen carefully for other associated fractures. In older children, which encompasses most of those old enough to participate in sports, fall from standing height is the most common mechanism.⁶⁸

Fractures of the fifth metatarsal typically appear as avulsion fractures of the base after a foot inversion injury. These are treated with short-leg walking cast or CAM boot for 4–6 weeks, though NWB casts providing better relief than walking casts.⁶⁹ A nondisplaced or minimally displaced fracture of the fifth metatarsal is often difficult to differentiate from an apophysitis or Iselin's disease. A repeat radiograph 10 days after injury may demonstrate a periosteal reaction with callous formation and confirm the diagnosis of fracture. If the diagnosis is unclear, another approach would be simply placing the patient in a CAM boot that can be weaned as tolerated. Young athletes with these injuries should return to prior levels of play after treatment.⁶⁹

Pediatric Jones fractures, fractures of the proximal diaphyseal-metaphyseal junction, are a special concern, as in adults. They are more frequently seen in athletes, and they are a higher-risk fracture type because they lie at a watershed area of the vascular supply and thus are more predisposed to nonunion, particularly in children older than 13.⁶⁹ Most pediatric patients heal and recover well from this fracture, with a NWB boot brace or short-leg cast. Operative treatment is indicated for those that fail cast treatment and for adolescent patients, in order to facilitate a speedy return to sports. As with adults, surgical correction involves fixation with an intramedullary screw.⁷⁰

Calcaneal Fractures

Calcaneal fractures in children are relatively rare. The usual mechanism is landing on the heel after falling from a height. These fractures may be intra-articular or extra-articular, with CT supplementing standard radiographs, as needed, to better assess the location and extent of the injury. Most nondisplaced extra-articular fractures and minimally displaced intra-articular fractures in the younger child are amenable to casting for 4–6 weeks. Grossly displaced extra- or intra-articular calcaneal fractures can be treated with reduction and internal fixation, similar to adults. Evidence has shown that closed reduction with percutaneous fixation of pediatric intra-articular calcaneal fractures may offer similar functional outcomes while resulting in a lower complication rate.⁷¹

ANKLE SPRAINS

Ankle sprains are common injuries in young athletes. Traditionally it has been thought that, because of both the pliability of pediatric bone and the cartilaginous physis, inversion injuries cause fractures more often in children than they do in adults. Then, as children mature, fractures become less likely, as the bone becomes less pliable and the physis closes. While this concept is true, it is important to remember that most inversion ankle injuries in children, as well as most suspected distal fibular physeal fractures, are in fact ankle sprains. The pathology, evaluation, and treatment of a pediatric ankle sprain are similar to that of an adult sprain.

Traumatic inversion with plantarflexion is the predominant mechanism for ankle sprains. The most commonly injured ligaments, in order, are the anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), and posterior talofibular ligament (PTFL).⁷² Ankle sprains are the single most common injury in high school athletics, with 40% of all injuries occurring in the foot or ankle.⁷³ Basketball is a sport that has historically had one of the highest percentages of ankle injuries, at 25% for both genders, whereas baseball and softball have had some of the lowest, at 7% and 10%, respectively. Of note, women's lacrosse (23%) has been reported to have twice as many ankle injuries as men's lacrosse (11%).⁷⁴

Patients with ankle sprain will complain of pain and difficulty weight bearing immediately after injury. The Ottawa Ankle Rules are useful guidelines to indicate when radiographs are necessary with ankle injuries. They specify that radiography is not necessary unless there is bony point tenderness along the malleolus or the patient is unable to walk four steps.⁷⁵ These rules were initially validated only for adults 18 and older, but recent meta-analysis has confirmed that this guideline is appropriate to use in children 5 and up, as well.⁷⁶

Ankle sprains are classified as having minimal loss of function and involving only the ATFL (mild/Grade I), moderate loss of function with some tissue disruption and involvement of the CFL (moderate / Grade II), or significant loss of function, with complete tearing of all lateral ligaments (severe/Grade III). Grade III sprains may be accompanied by a high ankle syndesmotic injury, which is diagnosed by the squeeze test and is more common in high-impact sports such as basketball, football, hockey, rugby, and soccer.^{77–79} More than 5-mm widening of the syndesmosis, on AP radiograph, indicates syndesmotic injury.⁷²

Acute lateral ankle sprains should undergo appropriate immobilization, rest, and rehabilitation. If shoe modification is desired, flares will provide the greatest improvements to stability.⁸⁰ If a physeal injury is suspected in the absence of an obvious fracture, lack of periosteal reaction on repeat radiographs 7–10 days after the initial injury will confirm the injury is a sprain and not a fibular fracture. In the interim, prior to diagnosis confirmation, weight-bearing immobilization is safe. Most young athletes recover well from ankle sprains. Surgical intervention is reserved for those who have failed a complete course of rehabilitation and have recurrent instability episodes.⁸¹ There is a lack of Level I evidence to support one particular surgical treatment for chronic ankle instability, but the Brostrom method—direct repair and imbrication of the ATFL and CFL—is the most widely used approach.

CONCLUSION

Foot and ankle problems are a common cause of deformity and disability in children and young athletes. While they resemble adult concerns in many aspects, the pediatric skeleton differs from that in adults, and thus a specialized approach is required to discern pathologic findings from typical development or an anatomic variant. Additionally, some presentations, ranging from Sever's disease to triplane fracture, are unique to the growing skeleton. Children have excellent healing potential

compared to adults, but the presence of an active growth plate means that they are also at greater risk for eventual limb length and alignment deformities, a fact that necessitates particular attention and monitoring to the injuries around the physis. With these concepts in mind, most young athletes can be successfully treated and returned to sports without significant longterm sequelae.

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Unique Considerations for Foot and Ankle Injuries in the Female Athlete

Lara Atwater, Rebecca Cerrato

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INTRODUCTION

The passage of Title IX is arguably the most important event in the timeline of women's participation in US athletic endeavors. By almost any measure, the numbers of female athletes have exploded in multiple sports in the decades since 1972.

"No person in the United States shall, on the basis of sex, be excluded from participation in, or denied the benefits of, or be subjected to discrimination under any educational program or activity receiving federal assistance."

**Title IX of the Education Amendments of 1972
to the Civil Rights Act of 1964.**

Before Title IX, approximately 1 in 27 girls participated in college sports; that number today is greater than 1 in 2.¹ The National Collegiate Athletic Association (NCAA) tracks women's sports participation in its member institutions; the data are self-reported and are useful for evaluating general trends. In 1981, total athletes numbered 74,239. By 1993, the total had risen to 105,532 and then in 2016 to over 200,000.^{1,2} In general, the increase in participation reflects the addition of women's teams to institutions. The increase also reflects the elevation to NCAA championship sport status of sports such as ice hockey, water polo, and bowling. Additional emerging sports, such as equestrian, triathlon, and beach volleyball, also have increased the numbers of female athletes (Fig. 28.1).¹

The increase in the number of women participating in sports and the increase in their level of competition has provided a new opportunity to study the effects of different sports on the female athlete. Some sports provide the opportunity to directly compare injury rates for both genders. Other sports are the exclusive domain of the female athlete. Prevention of injuries, whether sport specific or gender specific, has been a primary focus in sports medicine. The sports identified as causing the highest number of injuries in the female athlete are basketball, volleyball, field hockey, and gymnastics.² Sports played on

artificial turf, such as women's soccer and lacrosse, now have a higher rate of ankle injuries and turf toe.³

Men and women have slightly different physical attributes, which have advantages and disadvantages. For example, women's lower extremities constitute less of their total height, which lowers their center of gravity and makes them more amenable to tasks such as the balance beam. Moreover, women's androgenic makeup promotes maintenance of a higher percentage of body fat, which is less desirable in sports such as cross-country. Care of the female athlete must be grounded in awareness of the Female Athlete Triad, which is a spectrum of decreased energy availability, menstrual dysfunction and low bone mass.

When approaching the study of foot and ankle problems in female athletes, disorders can be divided into sport/activity-specific disorders and gender-specific physiologic considerations.

SPORT-SPECIFIC DISORDERS

Gymnastics

The female gymnast is at higher risk for ankle injury than the male gymnast.⁴ Areas of complaint in the foot in female gymnasts include the bottom of the heel, the plantar fascia origin, and the medial longitudinal arch. Direct blows during landings or striking the heel on the floor while swinging under the lower uneven parallel bar cause pain under the heel. Tumbling is the typical cause of pain at the origin of the plantar fascia, whereas landings cause pain in the medial longitudinal arch or the forefoot. Interestingly, analysis of flexibility parameters in gymnasts compared with controls does not demonstrate a correlation to injury patterns or frequency.⁵ Ankle impingement syndrome (see Chapter 2) occurs in the female gymnast, with impingement occurring anteriorly when she lands short on her dismount, forcing her ankle into hyper dorsiflexion. If acute pain occurs with an instance of injury, the ankle should be rested and iced. Physical therapy modalities and antiinflammatory drugs are useful adjuncts.⁶

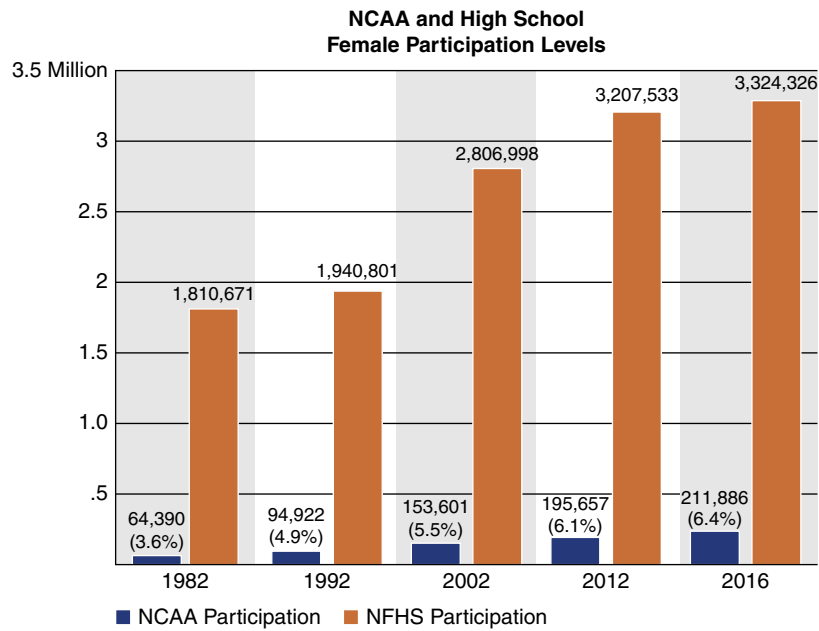


Fig. 28.1 NCAA and High School Female Participation Levels. (From 45 Years of Title IX The Status of Women in Intercollegiate Athletics.) (http://www.ncaa.org/sites/default/files/TitleIX45-295-FINAL_WEB.pdf, pp. 17)

Basketball

The most common injuries in athletes who play basketball affect the knee and ankle. With increased participation of women in collegiate basketball, gender-related injury patterns have been recognized. Previous studies have highlighted the increased incidence in anterior cruciate knee injuries among female basketball players compared with their male counterparts. Additionally, female basketball players are at a 25% greater risk of sustaining a grade I ankle sprain than the male players. The risk was similar in both genders for grades II and III, ankle fracture, and syndesmotic sprains.⁷ A balance study comparing NCAA Division I female athletes from soccer, gymnastics, and basketball found the basketball players demonstrated inferior static balance compared with gymnasts and inferior dynamic balance compared with soccer players.⁸ These studies emphasize the need to create a gender-specific training program designed to strengthen and prevent ankle injuries.

Soccer

A study evaluating the incidence of foot and ankle injuries at a single NCAA Division I athletics program found injury rates and missed-time injuries in women sports were higher than previously reported, and one of the four sports identified with the highest incidence rate is women's soccer.⁹ Similar to basketball, studies have demonstrated the knee and ankle are the most common locations for injury in female players.¹⁰ Furthermore, later prospective studies following young female elite soccer players noted ankle injuries to have the highest incidence.¹¹ Over the past two decades, great attention has been placed on understanding the risk factors for ACL injuries in female athletes and placing focus on injury prevention protocols. As the predominant proportion of injuries to female soccer players involve the ankle, a similar effort in creating prospective interventional studies should be initiated.

Volleyball

Ankle sprain is the most common injury in women's NCAA volleyball, accounting for 22% of all injuries from 2013 to 2015.¹² In both men and women, 90% of these injuries are caused when one player lands on another.¹³ Predisposing factors for ankle injury include prior ankle injury (80%), increased tibial varum, increased calcaneal range of motion, and higher range of motion through the first metatarsophalangeal joint. Prevention programs in higher-risk athletes should focus on functional neuromuscular control and improve joint position sense, kinaesthesia, and joint force sense.¹⁴ Bracing has been shown to reduce the risk of ankle sprains, especially in those with prior ankle sprains.¹⁵

Cheerleading

Cheerleading, a sport that is 96% female, has steadily become more popular over the past 30 years, with a corresponding 400% increase in cheerleading-related emergency department visits between 1980 and 2007. The percentage of ankle injuries is between 28% and 45%.^{16–18} In a study by Shields et al., 45% of cheerleaders who sustained a sprain returned to full cheerleading activity by the very next performance or practice. As more 5- to 11-year-olds participate in independent "all-star leagues," providers should be aware these girls are 1.6 times more likely to sustain a fracture/dislocation than older cheerleaders (age 12–18).² Coaches and providers can use anti-ankle sprain strategies such as functional neuromuscular control training. Moreover, cheerleading is not an NCAA championship sport and is not recognized as a sport by high school athletic associations in some states. Studies suggest that lack of proper training surfaces and coach education contribute to the rate of injury. Therefore, providers should be aware of the level of coach education as well as the kinds of surfaces being used for practice and performance (fewer injuries occur on spring flooring than hard gymnasium floors made of rubber/polyurethane).¹⁹

Ballet (also see Chapter 24)

The female classical ballet dancer is unique in her requirements for the lower extremities.^{19,20} The dancer uses either a thin-soled slipper or pointe shoe. In pointe work, the stiff shank and hard toe box of the pointe shoe support the foot. The lower extremities are called on to absorb all the force of landings on the wooden dance floor. The consequence of the schedule of training and performance and the type of shoe for the foot leads to chronic injuries such as tendinitis, tendinosis, and impingement syndromes. The most common acute injury is the inversion sprain, usually occurring on landing a jump.²¹ Overuse, fatigue, improper technique, and anatomic variation from optimal body type all can be factors in acute and chronic injuries. The lower leg, foot, and ankle make up approximately 40% of dance injuries in a sport in which the lifetime incidence of injury is 90%.²²

Ballet requires extreme plantarflexion of the foot for *en pointe* work. In this position, soft tissues posterior to the ankle can be compressed and irritated. Structures vulnerable to these forces include: an os trigonum, a Stieda process, or a large dorsal process of the calcaneus. Symptomatic flexor hallucis longus (FHL) tendinitis can be caused by these impingement scenarios. Diagnosis of this suspected condition can be supported by local tenderness proximal to the sustentaculum tali and pain with resisted plantarflexion of the great toe. Magnetic resonance imaging (MRI) typically will demonstrate fluid within the sheath of the tendon and sometimes marked tenosynovitis.²³ Preservation of the function of the FHL tendon is paramount in dancers. Treatment should be aimed at minimizing the inflammatory condition, with surgical intervention timed to allow appropriate recovery. In some instances, simple release of the FHL is adequate; in other cases, excision of the os trigonum or posterior process of the talus may be required. Flexor hallucis longus tendon symptoms are most commonly associated with ballet; however, participants in other sports such as gymnastics and soccer increasingly are demonstrating the same entity (Fig. 28.2).²⁴

The most common acute ballet injuries occur as the dancer lands with a loss of balance. If the dancer lands in *en pointe* position, the ankle is more stable, causing a midfoot injury rather than the typical ankle sprain. The most commonly overlooked fractures include the talar dome (see Chapter 16), the lateral process of the talus (see Chapter 16), the os trigonum (see Chapter 2), the anterior process of the calcaneus, and the proximal fifth metatarsal. Younger dancers can be more difficult to evaluate, often requiring repetitive x-rays. A high index of suspicion should be maintained, especially in the face of soft-tissue swelling over the physes of ankle or foot bones.^{25, 26}

As in other athletes, inversion injuries can cause damage to structures other than the anterior talofibular ligament. Syndesmosis tears, osteochondral lesions of the talus, and subluxation or longitudinal tears of the peroneal tendons all may occur. Dancers also are at risk for subluxation of the cuboid, either associated with an inversion injury to the ankle or from repetitive plantarflexion and dorsiflexion. In this clinical entity, the base of the fourth metatarsal becomes dorsally displaced and the fourth metatarsal head displaces in a plantar direction. Additionally, cuboid dysfunction can interfere with normal function of the peroneal tendons and must be considered in



Fig. 28.2 Prominent Steida process (posterior lateral process of talus). (From Kadel N. Foot and ankle problems in dancers. *Physical Medicine and Rehabilitation Clinics*. 2014;(25)4:829-844.)

dancers with peroneal tendinitis. Treatment of this unusual condition requires reduction of the cuboid with a squeeze technique after the hindfoot is mobilized and the forefoot is adducted.²⁷

Midfoot injuries in the dancer present a significant treatment dilemma because of the prolonged healing time required for stability of the foot and the difficulty of restoring the mobility required for dancing. Midfoot injuries occur when the dancer lands in full *pointe* position, with the posterior lip of the tibia resting and locked on the calcaneus. In this position, the subtalar joint also is locked, and the heel and forefoot both are in varus. Because the ankle joint is relatively stable in full *pointe*, the forces at landing are transferred to the midfoot. Treatment of these acute injuries requires evaluation of both stabilities of the involved tarsometatarsal joints and amount of collapse of the longitudinal arch (see Chapter 7). Imaging obtained should include bilateral weight-bearing foot radiographs, possible stress radiographs, and in some cases, advanced imaging such as an MRI or computed tomography (CT) scan.

The fifth metatarsal is a common area of injury for dancers. Zone 1 fractures of the fifth metatarsal involve an avulsion of the base. Open reduction internal fixation (ORIF) is recommended only if the fracture fragment involves greater than 30% of the articular surface and is significantly displaced. The most typical fracture involves only the most proximal 1 cm of the bone and usually is associated with an inversion ankle sprain.²⁷ It can be treated with appropriate immobilization and progressive activity as healing permits. The Jones fracture (see Chapters 5, 7) occurs by the mechanism of adduction of the fifth metatarsal, usually while the foot is plantarflexed. Because of higher risk of nonunion combined with negative effects of prolonged immobilization, many surgeons advocate for early operative management for these fractures at the metaphyseal-diaphyseal junction.

Repetitive adduction forces that occur with cutting or pivoting movements can result in diaphyseal stress fractures. There

usually are prodromal symptoms preceding an acute event. The history is informative, as is review of radiographs, which typically demonstrate periosteal reaction, cortical thickening, intramedullary sclerosis, and widening of the fracture line. Because this is a vascular watershed zone, these stress fractures should be treated with intramedullary screw fixation, bone graft, or both.

When dancers perform the *demi-pointe* position, the foot is twisted and inverted and can incur an oblique or spiral fracture of the mid- to distal portion of the fifth metatarsal. This “dancer’s fracture” now has been shown to heal well with conservative and symptomatic treatment rather than ORIF.²⁸

GENDER-SPECIFIC CONSIDERATIONS

Anthropometric studies provide data concerning anatomic differences between women and men.²⁹ The increased number of androgens in males promotes lean body mass, with the percentage of body fat in men (12%–16%) being less than in women (22%–26%). Given the same body weight, women also have a smaller heart, lower blood pressure, and smaller lungs, with a slightly lower aerobic capacity.³⁰

For women, lower extremities constitute 51% of total height, compared with 56% in men. This difference improves the mechanical advantage for men in activities requiring striking, hitting, or kicking because greater force can be generated by their legs as longer levers. The female has a wider pelvis, greater varus of the hips, and greater genu valgus than the male, resulting in a lower center of gravity. In sports requiring excellent balance, such as gymnastics, this gives females a distinct advantage.³¹ As such, the balance beam is a required element in competition for female gymnasts and is not included in the competition for male gymnasts.

Female gymnasts typically also have better joint mobility than males. This improves their flexibility—another trait valued in gymnastics. The alignment differences between male and female at the hip and knee may be one factor, along with the level of conditioning, contributing to higher percentages of overuse syndromes in the lower extremity in female athletes.

Clinicians caring for the female athlete must be aware of the Female Athlete Triad. This phenomenon refers to the interrelated problems of decreased energy availability (with or without disordered eating), menstrual irregularity, and decreased bone mineral density.³² Each of these elements exists on a continuum, and the female athlete may have dysfunction of one or all three elements. The most severe manifestation of the triad would be low energy availability with an eating disorder, functional hypothalamic amenorrhea, and osteoporosis. The prevalence of the Female Athlete Triad is higher in sports that select for a slim body habitus or involve subjective judging, but it has been reported in all sports.

Physician awareness of the Female Athlete Triad begins during the preparticipation physical. The Female Athlete Triad Coalition published a 2014 consensus statement on preparticipation screening questions and risk factors triggering workup and return to play. The preparticipation interview should involve questions that gauge the onset of menses and frequency of periods, weight-consciousness, history of food allergies or eating disorders, and/or stress fracture history.³³ The Female Athlete Triad Coalition also developed a risk factor stratification tool that aids in determining an athlete’s clearance of sport participation. Important high-risk criteria include body mass index (BMI) <17.5, fewer than six menses over 12 months, Z-score < -2.0, or more than two stress fractures (Fig. 28.3). For all female athletes, the NIH recommends 1300 mg and

Risk Factors	Magnitude of Risk		
	Low Risk = 0 points each	Moderate Risk = 1 point each	High Risk = 2 points each
Low EA with or without DE/ED	<input type="checkbox"/> No dietary restriction	<input type="checkbox"/> Some dietary restriction‡; current/past history of DE;	<input type="checkbox"/> Meets DSM V criteria for ED*
Low BMI	<input type="checkbox"/> BMI ≥ 18.5 or ≥ 90% EW** or weight stable	<input type="checkbox"/> BMI 17.5 < 18.5 or < 90% EW or 5 to < 10% weight loss/month	<input type="checkbox"/> BMI ≤ 17.5 or < 85% EW or ≥ 10% weight loss/month
Delayed Menarche	<input type="checkbox"/> Menarche < 15 years	<input type="checkbox"/> Menarche 15 to < 16 years	<input type="checkbox"/> Menarche ≥ 16 years
Oligomenorrhea and/or Amenorrhea	<input type="checkbox"/> > 9 menses in 12 months*	<input type="checkbox"/> 6-9 menses in 12 months*	<input type="checkbox"/> < 6 menses in 12 months*
Low BMD	<input type="checkbox"/> Z-score ≥ -1.0	<input type="checkbox"/> Z-score -1.0*** < -2.0	<input type="checkbox"/> Z-score ≤ -2.0
Stress Reaction/Fracture	<input type="checkbox"/> None	<input type="checkbox"/> 1	<input type="checkbox"/> ≥ 2; ≥ 1 high risk or of trabecular bone sites†
Cumulative Risk (total each column, then add for total score)	___ points +	___ points +	___ points = ___ Total Score

Fig. 28.3 Cumulative Risk Assessment. (From Joy E, De Souza MJ, Nattiv A, et al. 2014 Female Athlete Triad Coalition consensus statement on treatment and return to play of the Female Athlete Triad. *Curr Sports Med Rep.* 2014;13(4):219-232.)

1000 mg of calcium supplementation for women less than or greater than 18 years of age, respectively. Six hundred international units of daily vitamin D are recommended for adults <70 years of age.³⁴

Female gender is an independent risk factor for stress fractures, regardless of Female Athlete Triad dysfunction. A prior stress fracture is the best predictor of future stress fractures in all age groups.³⁵ The Female Athlete Triad makes women more susceptible to stress fractures that occur as a result of repetitive, low-level stresses on bone and have multifactorial causes. In addition, menstrual irregularities such as exercise-induced amenorrhea may result in lower bone density, which increases stress fracture risk. One study of 748 high school runners found menarche after age 15 was an independent risk factor for stress fractures.³⁶ The use of oral contraceptives was previously advocated to regulate and maintain bone density, but recent research suggests that it causes further harm by reducing bone mineral density if taken for more than 2 years.³⁷ In a study of NCAA athletes by Tenforde et al., sports emphasizing leanness such as cross-country, gymnastics, and lacrosse had the greatest proportion of athletes at moderate to high risk of stress fracture. Cross-country runners had the highest prevalence (34%) of such fractures followed by basketball players (22.2%).³⁸

Clinical presentation of stress fractures is often insidious, beginning earlier in each training session and eventually occurring during the course of normal activity. Point tenderness localizing to the affected bone is common in most fractures; however, specific fractures can present with misleading symptoms. For example, calcaneal stress fractures can present like plantar fasciitis with morning heel pain but will have a positive heel squeeze test; navicular stress fractures are uncommon but can produce otherwise unexplainable midfoot pain.³⁹ Running athletes commonly get stress fractures in the distal metatarsals, whereas dancers are at risk for stress fracture at the base of a second metatarsal, which has a higher rate of nonunion. Early radiographs of stress fractures are often unremarkable. Bone scans can be positive within a few days after injury, but MRI is the recommended modality for diagnosis.²²

CONCLUSION

All medical support personnel involved in the care of the female athlete must keep several salient facts in mind. First, the number of females involved in all sports has increased dramatically and will continue to increase. Second, like their male peers, female athletes will continue to push their limits, likely incurring injury in the process. Third, injuries in the female athlete will fall into either sport-specific or gender-specific categories, the latter of which require a higher index of suspicion and perhaps greater depth of knowledge for diagnosis and treatment. This is a pivotal time for women's sports medicine, with great opportunity to gather data, to refine optimal treatment recommendations, and to develop strategies to prevent injury. This chapter delineates many of the injuries that the medical personnel will encounter and the salient concepts of diagnosing and treating these injuries in women.

PEARLS

Ankle sprains are extremely common in female athletes, and prevention programs should focus on functional neuromuscular control to improve joint position sense, kinaesthesia, and joint force sense.

The lower leg, foot, and ankle make up approximately 40% of dance injuries, and the lifetime incidence of injury is 90% in the dancer.

The Female Athlete Triad consists of low energy availability, menstrual dysfunction, and low bone mineral density. It places the female athlete at an increased risk of stress fractures in the foot and ankle.

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Orthobiologics in Foot and Ankle Applications

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INTRODUCTION

In recent years, as orthobiologic interventions for a variety of sports-related foot and ankle conditions have become more readily available, results are beginning to appear in the literature. The notion that we can affect the biology of the musculoskeletal tissues with these treatments and trigger a healing response is generally accepted. What conditions can be affected, how these orthobiologics are delivered, the mechanism of action, and the posttreatment modalities are being defined. Since these preparations can be delivered percutaneously, applied topically, and used in surgery as stand-alone or adjuvant approaches, there are often confounding variables that affect their assessment.

By the U.S. Food and Drug Administration (FDA) definition, biologics are biological products made from a variety of natural sources (human, animal, or microorganism) or the final products of advanced biotechnologies for medical applications (www.Fda.gov/aboutfda). The most common autologous orthobiologics, biologics for orthopaedic applications, in use today are platelet-rich plasma (PRP) and bone marrow concentrate (BMC). There is an increasing frequency of use of autologous fat tissue-derived treatment preparations, including stromal vascular fraction (SVF) and “fractured fat.” There also are allogeneic (donor tissue, cell, or growth factor) products obtained from donated placentas, amniotic membranes, or fluids. These products contain growth factors with or without viable cells, depending on the specific product. Because biologics are often a mixture of multiple biological components that are difficult to quantify in many cases, their mechanisms of action through

cellular signaling may not be the same as that in vitro or are still unknown. Recombinant growth factors (e.g., recombinant human bone morphogenetic protein [rhBMP] and recombinant human platelet-derived growth factor [rhPDGF] and synthetic compounds (hyaluronic acid, calcium phosphate, or calcium sulfate) are regulated by the FDA as devices or drugs and are released with specific indications for orthobiologic use.

Often these biologic products are used in an “off-label” fashion for specific conditions “within the standard of care” (i.e., within the standards of appropriate medical practice without committing malpractice), even though they are not approved for that situation. Off-label use is not against the law, since the FDA does not regulate the practice of medicine. The companies that provide the products must comply with the FDA and cannot advertise or encourage an off-label use. Practitioners determine treatment on the basis of their clinical and scientific experience in accordance with their medical community’s reasonable standards, given the patient’s specific condition and setting.

From a regulatory perspective, human cells and tissues that comply with standards of the American Association of Tissue Banks are minimally manipulated in processing, and are marketed for homologous use are in compliance with US FDA regulation 21 CFR 1271 on human cells, tissues, and cellular and tissue products (HCT/P). Examples given in the regulation are “amniotic membrane when used alone or without added cells, bone, cartilage, cornea, fascia, ligament, pericardium, peripheral or umbilical cord blood stem cells for autologous use or use in a first or second degree blood relative), sclera, skin, tendon, vascular graft, heart valves, dura mater, reproductive

cells, and tissues (e.g., semen, oocytes, embryos). On the other hand, if an HCT/P product is “dependent upon the metabolic activity of living cells for its primary function,” it is not regulated as a section 361 product according to an FDA guidance document on minimal manipulation and homologous use of HCT/Ps, and requires FDA clearance.¹ HCT/Ps that are not minimally manipulated or used for homologous applications need FDA approval, since they are considered to be biological drugs. PRP and BMC are fully compliant with the existing regulatory framework established by the FDA, when used on an autologous basis and with homologous use. Interestingly SVF, although widely used in medical practice, is accepted as within the standard of care but falls outside the current standards established by the FDA for point-of-care (POC) therapeutic agents (e.g., 21 CFR 1271) as indicated in the FDA Guidance issued in late 2017. Placental products vary broadly and often fall into a gray zone, in which the tissue may be treated as an allograft but may only be marketed for specific uses, or the material contains living cells that require FDA clearance. The only noncontroversial placental-derived products on the market are sheet-forms used as a covering for wounds, or amniotic fluid without cells or micronized tissue. Neither of these types of products are “cleared” by the FDA, although the agency has taken action against companies who advertise the sheet-form for promoting healing or reducing pain. Use of the word “indication” implies a formal clearance by the FDA. Manufacturers can state what the material is used for, but not as an “indication for use” in the classic sense. Companies that produce these products must register and comply with current good manufacturing procedures (cGMP) and donor screening and tracking to qualify for regulatory clearance. In general, further definition and oversight for these products are expected to expand.¹

UNDERLYING MECHANISMS OF THERAPEUTIC BENEFIT

There are a number of biologically derived materials that have been used for regenerative therapy, including autogenous (e.g., PRP, BMC, and SVF) and allogeneic materials (e.g., placental/amniotic tissue-derived fluid and membranes).

A key distinction between PRP and BMC is the presence of progenitor cells in the bone marrow, including multipotent mesenchymal stromal cells (MMSC) formerly known as mesenchymal stem cells (MSC). In the lab, those cells can be isolated from bone marrow and demonstrate two critical behaviors: 1) they can differentiate into adult tissue cells under specific conditions (e.g., becoming chondrocytes, osteoblasts, and adipocytes); and 2) they can replicate without undergoing differentiation. Therefore, harvesting these stem cells does not create a deficiency given their ability to replenish their population. These cells are, in fact, present in all tissues of the body and produce trophic factors that help to coordinate/organize repair and regeneration of pathologic tissue through the production of growth factors and cytokines.² The cells can immunomodulate and help control inflammation. They home to the site of injury and reside in the local injured tissues around blood vessels.³

The distinction between the classical term “MSC” and the newer iteration “MMSC” must not be overlooked, as this distinction is central to understanding the origin of these cells and how they can be applied for therapeutic benefit. Historically, *mesenchymal* stem cells were aptly named because they were thought to derive from the connective tissue (stroma) of bone marrow and other tissue subtypes. The publication of a landmark article by Crisan and colleagues in 2008 marked a significant paradigm shift with the realization that MSCs are not stromal in nature, but rather derived from perivascular cells (pericytes).⁴ Indeed, Crisan’s data were so compelling that Arnold Caplan himself—a pioneer of the MSC—actually went so far as to conclude that all MSCs are pericytes.⁵

This change in dogma is particularly significant because it has had such a profound effect upon the way we view the functionality of MMSCs and, consequently, the way we view their therapeutic capacity. With the characterization of MMSCs as pericytes, the therapeutic focus of these cells has shifted from their multipotent differentiation to their secretory ability, which leads to immunomodulatory and trophic effects.⁶ This shift has paved the way for a “new era” of cell-based orthobiologics to arise over the past decade, with applications ranging broadly from tendon healing to fracture union to cartilage regeneration.^{7–13}

The increased emphasis upon the secretory function of MMSCs is perhaps best illustrated by MMSC-derived exosomes, which have recently gained traction in biomedical research for their paracrine therapeutic potential. The exosome is a type of extracellular vesicle that is released by the MMSC and, much like its cellular counterpart, helps to maintain tissue homeostasis.¹⁴ While the precise mode of action is not completely understood, exosomes are believed to deliver mRNA, miRNA, and proteins to the target cell, thereby modulating the recipient cell function through complex cellular signaling cascades.^{15,16} Furthermore, exosomes have several possible advantages over traditional cell-based therapy: eliminated risk of mutation, decreased likelihood of immune rejection, improved tissue migration, and circulation through small blood vessels (e.g., capillaries).^{17,18}

The therapeutic possibilities for MMSC-derived exosomes are extensive, with applications in orthopaedic surgery and several other medical disciplines. With regard to musculoskeletal biology, in vitro and animal studies have proposed a role for exosomes in fracture healing, osteoporosis, cartilage regeneration, intervertebral disc degeneration, and osteonecrosis of the femoral head.^{19–28} Although donor-derived exosomes currently remain in the preclinical phase, they have shown great promise in these early investigations and may prove to be an important component of nonoperative therapeutics for a variety of orthopaedic conditions in the not-so-distant future.

Platelet-Rich Plasma

PRP, being derived from whole blood, contains very few circulating stem cells under normal conditions. What gives PRP a therapeutic potential in treating certain types of pathologies is the concentrated level of platelets, which release vesicles like exosomes, which might contribute to their therapeutic potential.

These exosomes are filled with a wide variety of growth factors and other biologically active proteins. The plasma component in PRP contains several proteins that have been shown to have a beneficial effect, including interleukin 1-receptor antagonist protein (IL-1RAP or so-called IRAP) and α -2-macroglobulin (α -2-M).^{29,30} IL-1RAP directly blocks the pro-inflammatory action of IL-1, a cytokine released by macrophages and neutrophils, in particular, at sites of inflammation, and is responsible for pain and sustaining a pro-inflammatory environment. At the site of injection, platelets release growth factors that are thought to stimulate/interact with adult tissue cells, enabling those cells to initiate a repair sequence. Releasing growth factors in a small-volume tissue (i.e., intratendinous) creates a stimulatory gradient of growth factors as they diffuse away from the site of implantation. As a result of this gradient, adult stem cells in the adjacent tissue may migrate to the treatment site. Thus, the benefit of PRP is an indirect one, since the growth factors act on adult tissue cells and stem cells present in the adjacent tissue to begin the reparative process.

Bone Marrow Concentrate

Bone marrow contains cells needed to continuously replenish the cellular components of blood: white blood cells (WBCs), red blood cells (RBCs), and platelets. These adult peripheral blood components are derived from hematopoietic stem cells (HSCs). In bone marrow, there are also MMSCs or MSCs, which don't give rise to blood cells. They were initially identified in bone marrow stroma and believed to provide microenvironmental support to HSCs. In the 1960s, the pioneering work by Alexander Friedenstein proved that they are multipotent stem cells, like HSCs, being able to differentiate to osteoblasts, chondrocytes, and adipocytes.

In view of the high cellular content of BMC, which includes a wide variety of progenitor/stem cells, BMC provides a direct therapeutic benefit at the site of implantation. The plasma of the BMC preparation is a source of additional benefit, as mentioned for PRP, but with some differences. For example, BMC has been shown to contain 20-fold higher levels of IL-1RAP compared with patient-matched PRP preparations, while BMC will contain less α -2-M compared to PRP, since α -2-M is actively secreted into peripheral blood.^{29,30} One advantage of using BMC over PRP is that cellular activity in BMC treatment is initiated much more quickly, since the progenitor and stem cells are present in the injectate and don't need to migrate to the treatment site, thereby shortening the time lag for initiation of repair.³¹

Since MMSCs are able to differentiate into a wide variety of tissue cells, it was thought that MMSCs were therapeutically beneficial due to their ability to differentiate. However, after studies reported that MMSCs in most cases differentiated into adult tissue cells at a very low rate (< 2%–3%), research focus shifted to the fact that MMSCs are able to secrete a diverse set of cytokines and growth factors. Murphy and colleagues have reviewed the extensive list of biologically active molecules MMSCs produce when activated.² For example, Murphy et al. indicated that there are five separate biochemicals that MMSCs secrete in a pro-inflammatory environment. This approach is called the paracrine effect and is similar to what platelets do when they degranulate and release growth factors. An important advantage of working with BMC is

that the stem cells present do not disappear shortly after initiating their paracrine effect, but continue to respond to the environment, thereby modulating their response as tissue is being repaired over a longer period.

Dose–Response Relationships of PRP and BMC

As indicated above, the important components in PRP and BMC that are associated with a therapeutic benefit have been identified. Counting the number of platelets present in a PRP sample and assessing the levels of critical growth factors contained is possible, but very few of the clinical studies include these data. Furthermore, the use of RBC- and/or WBC-reduced PRP preparations creates more challenges. More work is needed to define these variables and their impact on healing.

On the other hand, mostly due to the pioneering work of Philippe Hernigou (Chief of Orthopaedic Surgery, Henri Mondor Hospital, East University [University of Paris], Créteil, France), BMC preparations have been characterized in terms of the levels of MMSCs present in the injectate. For example, Hernigou and his coworkers observed that study subjects receiving an autologous BMC treatment following a standard surgical repair of a full-thickness supraspinatus tendon tear were twice as likely to have an intact tendon at the 10-year milestone compared with patients receiving just the standard surgical repair.³² They showed that all of the BMC-treated patients who had more than 30,000 MMSCs (as determined by assessing the number of colony-forming unit fibroblasts [CFU-Fs] in the preparation) per injectate had intact tendons at the 10-year milestone, whereas those patients receiving fewer than 30,000 MMSCs all had tendon failures by the 10-year point. Hernigou and colleagues have also reported similar threshold effects in treating other pathologies, including tibial nonunion and avascular necrosis of the femoral head.^{33,34} The problem with trying to establish stem cell dosing guidelines is that each patient has their own level of adult stem cells in their bone marrow. Thus, an explicit knowledge of the number of MMSCs in a preparation is of retrospective benefit only due to the lag time in obtaining a CFU-F value, but it can help physicians refine how they aspirate bone marrow in order to maximize the potential to get a therapeutically beneficial level of adult stem cells. Dr. Hernigou's group has published several papers on his bone marrow aspiration technique, including specific recommendations on the practical aspects of aspiration.^{35–37}

ADIPOSE STEM CELLS

As early as the turn of the 21st century, adipose tissue was identified as a potential autologous source of MMSCs.³⁸ Much like bone marrow, adipose contains an easily isolated stroma-derived cell type that can differentiate into osteogenic, tenogenic, myogenic, and chondrogenic lineages.³⁹ These unique properties led to the use of lipoaspiration as a means of harvesting adipose from fat-rich areas, with the end goal of applying this tissue for therapeutic benefit in other parts of the body. However, before the adipose tissue can be re-injected, it must first be processed in order to isolate an MMSC-rich product.

Several methods for processing adipose tissue have been proposed in the literature. Classically, the preferred technique has relied upon manipulation of the cells through enzymatic degradation.³⁸ This method involves treating the lipoaspirate with collagenase to digest the extracellular matrix, then centrifuging the sample to obtain the product known as the stromal vascular fraction. Because the SVF contains a variety of cell types, cultures are required to isolate the adipose-derived stem cells (ASCs). The longer time required for culture has limited the clinical utility of this technique, since it is not feasible to perform during a single procedure in the operating room. Moreover, the FDA has yet to approve the use of enzymatically digested ASCs for musculoskeletal disorders in humans.

In an animal study, local injections of enzymatically digested ASCs significantly increased the tissue volume of the foot fat pad in rats.⁴⁰ A few studies, all performed outside the US, have reported clinical outcomes following the injection of enzymatically digested ASCs or SVF for foot and ankle conditions.^{41–43} In a study of 49 patients with varus ankle osteoarthritis (OA) who underwent arthroscopic bone marrow stimulation with lateral calcaneal osteotomy, clinical outcome scores were significantly better in patients who received a supplemental injection of enzymatically digested ASCs compared with those who underwent the surgical procedure alone. Similarly, a study of 49 patients with osteochondral lesions of the talus revealed that bone marrow stimulation supplemented by SVF injections was associated with superior clinical and MRI outcomes compared with marrow stimulation alone. Further, SVF injections also showed benefit for Achilles tendinopathy in a randomized controlled trial, with significantly better outcome scores at 15 and 30 days postprocedure compared with PRP injections.

Recently, a novel processing method has been proposed that employs mechanical forces to create a reduced-volume preparation with viable progenitor cells, including ASCs.⁴⁴ This technique, known by the trade name Lipogems (Lipogems International SpA, Milan, Italy), is a closed system that processes adipose tissue using microfragmentation as opposed to enzymatic digestion. Preclinical studies of microfragmented adipose tissue have demonstrated promising results when compared with the conventional processing methods. In two studies, microfragmentation produced a homogenous adipose tissue product with a significantly higher percentage of ASCs and a lower number of hematopoietic elements compared to enzymatic digestion.^{44,45} A separate study demonstrated that microfragmented adipose tissue releases more growth factors and cytokines involved in tissue repair and regeneration compared with enzymatically degraded adipose tissue.⁴⁶ Finally, microfragmented lipoaspirate has also shown the ability to induce the production of connective tissue in a paracrine fashion, suggesting that this technique may have a therapeutic role in cartilage regeneration or repair.⁴⁷

To date, the authors are not aware of any clinical studies in the foot and ankle literature that have reported clinical outcomes following the injection of microfragmented fat. However, preliminary data from the knee literature do suggest that this may be a safe and effective therapeutic option for cartilage disorders of the foot and ankle. In a small study of 17 patients with knee OA, microfragmented fat injections were associated

with no adverse events and a significant improvement in Knee Society Scores at 6 weeks and 12 months.⁴⁸ Similarly, a larger study showed improvements in both International Knee Society scores and Visual Analogue Scale (VAS) scores in 52 patients with knee OA who underwent microfragmented fat injection plus arthroscopic debridement.⁴⁹ Given the lack of robust clinical data in the literature, a prospective randomized controlled trial has recently been initiated to compare the efficacy of microfragmented fat and hyaluronic acid for mild-to-moderate knee OA.⁵⁰ Further studies are necessary for foot and ankle applications before conclusions may be drawn regarding the utility of microfragmented fat injections in this patient population. Also, regulatory issues on the technique and technology are being clarified.

PLACENTAL/AMNIOTIC PRODUCTS

The human placenta supports fetal development and has been used since 1910 as a source of allogeneic tissue to assist healing.⁵¹ The placenta is delivered after a birth and consists of the chorion, the amnion, the umbilical cord, and the amniotic sac.⁵² These tissues are rich in cells and growth factors; they can be donated by consenting mothers and tested for viruses (human immunodeficiency virus, hepatitis, cytomegalovirus, human T-lymphotropic virus), then processed according to standards established by the American Association of Tissue Banks.^{53,54} Placental tissue allografts have a role in the healing of conjunctival lesions, ulcers, and wounds.^{55–57}

Currently, placental or amniotic-derived tissue products work as a delivery system of growth factors and as a scaffold that play a unique role in wound healing. In some products the presence of cells may provide additional benefit.^{58,59} Also, the cells may not be fully immune-privileged and may incite a host response. While the allograft material for creating patches (non-micronized) from placental tissues contains viable stem cells and adult tissue cells, most products that are FDA-compliant are those processed using techniques that kill any viable cells present in the tissue. Both the growth factors and the matrix itself are thought to be responsible for the therapeutic benefit of using placental tissue-derived membrane patches in wound healing. It is thought that the growth factors migrate from the tissue implant into the underlying wound bed to activate local stem and adult tissue cells residing there. These products also act as a physical barrier, helping to reduce the chance of infection. Finally, the matrix of the tissue has been shown to promote the activity of cells that have migrated into the tissue implant itself. This in-migration is thought to promote the melding of the tissue patch with the wound bed, thereby aiding in re-epithelialization of the wound.⁶⁰

Cellular products such as the Pluristem allogeneic cell (Pluristem Therapeutics Inc., Haifa, Israel) can be harvested and expanded for use as growth factor delivery agents. Studies have been initiated to establish a role for these cells in orthopaedics.⁶¹ Currently, the use of this biologic in the musculoskeletal system is still investigational in the United States. Another cellular product associated with tissues that contain growth factors is available, Grafix (Osiris Therapeutics, Inc.,

Columbia, Maryland). Currently it is in Phase 2/3 clinical studies for approval by the FDA as a biological drug product, per an agreement reached between Osiris and the FDA in 2015. This human-derived, viable wound matrix, composed of cryopreserved amniotic membrane, was applied weekly to treat diabetic foot ulcers ($n = 50$), resulting in a wound closure rate of 62.0% at 12 weeks, compared to a 21.3% closure rate with standard of care ($n = 47$).⁶²

Amniotic fluid also has been found to contain MMSCs, but applications of these cells in healing are largely experimental.⁶³ There is debate about the viability of the cells *in vivo* and their engraftment into local tissues. However, these fluids may contain growth factors that may provide symptomatic relief in musculoskeletal conditions.⁶⁴ In one study of 44 patients with plantar fasciitis and Achilles tendinopathy, good resolution of symptoms was demonstrated using an injection of cryopreserved amniotic membrane and amniotic fluid-derived cells.⁶⁵

Umbilical cord-derived cells are currently banked as cord blood but do not yet have an indicated use in orthopaedics. The umbilical cord consists of an umbilical vein and two arteries surrounded by a collagenous, hyaluronan matrix called Wharton jelly. The cord components can be processed and used to produce growth factors, but there are no studies to support their efficacy.^{66–68}

Lyophilized human umbilical cord is decellularized and may be a source of growth factors for certain applications.

Most of the placental products available are decellularized and either dehydrated or cryopreserved. They are typically derived from the chorion, which is on the maternal side of the placenta, or the amnion, which is on the fetal side. These tissues are immune-privileged and, with proper processing, can be used as sheets or particles of growth factors. In general, the dehydrated tissues have been shown to decrease tissue fibrosis, enhance soft-tissue healing, and modulate immune function.⁶⁹

In addition to their widespread use for corneal lesions, placental products have also shown benefit in the musculoskeletal system for wound healing.^{70–72}

The various products are not equal due to procurement, processing, and preservation procedures. They are not all well studied, but in general have been used for diabetic ulcers and to promote healing. The best-studied products are EpiFix and AmnioFix by MiMedx (MiMedx Group, Inc., Marietta, GA), which has characterized over 226 growth factors.⁷³

AmnioFix is one of the new regenerative medicine advanced therapy (RMAT) category products that the FDA created after the 21st Century Cures Act, so it is in an investigational new drug clinical study premarket approval program. AmnioFix and EpiFix have been used in clinical studies and have shown consistent safety and efficacy in diabetic wounds, vascular wounds, and plantar fasciitis.^{74–76} A case study of 22 patients has also shown benefit in Achilles tendonitis.⁷⁷ The benefit of this path of delivery is that it is relatively cost effective and requires minimal processing by the physician to inject. Future studies may show benefits in a greater number of indications including articular cartilage defects and osteoarthritis.^{78,79}

A similar product by Amniox Medical, Inc., a TissueTech company in Miami, Florida, has been evaluated in a retrospective

study of 124 foot and ankle patients for several conditions including open repair of peroneal and Achilles tendinopathy with an overall wound complication rate of 5.64% and a reoperation rate of 1.6% (2/124).⁵⁹

In a randomized, controlled, double-blind pilot study of 23 patients with plantar fasciitis treated with either steroid or cryopreserved human amniotic membrane, the amniotic membrane was found to be safe and effective relative to the steroid injection.⁸⁰

RECOMBINANT GROWTH FACTORS

Across the field of orthopedics, recombinant growth factors such as BMP and PDGF have become increasingly popular adjuncts to promote bone healing. Although BMP is only FDA-approved for lumbar fusions and tibial nonunions, there is a mounting body of evidence to support its use in foot and ankle surgery. Several articles in the literature have reported increased fusion rates with the addition of BMP-2 and BMP-7 in high-risk patients undergoing arthrodesis of the ankle or hindfoot.^{81–85} However, given the retrospective design of these studies, the overall level of evidence regarding the efficacy of BMP in foot and ankle fusion remains low. Randomized prospective data must be presented if BMP is to become ubiquitous in this patient population.

In contrast to BMP, multiple randomized prospective trials have compared PDGF to autogenous bone graft (ABG); the current gold standard in foot and ankle fusion.^{86,87} A review article by Sun and colleagues aggregated clinical, radiographic, and safety data from the 634 patients included in these three high-quality studies.⁸⁸ No significant difference was observed between PDGF and ABG with regard to safety or radiographic outcomes. Comparative analysis of the clinical data also yielded similar outcomes between groups, with the exception of the long-term SF-12 Physical Component scores, which were superior in the ABG cohort. Based upon these results, the authors concluded that PDGF is a viable alternative to ABG in foot and ankle fusion.

In addition to the value of PDGF as an adjunct to enhance bony fusion, emerging studies have begun to expand the scope of this growth factor in foot and ankle surgery. Novel arthroscopic procedures have utilized PDGF in the treatment of talar osteochondral defect (OCD) lesions, with encouraging preliminary results in a proof-of-concept trial and a case report of a professional rugby player.^{89,90} Likewise, a recent animal study used a chicken foot model to propose a role for PDGF as a bioinductive platform for flexor tendon repair.⁹¹ These innovative applications—including arthroscopic and soft-tissue procedures—suggest that growth factors may prove to be extremely versatile resources in the future of foot and ankle sports medicine.

SYNTHETICS

Calcium Sulfate and Calcium Phosphate

In foot and ankle surgery, calcium sulfate and calcium phosphate have long been used to augment the fixation of displaced calcaneus fractures. In a randomized prospective study of 90

patients with displaced intra-articular calcaneus fractures, percutaneous screw fixation with calcium sulfate cement grafting was associated with less blood loss, better range of motion, lower infection rate, and superior functional outcome scores compared with open reduction and internal plate fixation.⁹² A separate prospective study reported a shorter time to union for displaced intra-articular calcaneus fractures augmented with demineralized bone matrix calcium sulfate compared to those treated with open reduction and internal fixation alone.⁹³ Similarly, there is a substantial body of literature supporting the use of calcium phosphate bone cement to augment calcaneal fixation constructs. With the addition of calcium phosphate bone cement, studies have demonstrated superior compressive strength and earlier return to full weight bearing postoperatively, with comparable clinical outcome scores to conventional bone grafting.^{94–97}

Aside from their well-documented utility in calcaneus fracture fixation, calcium phosphate and calcium sulfate have also been used to improve screw purchase in a biomechanical model of osteoporotic ankles.⁹⁸ Additionally, there may be a role for calcium phosphate cement in patients with symptomatic bone cysts of the foot and ankle. In a case series of 16 young athletes with symptomatic unicameral cysts of the calcaneus, endoscopic curettage with injection of calcium phosphate bone substitute was associated with significantly improved pain and functional outcome scores, as well as an early return to play (< 8 weeks) and zero instances of recurrence or pathologic fracture at 2 years postoperatively.⁹⁹ However, given the paucity of literature investigating the use of calcium sulfate and calcium phosphate outside the realm of calcaneus fracture, further high-quality studies are needed to establish them as a mainstream therapeutic option in foot and ankle sports medicine.

Hyaluronic Acid

Hyaluronic acid (HA) injections have a well-established role in the treatment of OA. With regard to ankle (tibiotalar joint) OA, many prospective studies have reported positive results in terms of pain, functional scores, and patient-reported outcomes following HA injections.^{100–108} In a recent systematic review, Vannabouathong and colleagues pooled the results of three high-quality randomized controlled trials (109 patients) and concluded that Ankle Osteoarthritis Scale scores improved significantly with HA compared with normal saline at 6 months (mean difference of 12.47 points between groups).¹⁰⁹ Early-stage disease and shorter symptom duration have been identified as favorable prognostic factors for improvement with HA injection for ankle OA.¹¹⁰ Some data in the literature also suggest that imaging guidance may further optimize the success rate of HA injection for ankle OA.¹¹¹

In addition to their utility in the tibiotalar joint, HA injections have been investigated as a therapeutic strategy for osteoarthritis of other joints within the foot. Mei-Dan and colleagues reported significant improvements in pain, walking distance, and clinical outcomes at 7 months after a series of three HA injections were administered to the subtalar (talocalcaneal) joint.¹¹² In the metatarsophalangeal (MTP) joint, however, Munteanu and colleagues found no difference in pain scores

at 3 months following injections of HA compared with normal saline.¹¹³ Further randomized controlled studies are needed in order to draw definitive conclusions regarding the efficacy of HA injections for subtalar and MTP osteoarthritis.

Outside the realm of osteoarthritis, HA has other notable applications in foot and ankle sports medicine. Several studies have reported decreased pain and increased functional outcome scores with HA injections for OCD of the talus.^{114–116} Similarly, HA has demonstrated benefit as an adjunct to microfracture surgery that may offer improved outcomes for talar OCD compared with the operative procedure alone.^{117–119} Finally, peri-articular HA injections have also been successful in reducing pain and accelerating return to play in athletes with lateral ankle sprains.^{120,121}

Autologous Chondrocyte Implantation

Autologous chondrocyte implantation (ACI) is a two-stage surgical procedure in which chondrocytes are harvested from a patient's joint, expanded *ex vivo*, and re-implanted over an articular cartilage defect. This method was originally promoted for focal cartilage defects of the knee, but the indications for ACI and the technique itself are evolving. The newer-generation ACI technique, so-called Matrix-assisted ACI (MACI), incorporates collagen matrix or other materials with chondrocytes for implantation.¹²² The matrix supports chondrocytes with a three-dimensional structure and helps cell manipulation during surgery.

ACI has also expanded its applications to foot and ankle surgery. There is now a growing body of evidence in the foot and ankle literature to suggest that it is an effective therapeutic strategy for talar OCD.^{123,124} Classically, the chondrocytes are harvested from a nonweight-bearing portion of the ipsilateral knee or ankle, though the talar OCD cartilage itself has also proven to be a viable donor site.¹²⁵ ACI can be performed as either an open or arthroscopic procedure, with positive long-term clinical outcomes reported for both approaches.^{126–129} Long-term MRI results have also been encouraging, as the T2-mapping values of ACI-produced cartilage are compatible with normal hyaline cartilage.^{130,131} Two recent studies have investigated the relationship between MRI T2 mapping and clinical outcomes, with conflicting results reported in terms of whether these metrics may be used as a surrogate for one another in the post-operative setting.^{132,133} Despite the promising early results in support of ACI for the treatment of talar OCD, prohibitive cost and low-level evidence have limited the widespread adoption of this technique in foot and ankle sports medicine.¹³⁴

Still, the utility of ACI continues to be a source of considerable debate. First, chondrocytes lose their original phenotype during laboratory culture. From the viewpoint of cartilage biology, the dedifferentiated chondrocytes produce mechanically inferior fibrocartilage, rather than hyaline articular cartilage, which could certainly be detrimental to the outcome of ACI. Next, the incorporation of the reparative cartilage into the surrounding host cartilage is another key aspect of a functional repair product. Technical advances, including the advent of new matrices, may eventually help to address these fundamental issues in cartilage repair. Finally, it is worth noting that joints of the foot and ankle are anatomically different from the knee joint, particularly with regard to the quality and thickness of

articular cartilage. As a result, the original ACI techniques that were developed for knee surgery may need to be modified to better suit the foot and ankle. High-quality randomized controlled trials are necessary in order to establish ACI as a treatment strategy that is both successful and cost-effective for talar OCD.

Clinical Applications

Biologic therapies can be applied in both stand-alone percutaneous injection and surgical augmentation modes. The decision to choose injection rather than surgical implantation depends on many factors, notably the mechanism for dysfunction (inflammatory versus mechanical), severity and acuity of injury or disease, and healing capacity of the target tissue. The decision is also based on risk assessment and cost. In orthopedic disorders, biologics have been used to target tendon, ligament, cartilage, bone, muscle, meniscus, labrum (hip and knee), fascial tissue, and intervertebral disc. The decision to utilize allogeneic growth factors in the form of placental/amniotic material, PRP, BMC, SVF, or allogeneic cells versus expanded autologous cells in a specific indication has not been clearly elucidated. Clinical trends, based in part on published data and in part on anecdotal reports, have suggested that allogeneic growth factors or PRP may be a good first-line choice in mild-moderate soft-tissue extra-articular applications, whereas BMC or SVF may be a better option for more severe and chronic soft-tissue pathologies and intra-articular applications. Expanded autogenous cells require two interventions: one for harvesting and one for implanting after expansion and may have limited current usage for cartilage defects. Allogeneic cells do not require a surgical harvest like BMC or SVF but only provide one cell type. They may have a potential role for more severe stages of disease or for situations where the patient's own cells are inadequate due to systemic disease or exposure to certain medications.

Harvest of Bone Marrow Aspirate.

Bone marrow aspirate can be harvested from the anterior or posterior iliac crest. It can be also harvested from the proximal or distal femur, proximal or distal tibia, or calcaneus. Typically, the author, Lew C. Schon, MD, uses the following protocol to harvest a high yield of cells from the anterior iliac crest. The crest is selected as it has the proper cell density and volume and is relatively safe to approach due to palpable landmarks. The patient is positioned supine on the operating room table. Anesthesia or sedation is used. The lower extremity and pelvis are prepped and draped in standard sterile fashion. The local area 3–7 cm proximal to the anterior-superior iliac spine is blocked with 0.5% Marcaine and 1% lidocaine without epinephrine. A 2 mm incision is made 3–4 cm posterior to the anterior-superior iliac spine. The trocar trephine device with a 2 mm central bore is inserted. Bone marrow is harvested in 5 mL aliquots via multiple trajectories from the iliac crest. The obtained bone marrow is then concentrated for 15 minutes using a specialized commercially available chamber and centrifugation system (e.g., Spinesmith or Biomet). BMC is then delivered back onto the surgical field and applied via injection to the site of disease. The iliac crest incision is irrigated. 4-0 Monocryl is used to close the skin. A simple dressing is applied. The patient is reversed from anesthesia and allowed to recover under monitored conditions.

Posttreatment Protocol for BMC or PRP Injection

After BMC injection, patients are instructed to rest the leg and minimize weight bearing. A boot brace and ambulatory aids (walker, crutches, knee scooter, or cane) are used. Surgical dressings are kept in place until follow-up in 2 weeks. The patients are given a small prescription for oxycodone and instructed to take acetaminophen to control their pain. After the 2 week follow-up, patients are allowed to ambulate with or without the boot brace, according to tolerance, but advised to maintain minimal activity until follow-up at 6 weeks.

Percutaneous Applications: Foot and Ankle

Tendinopathy/Soft Tissue

A growing body of peer-reviewed evidence is available regarding use of PRP in tendon pathology. This includes the first Level 1 data generated from a study of PRP versus placebo in chronic lateral epicondylitis, demonstrating significant and sustained improvements at 2 years posttherapy.¹³⁵ Results of published clinical data have been mixed but suggest a general positive trend toward efficacy in percutaneously delivered therapies for treating soft-tissue pathology. Optimal volumes, periodicity of repeat interventions, and utility of adjunctive techniques like needle fenestration and postprocedure rehab protocols remain largely undetermined.

Acute Versus Chronic Pathology. While commonalities exist regarding origins of pain and dysfunction between acute and chronic pathologies, enough differentiators exist to consider different approaches from the perspective of biologic therapies. In acute conditions, disruption at the connective tissue and cellular levels with associated rapid activation of the inflammatory cascade results in an effort to initiate repair. Thus, biologic approaches, depending on the tissue involved, should focus on supporting and augmenting the natural processes set in motion. Healing occurs in phases and application of healing elements may augment more efficient progression through the healing phases, which sometimes can be overwhelmed by the injury severity, tissue disruption, and perfusion to the tissue involved. High cellularity in acute injury is typically present, suggesting a potential role for acellular (indirect) approaches like PRP as initial therapeutic considerations.

With chronicity there is fibrosis, loss of intrinsic tissue integrity, abnormal biomechanics, and typically chronic inflammation. Healing capacity and progression are halted with a subsequent shift from healing to scarring. A theoretical biologic approach would look to re-initiate and amplify healing mechanisms, providing the cellular and protein building blocks that are rendered inaccessible to the damaged tissue due to fibrosis. Remodeling of the tissue with re-establishment of perfusion and/or alternative supportive pathways to maintain integrity would reactivate the healing process. Considering challenges with innate cellular migration to the pathologic tissue, cell-based biologic approaches (direct, i.e., BMC) have therapeutic value in jump-starting local remodeling to treat chronic pathology.

Plantar Fasciitis. Plantar fasciitis (PF) is a challenging soft-tissue disorder, often suboptimally responsive to conservative management. The location of the PF can be targeted percutaneously by palpation or with imaging studies such as

fluoroscopy, MRI, and/or ultrasound. In acute or subacute manifestations, PRP infiltration into the intra-substance of the PF near the insertion into inferomedial calcaneus represents an early-intervention treatment option for a process that often becomes chronic and more refractory to treatment. Using a 22-gauge needle, 3–10 cc of injectate can be implanted at the insertion and just distal to insertion if pathology is noted to extend into this area. Often 3–6 punctures are made to cover a large area of fascia. Through one skin puncture, multiple trajectories (4–6) into the affected tissues can be performed. After each needle withdrawal from the skin, a finger is used to apply local pressure and avoid back flow of injectate. Care must be taken not to penetrate the plantar fat pad in the area of weight bearing. Also, avoiding the tibial nerve and its branches—specifically the first branch of the lateral plantar nerve, the lateral plantar nerve, and the calcaneal nerves—is important to decrease the risk of neuralgia or dysesthesias. Procedural and postprocedural pain control is required for administration in an outpatient setting.

Considering the load-bearing nature of the structure, caution is warranted with needle fenestration in addition to biologic implantation. Thus, post-procedure, the patient is requested to be nonweight bearing with use of cane, crutches, knee scooter, and/or brace. For more chronic, postop, or scarred presentations, serial rounds of PRP spaced several months apart could be considered versus BMC infiltration. Postop minimal-weight-bearing protocol is used for 2 weeks followed by protected activity for 2–6 weeks. The patient is instructed that the pain may be flared for 6 weeks following the injection. Optimized post-implantation care and rehab protocols to augment the treatment have not been well established.¹³⁶

Achilles Tendon. Achilles tendon pathologies are a common but challenging problem to address with purely conservative measures. Research on PRP infiltration for chronic Achilles tendinopathy has yielded mixed results.¹³⁷ Variability in clinical study protocols, including implantation and post-procedure care standpoint, further serve to complicate identifying a useful treatment protocol. From a clinical perspective, refractory nonsurgical Achilles pathologies are good candidates for application of biologics. Patients with elongated tendons from a missed rupture are not good candidates as they need surgical tightening and or grafting with surgical reconstructions.

Consistent with general approaches to subacute versus chronic tendon conditions, for subacute problems, allogeneic growth factors or PRP are easy to use and have a low potential for harm, making them reasonable options with ultrasound guidance and intra-substance placement. Considering the size and tension-bearing nature of the structure, the authors suggest using a 22- or 25-gauge needle for penetrating the tendon. The authors also recommend placement of multiple small aliquots of injectate at the borders and within the region of injury/pathology, as opposed to a single higher-volume injection through a single needle, creating a fluid void and raising concern for excessive intra-substance pressure. Special mention is made to consider co-treatment of the retrocalcaneal bursa, which often becomes inflamed and fibrotic, in combination with chronic morbidity of the tendon. Using a medial approach to the tendon avoids inadvertent penetration of

the sural nerve, which is lateral to the tendon in the distal quarter of the leg and travels from a lateral position to a central posterior position in the third quarter of the leg.

For more chronic, postop, or scarred presentations, serial rounds of PRP spaced several months apart could be considered versus BMC infiltration. Optimized post-implantation care and rehab protocols to augment the treatment have not been well established.¹³⁷

When using BMC, after harvest and concentration, it is sterilely injected through five different punctures with 5–7 trajectories per puncture in multiple directions, avoiding the sural nerve going from the medial side to posterior and to lateral along the zone of maximal tenderness, which is marked prior to surgery. The sites are manually compressed to minimize leaking out of the BMC after injection. Postop minimal-weight-bearing protocol is used for 2 weeks followed by protected activity for 2–6 weeks. The patient is instructed that the pain may be flared for 6 weeks following the injection.

Smaller tendon and ligamentous pathologies involving foot and ankle are common, and include: peroneal tendons, anterior tibialis, posterior tibialis, digital tendons, and medial and lateral collateral ligaments. Natural healing with standard conservative modalities can occur without intervention depending on the injury and chronicity. Those structures that are completely disrupted or stretched beyond functional limits are not good candidates for biologic treatments except as a supplement to surgical repair or reconstruction. A thorough history, physical, and appropriate imaging studies are mandatory to determine the extent of tissue damage and the prognosis for recovery.

Studies suggesting efficacy of PRP applied via intra-tendinous injection reveal a potential role for PRP in pathology involving these structures.¹³⁵ All these structures are relatively accessible and are low-risk interventions when targeted with ultrasound-guided intervention and utilizing meticulous technique. Attention to anatomical relationships with associated neurovascular structures and care to avoid needle trauma to such structures is notable to minimize potential for procedural complications.¹³⁸

Osteoarthritis. Limitations of valid therapeutic options for arthritis remain a source of frustration for the musculoskeletal provider. Many factors including trauma and overuse result in joint instability, tendon pathology, and abnormal biomechanics that contribute to the development of the condition. Treatment options to alter the course of the disease process have been limited, and mostly involve surgical reconstruction to improve alignment, stability, and tendon function. Debridement of arthritic lesions with or without application of orthobiologic materials (BMC, PRP, bone grafts, chondrocytes, synthetics) is being studied. Ultimately joint replacement or fusion may be the only alternatives.

It has been suggested that intra-articular and/or subchondral applications of biologic therapeutics represent potential options as both a stand-alone treatment and an adjunct to surgery. There are limited published data for such applications pertaining to treating OA in joints of the foot and ankle. Limited, small-scaled studies have suggested potential utility of PRP and BMC for intra-articular pathology, mostly involving hip and knee joints.¹³⁹

Intra-articular injection of HA for ankle OA has been investigated in small cohorts, using varied protocols and follow-up, and demonstrated contradictory results.^{103,140}

There are applications of intra-articular injection of HA for OCD of the talus.^{114,115} In general, HA improved the ankle function, as assessed with the American Orthopaedic Foot and Ankle Society's Ankle-Hindfoot Scale, and relieved pain, as measured with VAS scores. However, there was no direct or indirect evidence that HA facilitated the healing of OCD lesion. Treatments may have an effect by stabilizing chondral lesions, treating subchondral bone reaction or on the synovium.

Techniques of intra-articular injection have been described.¹⁴¹

The tibiotalar joint is accessible via palpation, ultrasonographic, or fluoroscopic approaches. The author, Lew C. Schon, MD, accesses the ankle joint via a medial approach in the Hardy notch, medial to the anterior tibial tendon. The author, David Karli, MD, uses an anterior approach, medial or lateral to the dorsalis pedis artery and nerve bundle. A 22- or 25-gauge needle can be advanced through the joint capsule into the anterior joint recess, maintaining care to avoid any direct needle trauma to the articular surfaces. Typical volume of PRP or BMC infiltrated into the joint is 5–12 mL.

Subchondral applications of BMC for more advanced degenerative OA, particularly with evidence of bony edematous changes or erosion noted on radiographic workup is under study as an alternative to, or in conjunction with, intra-articular implantation. This intraosseous injection is performed in the operating room. Injection of calcium phosphate can also be performed for these lesions in the operating room (see Section: Treatment Option for the Management of Chronic Bone Marrow Lesions with Calcium Triphosphate).

The subtalar joint can be accessed via lateral oblique approach through the sinus tarsi, or less commonly posteriorly, lateral to the Achilles tendon. The joint is irregular and typically can hold 4–10 mL of a biologic preparation.

The talonavicular (TN) joint is narrow and low volume but can be accessed via a medial approach by palpating the navicular and injecting proximally in to the depression that exists at the TN joint with a small (25-G) needle. Ultrasound or fluoroscopic guidance is recommended. Target volumes typically are limited to 3–6 mL.

The metatarsal cuneiform (MTC) joints can be injected via a dorsal approach adjacent to tendons and nerves. Ultrasound or x-ray guidance can be helpful, and 3–6 mL of injectate can be administered.

The first MTP joint can be accessed via guided dorsal approach adjacent to the extensor hallucis longus tendon with a small gauge needle. Small injectates of 0.5–2 mL can be accommodated.

Surgical Implantation.

From a surgical perspective, potential benefits of biologics would be in promoting efficient and complete tissue recovery following surgical repair, control of inflammation postop, recruitment of progenitor cells and macrophages to the operative site, angiogenesis, hemostasis, and, with the exception of leukocyte reduced PRP, potential for antibacterial effect.¹⁴² Timing of application related to surgical augmentation is still not well defined, with two strategies utilized in the field to date.

For application at point of care, there are arthroscopic and open uses. In arthroscopic procedures, following completion, excess arthroscopy fluid is drained from the joint and the portals are closed. The biologic preparation is infiltrated once closure is complete to avoid egress of the fluid. In an open procedure, the biologic preparation can be infiltrated into target tissue during or following repair. Biologic preparations can be converted into an autologous fibrin matrix by reversing anticoagulation in the sample for physical implantation or infiltrating the final preparation into target tissue by bathing or injecting in and around the repair, debridement, and/or implant. The material can also be combined with a demineralized bone graft or with a synthetic calcium carrier and placed into a defect or zone of desired union in the bone or soft tissue.

Some physicians prefer application percutaneously following surgical closure with a lag time to allow the immediate postop environment to stabilize. Some surgeons are electing to wait days to weeks following surgical repair to deliver biologic preparations in an effort to optimize the potential stimulation and effect. Two primary rationales drive this preference. Residual joint fluid immediately following arthroscopy may dilute the potency/concentration of the biologic in the treatment site. There is already a good deal of anabolic stimulation in a freshly repaired or debrided tissue. To prolong that stimulation, the biologic can be implanted at a later date. The optimal timing of a biologic application following surgical intervention has yet to be determined. It is also unclear if a single application or serial applications creates the greatest potential for tissue healing in a surgical setting. More research is needed to optimize this strategy.

Soft-Tissue Repair: Potential Applications.

Three options exist for a soft-tissue repair. In the first option, the repair can be bathed with a biologic preparation at the time of repair. With the second option, the biologic can be injected into the repair prior to or at closure (the author, Lew C. Schon, MD, prefers method). Finally, the implant can be used with an activated biologic (requires reversal of anticoagulation of the biologic preparation at implantation) into the tissue at the time of repair. These techniques can be used for repair, debridement, or reconstruction of the tendons (peroneal, Achilles, posterior/anterior tibial tendons) or with medial or lateral ligamentous repair or reconstruction.

Chondral/Subchondral Application.

The intent of the chondral/subchondral application is to increase longevity of the cartilage repair, prevent progression of the degenerative process, increase aggrecan content, stimulate chondrogenesis, and minimize formation of fibrocartilage with microfracture, with osteoarticular transfer system procedures, with allograft tissue, or in association with scaffold applications. The addition of BMC to bone marrow stimulation techniques (BMS) such as microfracture showed superiority over BMS alone.¹⁴³ In general, there are beneficial outcomes for biologic and BMS treatment of moderately sized chondral defects without any reports of serious complications. Rates of return to high-level activity and MRI documentation of good filling of cartilage defects have been described.¹⁴⁴ Favorable outcomes in short- to medium-term reports for BMC-augmented treatment

of knee OA have been published.¹⁴⁵ Ilas et al. have described subchondral implantation of BMC for percutaneous treatment modeling in cartilage degeneration.¹⁴⁶ At the time of surgery, the biologic can be mixed with autograft, allograft, synthetics, or collagen carriers and packed into the lesion. Another option is to inject the biologic into the joint once the wound is closed.

Fusion, Fracture, or Nonunion Augmentation. Any fusion of the foot and ankle can be supplemented with biologics.¹⁴⁷ Also, high-risk fractures or nonunions can be repaired or reconstructed with augmentation by biologics. At the time of surgery, the biologic can be mixed with autograft, allograft, synthetics, or collagen carriers and packed in and around the fusion site. Another option is to inject the biologic into and around the site once the wound is closed. In some cases where there is good alignment and stability of the fracture or nonunion, percutaneous treatment may be considered. Hernigou treated stable nonunions of the tibia successfully with percutaneous BMC treatment.¹⁴⁸

TREATMENT OPTION FOR THE MANAGEMENT OF CHRONIC BONE MARROW LESIONS WITH CALCIUM TRIPHOSPHATE

The use of intraosseous-injected calcium triphosphate is performed for the treatment of patients with chronic trabecular fractures (bone marrow lesions [BML]). The term “subchondroplasty” is a company trade name (Zimmer Biomet, Warsaw, Indiana) applied to a procedure which has been in use for several years. It was originally developed for patients with OA of the knee, where there is scientific literature to support the association of pain from OA and bone marrow “edema” as seen on MRI. However, the diagnosis and treatment principles surrounding this technique have been around for decades and can now be applied to a variety of entities outside the knee. The procedure consists of core decompression and the insertion of an injectable hard-setting bone substitute material (calcium triphosphate) as internal fixation to treat BMLs in the subchondral bone. The material injected needs to be fairly viscous, as many of these lesions are not cystic in character. The application is analogous to the vertebroplasty/kyphoplasty philosophy and techniques of the compromised vertebral body of the lumbar spine. This approach provides an effective, minimally invasive option for the clinical management of chronic, painful BMLs as an alternative to more invasive techniques, thus attractive to the athlete with performance-limiting issues yet time constraints.

Patient Presentation/Indications

The typical patient may be an athletic individual who presents with localized pain in the foot or ankle of greater than three-month duration. Their symptoms have failed to improve with rest, antiinflammatory pain medications, and activity modification. Their function is limited with compromise of athletic activity involvement and performance. The initial evaluation and physical examination are often unremarkable, specifically without soft-tissue abnormalities, restricted motion, or instability.

Radiographs of the affected area often fail to demonstrate any obvious fracture or joint disorder and are generally interpreted as normal. In this situation where significant symptoms and dysfunction have persisted despite extensive nonoperative

care, and radiographs are not explanatory of the clinical situation, a diagnostic MRI is felt appropriate. As opposed to standard radiographs, an MRI can assess soft-tissue injury, can quantitate inflammation, and can demonstrate bone injury including BMLs. While these BMLs may be juxta-articular and associated with arthritis, many are intraosseous and result from stress/overuse, osteochondral lesions, and direct trauma with bony contusion.

Interpreting the MRI and Bone Marrow Lesions

A BML is best visualized on fat-suppressed MRI sequences, often labeled as T2, and appears as a white hazy signal (Fig. 29.1).

Radiologists may refer to a BML as an “osteochondral fracture,” “subchondral fracture,” “stress fracture or reaction,” “insufficiency fracture,” “microtrabecular fracture,” or “bone edema.” Histopathologists have identified the white signal apparent on an MRI as the body’s response to a trabecular fracture. Numerous histopathologic, morphologic, and histochemical investigations of bone marrow lesions have been published. This research has consistently demonstrated that subchondral bone in the region of a chronic BML is substantially altered from healthy, normal subchondral bone. When analyzed, subchondral bone affected by the BML demonstrates areas of trabecular compromise with fracture, bone marrow necrosis, and osteoblastic activity consistent with attempted fracture healing and regions where subchondral bone has been replaced by non-osseous elements reflecting nonhealing of bone fracture.^{149–151}

Replacement of normal subchondral bone with nonosseous bone essentially creates a bone void region, structurally compromises the support of that bone, and may lead to an environment in which normal subchondral bone will not spontaneously replace nonosseous tissue. Burr et al. explained that BMLs involve high bone turnover, microcracks, and increased bone mineral density similar in appearance to that seen with a fracture nonunion.¹⁵²



Fig. 29.1 Sagittal T2-weighted MRI image of a foot, highlighting the bone marrow abnormality in the talar body.

As mentioned previously, chronic BMLs can occur with direct trauma or in association with excessive stress or adjacent joint deterioration. Focal joint injury, as seen with articular cartilage damage and thinning, often in association with ligament injury with instability, can increase and focalize stress on the joint articular surface and create underlying bone compromise with inflammation. Chronic, increased stress on the subchondral bone may lead to microfractures and, in response, increased bone healing activity. When this stress exceeds the patient's ability to repair the resulting damage, a chronic BML results and is indicative of a stress reaction or fracture, with insufficiency of the bone. Quoting Erikson et al., "bone marrow lesions have been demonstrated in a wide variety of lesions in bone, but the common denominator for these conditions is some kind of injury to the bone and bone marrow through mechanical stress, inflammation, or ischemia. A common denominator for these lesions seems to be cortical or trabecular bone defects or micro-trauma."¹⁵³ An important distinction is that these bone lesions of the athlete are unrelated to osteoporosis. Both Burr and Lo have described the body's response to the damaged area as "thickening" and actually creating more bone density.^{152,154}

Clinical Effect of Bone Marrow Lesions

Numerous large-scale MRI studies have greatly contributed to knowledge of the significance of BMLs and their relationship to progressive bone and joint issues. Significantly, the presence of a BML has been shown to be highly predictive of patient-reported pain. While synovitis or cartilage loss may be present in a patient with chronic BMLs, those conditions are not the cause of the

underlying pain given the lack of pain fibers in those structures. Extrapolating from Cicuttini and the knee literature, a study of 250 patients (500 knees) found that "the presence or absence of joint space narrowing...was not significantly associated with knee pain."¹⁵⁵ This finding was confirmed by Hunter et al., who in a review of 243 papers for OA biomarkers found that pain is strongly related to the large bone marrow lesions, moderately related to synovitis and effusion, and only weakly related to cartilage volume and thickness.¹⁵⁶ The correlation between the patient's pain and the presence of BMLs has been shown in several large studies as highlighted in Table 29.1. There has been similar support in the foot and ankle literature.¹⁵⁷⁻¹⁶⁰

Zanetti states that the edema-like bone marrow abnormalities of the foot are clinically relevant and predictive of long-lasting pain. Similarly, Rios and his colleagues found that the painful BMLs seen in the foot and ankle are secondary to bone impaction or contusion and related to trabecular microfractures or reaction after stress.

Managing the Chronic (Nonhealing) Bone Marrow Lesion of the Foot and Ankle

Some BMLs, particularly in the younger athletic population who have joints with little deterioration or chondral loss, may heal with nonsurgical intervention. However, the BMLs that persist greater than 6 months and remain painful with functional limitation may be candidates for a subchondroplasty procedure. There are numerous applications for the procedure in the foot and ankle. Most common are bone marrow lesions of the talus, underlying an osteochondral defect or abnormality. There may

TABLE 29.1 Studies Correlating Bone Marrow Lesions (BML) and Pain

Authors	No. of Patients	Results	Conclusions
Felson et al. ¹⁶⁴	401 (351 with pain, 50 without pain)	BML found in 77.5% of patients with pain; BML found in 30% of patients with no pain	"Bone Marrow Lesions on MRI are strongly associated with the presence of pain in knee OA."
Felson et al. ¹⁶⁵	330 (110 case; 220 control) Case – initially no pain progressed to pain Control – initially no pain	49.1% of case patients showed an increase in BML score compared to 26.8% of control	"Development of knee pain is associated with an increase in BML as revealed on MRI."
Hayes et al. ¹⁶⁶	117 women (232 knees)	Large BML lesions were common in the pain and OA of the knee group; this group was significantly more likely to have defects of cartilage, meniscal tears, osteophytes, subchondral cysts, sclerosis, joint effusion, and synovitis	"In middle-aged women, there were significant associations between pain, radiographic severity of OA of the knee, and seven MR imaging-identified parameters [including BML]."
Zhai et al. ¹⁶⁷	500 adults (239 with knee pain)	Prevalent knee pain was significantly associated with...bone marrow lesions (OR 1.44, 95% CI; 1.04–2.00 per compartment)	"Knee pain in older adults is independently associated with both full and non-full-thickness medial tibial chondral defects, bone marrow lesions, greater BMI, and lower knee extension strength, but is not associated with radiographic knee OA."
Sowers et al. ¹⁶⁸	363 patients (724 knees)	The prevalence of bone marrow lesions in the medial, lateral, and patellofemoral compartments were 21.3% (154 knees), 13.4% (97 knees), and 45.4% (329 knees), respectively. The Kellgren–Lawrence scores were highly correlated with bone marrow lesions in the medial femoral and tibial compartments	"Large bone marrow lesions in the medial femoral condyle or the medial or lateral plateau were associated with substantially increased odds of reported pain" "BML in medial compartments was associated with 'marked decreases in walking and stair-climbing performance.'"
Zhang et al. ¹⁶⁹	1042 patients		"The resolution of knee pain was associated with shrinkage or resolution of bone marrow lesions."

or may not be a cystic component to the lesion. These lesions are often situated in the medial quadrant of the talus and accessible from an entry point in the lateral talar process. (Fig. 29.2)

Perhaps the most intriguing role for this procedure is a recalcitrant stress reaction or fracture of the cuboid or cuneiform in which there have been very few options for treatment. In this scenario, a surgeon can utilize this technique to provide a *biologic* internal fixation to stabilize and heal the microtrabecular fracture evident by the BML. While a formal open internal fixation procedure with plate and screws has been described as an option for these recalcitrant stress fractures and reactions, it is a more invasive technique than necessary for a microtrabecular fracture that is often without cortical compromise¹⁶¹ (Fig. 29.3).

The idea of injecting a bone graft substitute to strengthen compromised bone is not new. Russell et al. in a prospective, randomized trial of 119 subjects showed the effectiveness and healing capabilities of using a hard-setting calcium phosphate bone filler to treat tibial plateau fractures with superior results compared with autograft.¹⁶² The product described by Russell was enhanced and developed into one that could be injected with flow into trabecular bone canals, leaving the cortical shell intact. Ideally, the material should set hard via an endothermic

response and carry the same properties as natural trabecular bone.¹⁶³ The theory is that it will stabilize the area of the microfracture, providing fairly rapid pain relief and also will eventually remodel into host bone to heal the lesion or defect.

Technique

The subchondroplasty® procedure is a minimally invasive surgery performed on an outpatient basis that accesses and fills chronic subchondral defects, intraosseous lesions, and other symptomatic bone marrow lesions. While this procedure can be used to address cystic lesions of the foot and ankle, the BMLs are often noncystic, and therefore a viscous form of the injectable mineral is paramount. During the procedure, the surgeon will rely heavily on fluoroscopy, arthroscopy, and mini-open techniques to target a small, drillable cannula (11- or 15-gauge) into the area of the bony concern as seen on the MRI. Both T1- and T2-weighted images assist with precise targeting, utilizing axial/coronal/sagittal views. The limb being addressed is placed on a bump or blanket elevation to assist with clear intraoperative fluoroscopic imaging. Care is taken to avoid numerous errant passes into the lesion, or more than one cortical entry site, as appropriate filling is compromised, and extravasation

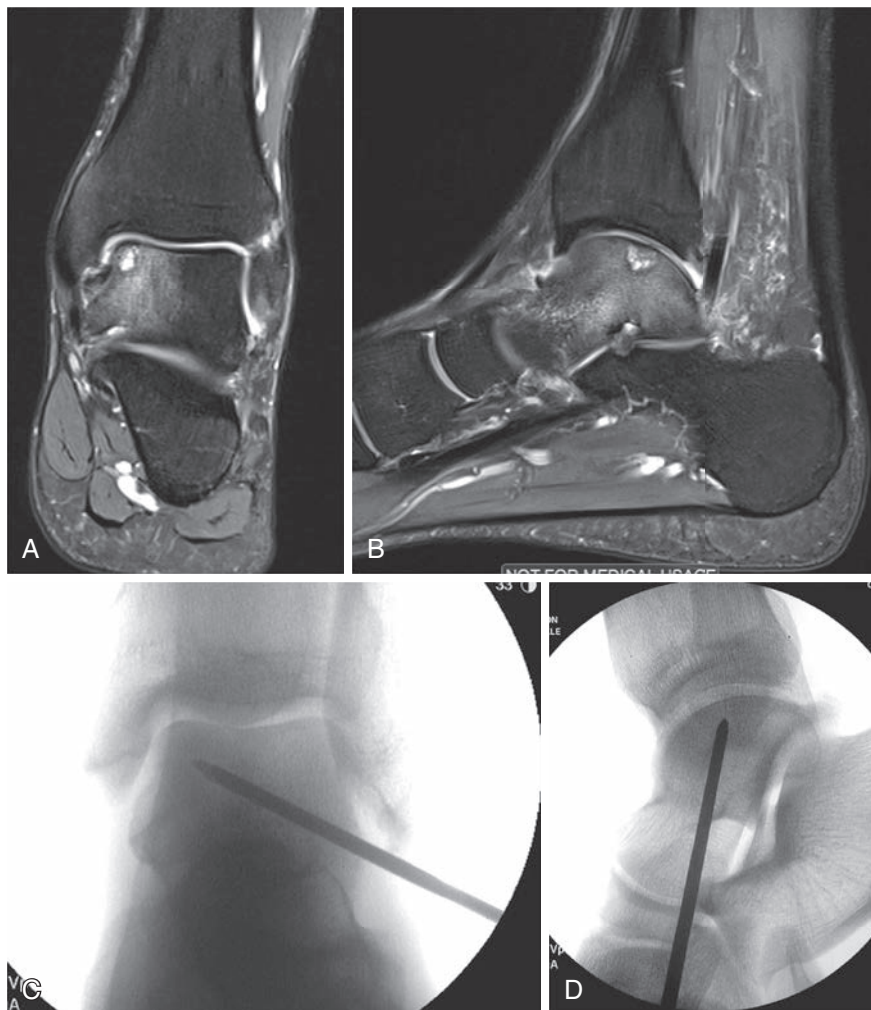


Fig. 29.2 T2-weighted MRI, (A) coronal and (B) sagittal images noting an osteochondral lesion in the medial talar dome. Intraoperative fluoroscopic (C) anterior-posterior and (D) lateral views of the ankle illustrating the placement of the drilling trochar into the talar lesion, entering from the lateral talus.

may occur. Following the core decompression, the surgeon will deliver a precise amount of the bone graft substitute into the lesion where it hardens endothermically to a material with properties that mimic cancellous bone. The material is radiopaque and can often be seen on the fluoroscan imaging. As mentioned previously, the calcium phosphate used is eventually resorbed and is replaced with new bone, a process that may require 2–3 years depending on the bone injected. In theory, the body recognizes the material as normal bone and replaces it with new bone through a cell-mediated remodeling process.¹⁶³

While this is a minimally invasive procedure with an expected low risk of complications and a short postoperative recovery period, it is important that the surgeon appreciates the location of neighboring neurovascular structures and minimizes extravasation into any adjacent joint or soft tissues. When extravasation is recognized, the material can be fully irrigated and removed via arthroscopy or direct access. Most important is to not inject an excessive amount of material into the lesion or bone itself. Most lesions of the foot and ankle only require 0.5–1.5 cc of volume, depending on the size of the bone and the lesion itself. Excessive filling of the bone may lead to trabecular congestion and vascular compromise, and subsequent avascular necrosis. One bone of obvious concern is the navicular, where this procedure may be contraindicated due to its already tenuous blood supply.

Postoperative management is dependent on the location of the BML undergoing the injection technique, associated

disorders, or concurrent surgical procedures such as arthroscopy. In general, nonweight bearing is instituted until the pain associated with the procedure dissipates. This averages 48 hours. In the case of a stable stress reaction, patients may bear weight by 2–3 days and return to sport in 10–14 days, as symptoms allow. X-rays and CT may be used to follow bone healing. Postoperative MRIs are not helpful given the artifact produced by the calcium phosphate material injected. Given that it takes 2–3 years for bone turnover and remodeling to occur, the MRI may remain difficult to interpret for some time.

Chronic BMLs can arise from a number of etiologies in the foot and ankle and are often a source of debilitating pain in the athlete, affecting his or her performance. Athletes can suffer from chronic stress reactions and fractures, as well as periarticular bone lesions associated with chondral loss for which there are few treatment options available. The literature supports the idea that many of these lesions identified on MRI result from a trabecular fracture in varying stages, and often prior to cortical disruption. In orthopaedics, the standard for bone fractures is to stabilize and create a biologic environment conducive for healing. A subchondroplasty procedure employing the placement of an injectable, hard-setting calcium phosphate into the BML provides a minimally invasive, joint preserving, and clinically beneficial treatment for patients who fail to heal with nonoperative means.

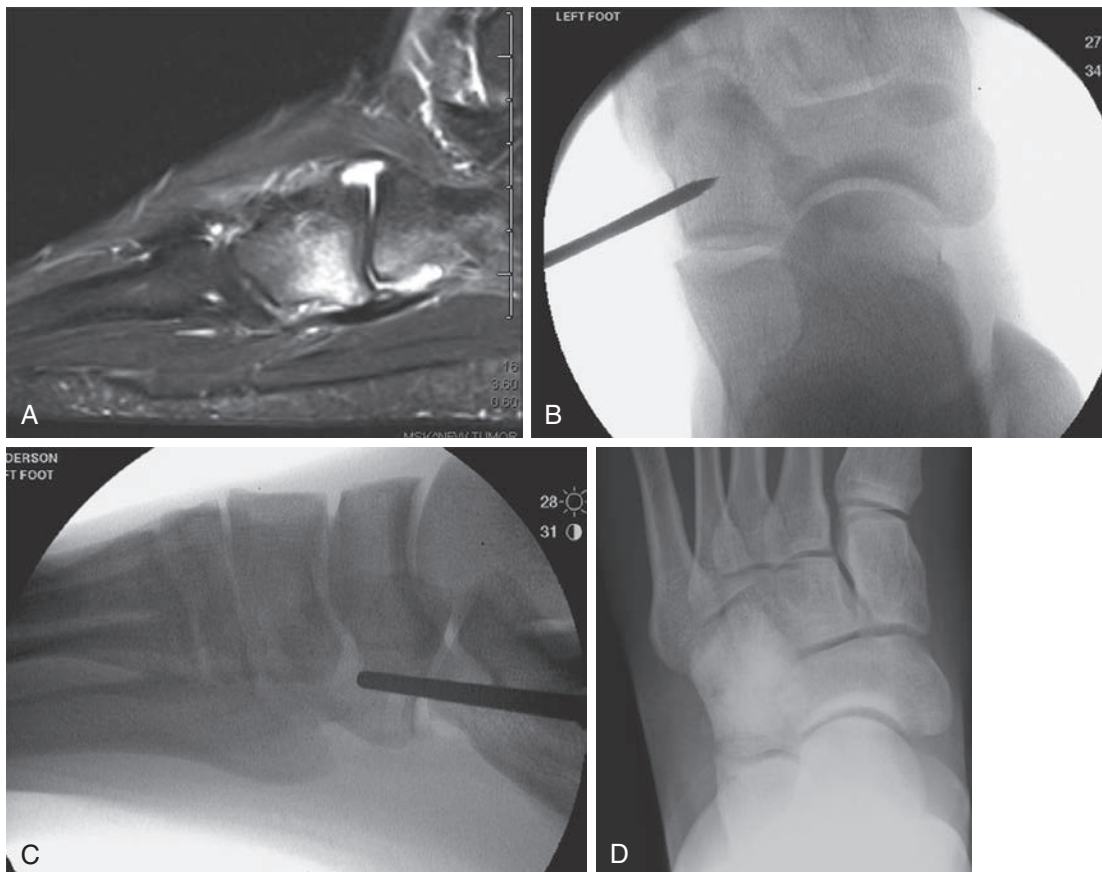


Fig. 29.3 (A) Sagittal T2 MRI image of the foot of a 23-year-old athlete with chronic lateral midfoot pain. Bone marrow lesions are noted in the cuboid and consistent with a stress reaction. Intraoperative fluoroscopic (B) anterior-posterior and (C) lateral images noting placement of the cannulated trochar into the cuboid. (D) Standing anterior-posterior radiograph performed 4 weeks after injection of 1 cc of calcium phosphate into the cuboid.

FUTURE DIRECTIONS

Building on the platform of quantitatively controlled biologics creates opportunity to further understand the science and improve clinical outcomes. Establishing validity beyond theory and potential will become increasingly important to define where such preparations have the greatest utility and how to optimize therapeutic potential. Optimizing biologic compositions specific to indication and individual represents a logical progression of the field by characterizing and quantifying biologic preparations to establish dose–response trends from which validation can occur. Standards and training pathways for best-practice techniques for biologic implantation in both surgical and percutaneous applications are limited. Opportunity in residency and fellowship training programs are sparse, creating the need for formal resources within industry and academia to fill this void to frame the basis of best-practice strategies.

In addition, establishing patient screening methods to aid in predicting probability of therapeutic success would have an impact on patient counseling, treatment decision making, and customization of biologic preparations. Opportunity to develop predictive screening methods depends on successful implementation of protocols to provide detailed information on the cellularity of biologic preparations, and track clinical outcomes to identify clinically relevant parameters with the potential for predictive value.

In the future, we may see the integration of synthetic or biologic scaffold materials and cellular therapies. Products that optimize delivery of the biologics and maintain them in the location while performing their therapeutic role may be developed. For example, the Stem Cell Suture (Bioactive Surgical, Inc., Clarksville, Maryland) is an MMSC bearing-suture for tendon or ligament repair that may have surgical advantages. One study by the senior author, Lew C. Schon, MD, showed increased biomechanical strength and significantly increased ultimate failure strength with the sutures loaded with MMSCs versus those without imbedded cells or no cells. The MMSCs seem to remain locally at the repair site and enhance the histologic repair quality of the tendon collagen.¹⁷⁰ Other products offer the ability to inject the biologic more efficiently. One such product is the Transplug (Bioactive Surgical, Inc., Clarksville, Maryland), which permits the intraosseous injection into the lesion or nonunion.

Advances in autogenous (PRP, BMC, and SVF), allogeneic materials (e.g., placental/amniotic tissue–derived fluid and membranes), recombinant growth factors, cellular-based therapies, and synthetics will continue. Techniques to deliver these more effectively will be developed and supported by lab- and clinic-based studies. The best practices will evolve and enhance our mechanical approach to most conditions. Ultimately the goal will be faster, more complete, more reliable healing with fewer complications to optimize the return to robust activity.

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Rehabilitation of Specific Foot and Ankle Issues

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OUTLINE

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INTRODUCTION

Injuries to the foot and ankle are some of the most common injuries in sport. These injuries can significantly affect the performance of an athlete and can often lead to disability and missed playing time. In a series of over 1600 athletic injuries, Garrick and Requa reported that more than 25% of the injuries occurred in the foot and ankle.¹ More recently, Hunt et al. reported on over 3800 musculoskeletal injuries in NCAA Division 1 athletics. Foot and ankle injuries made up 27% of those injuries, with over one-fifth of those resulting in time loss from sport. The average length of time missed was over 12 days.² These injuries are some of the most common injuries leading to time loss in sport.³⁻⁷ This missed time has led to advances in rehabilitation over the past 20 years aimed at reducing injury time, improving outcomes, and preventing the injuries from becoming chronic.

Athletes provide unique challenges in rehabilitation because of the significant demands required to participate in sports. The foot and ankle are especially vulnerable to these demands. It serves as the connection of the body to the weight-bearing surface. As a result, the foot and ankle are designed to allow efficient energy expenditure in both stance and locomotion. The foot and ankle are a small area of the body with specialized function and intricate muscle, tendon, ligament, and bone interactions designed to meet the strenuous demands of weight bearing and ambulation. Recent technologic and procedural advances contribute greatly to the treatment of the

competitive athlete. The principles of rehabilitation must continue to develop with the continuing advances made through improving technologies. An appropriate and advanced rehabilitation can provide the athlete with a full, complete, and functional recovery.

PROTECTION, REST, ICE, COMPRESSION, AND ELEVATION

Initial treatment of acute foot and ankle injuries and postoperative pain remains protection, rest, ice, compression, and elevation (PRICE) therapy despite any high-quality evidence that it works better than placebo.⁸ Protection involves any intervention aiming to prevent re-injury to the foot and ankle. Rest involves offloading the affected area to decrease the stress on the injured tissues. Ice, compression, and elevation are all methods to control edema and pain.

There are many common devices to aid in the protection of the healing tissues about the foot and ankle. These consist of elastic wrapping, taping/strapping, semi-rigid pneumatic ankle brace, non-rigid functional ankle brace, and the author's preferred method, a removable walking boot (Fig. 30.1). The device allows patients to weight bear immediately, work on range of motion (ROM) by removing the boot, and use a continuous cold/compression device. Once the ankle has healed, a more functional brace is used for return to activity (2–4 weeks after injury). We particularly stress the use of the boot at night for the first 3 to 4 weeks to keep the foot and ankle complex in a



Fig. 30.1 Walking Boots of various sizes and types.

90-degree dorsiflexion position during sleep, when the relaxation of muscular control and the forces on the heel passively place the complex in a plantar-flexed and inverted position. The rigid boot counteracts this position.

There are several cold agents to choose from, including the reusable cold pack, ice bags, cold whirlpool, ice immersion, and pneumatic cold compressive devices (Fig. 30.2). The primary objective of ice is to reduce swelling and help manage pain. Physiologically, cryotherapy acts through several mechanisms of action. The application of cold agents results in arteriolar vasoconstriction, thus decreasing local metabolism, oxygen utilization, and inflammation.⁹ Applying an ice pack to the knee for 20 minutes has been shown to decrease soft tissue blood flow 26% and bone uptake (reflecting changes in blood flow and metabolism) 19%.¹⁰ It has been found that cold therapy increases the pain threshold while decreasing the nerve conduction velocity.¹¹ Further, as the temperature decreases, there is a corresponding decrease in sensory and motor nerve velocity, eventually causing synaptic transmission to be blocked.¹²

Cryotherapy has been validated as an effective analgesic in many surgical fields including plastic surgery,¹³ otolaryngology,¹⁴ obstetrics and gynecology,¹⁵ and maxillofacial surgery.¹⁶ The application of cold is most effective immediately after injury. Hocutt et al. found that patients with grade III ankle sprains that were treated with ice in the first day returned to functional activities such as running and jumping after 6 days, whereas those treated on the second day went 11 days before they could run or jump.¹⁷ In contrast, those who received heat in the first day had a recovery time of 14.8 days. Cote et al. showed that cold therapy alone in the first 3 days after a severe ankle sprain limited the foot and ankle swelling to a 3.3 mL increase in volume versus a 25.3 mL increase seen when heat therapy was used.¹⁸

In addition to ice therapy, compression devices have been shown to be effective tools for edema control. Myerson et al. showed a 50% reduction in edema in both the acutely swollen and chronically swollen ankle when using an intermittent pneumatic device versus a control device daily for one month.¹⁹



Fig. 30.2 Cold compression device for swelling control.

Likewise, Thordarson et al. showed a 60 mL reduction in total foot and ankle volume when using a similar compression device compared to standard splint immobilization prior to surgery for displaced Weber B and C fibula fractures.²⁰ In our experience, we have found cold compressive devices for the ankle and foot to be most effective, as they combine the positive benefits of cryotherapy and compression simultaneously. These devices work even better when used in conjunction with elevation to reduce hydrostatic pressure and diminish edema.

A contraindication to cryotherapy is individuals with hypersensitivity to cold. Cold should be avoided in patients with Raynaud's syndrome or peripheral vascular disease. Cold therapy also must be monitored closely in postoperative patients who have wet dressings because the combination of wet dressings with cold application can decrease the skin temperature to a dangerous level. There have been case reports of severe frostbite occurring due to prolonged cryotherapy.²¹

RANGE OF MOTION/MOBILIZATION

There has long been a rehabilitation dilemma between the need for early ROM and the need to immobilize tissues to decrease swelling, allow the injured tissues to heal, and protect against pathologic motion. There has been ongoing research across all sections of orthopedics that promote the idea that early mobilization is best for most injuries. For example, early mobilization has been shown to have earlier return to work and better ROM when compared to splint immobilization for simple elbow dislocations.²² Another randomized controlled trial reported no increased risk of dislocation with improved pain scores, faster return of functional ROM, and increased patient satisfaction with accelerated rehabilitation following arthroscopic Bankart repair of the shoulder.²³

In 1892, Julius Wolff, a German anatomist, explained that every change in the function of a bone is followed by certain definite changes in internal architecture and external confirmation in accordance with mathematical laws. Stated simply, “form follows function.” Wolff’s law also may apply to these soft tissues, and physiologic stress may allow more functional and stronger healing of soft tissues. Experimental studies of ligaments after injury indicate that exercise and joint motion stimulate healing and influence the strength of ligaments after injury.²⁴⁻²⁸ In addition, it has been shown in animal models that complete removal of load is detrimental to rotator cuff tendon healing, especially when combined with immobilization.²⁹

Some of the early research on restoration of early ROM was performed in the hand. These historical papers revealed insight on how early ROM decreases complications and enhances the healing process. Early mobilization may result in an earlier return to work and daily activity, less muscle atrophy, and better mobility compared with immobilization by casting.^{27,30,31} The value and benefit of early motion was investigated after flexor tendon repairs of the hand. The obvious need for full motion in the hand prompted investigation into safe rehabilitation practices, which would eliminate postoperative adhesions and stiffness but still allow reliable healing of the tendon. Gelberman et al. noted an improved healing response, improved strength, and a more normal pattern of vascularity to the healing tendon with protected early mobilization.^{32,33} Several other studies also noted that early ROM decreased adhesions around the repaired tendon and had a positive influence to the healing tissue.³⁴ Early motion after flexor tendon repair has become standard today.

Robert Salter and associates investigated the effect of joint motion on cartilage nutrition. They found that early, continuous passive motion in synovial joints promotes cartilage nutrition and health.³⁵ Salter demonstrated that small cartilage defects could heal with continuous motion, further supporting the benefit of motion on articular cartilage nutrition and healing. Joint motion produces mechanical signals that are perceived by chondrocytes that in turn influence and stabilize the internal cartilage structure and prevent the cartilage thinning seen with prolonged immobilization.³⁶

Eiff et al. used a prospective randomized study to determine which treatment for first-time ankle sprains, early mobilization or immobilization, is more effective. They reported that, in first-time lateral ankle sprains, early mobilization allows earlier return to work and may be more comfortable for patients than prolonged immobilization.³⁰ Both active and passive ROM are utilized to regain motion in cardinal and diagonal planes. Passive ROM allows the muscles to relax while active ROM requires independent muscle action and incorporates muscle re-education. It is important to first work ROM in the direction opposite of the mechanism of injury (i.e., we allow dorsiflexion and eversion and avoid plantarflexion and inversion initially after a grade II or III lateral ankle sprain). Once the injury has healed, ROM should include all directions.

Several recent publications have shown promising clinical results with accelerated rehabilitation protocols. One randomized controlled trial showed that patients undergoing an accelerated rehabilitation protocol after Achilles tendon repair had

significantly less tendo-Achilles lengthening and quicker return to full running than patients with traditional immobilization.³⁷ Another study found that early ROM after anterior talofibular ligament reconstruction for chronic ankle instability led to a 5-week earlier return to full athletic activity when compared to a 4-week period of immobilization with no cases of re-injury or differences in postoperative outcomes.³⁸ These and other studies have led us to adopt early ROM as a staple in the post-injury and post-surgical management of almost all foot and ankle pathologies.

PROTECTED WEIGHT BEARING

Early protected weight bearing has been shown in several well-conducted studies to provide excellent clinical outcomes without an increased rate of complications. One randomized controlled trial showed improved patient reported outcomes and ankle ROM with weight bearing begun at 2 weeks postoperatively versus the control group (6 weeks of nonweight bearing, NWB) for operatively treated unstable ankle fractures.³⁹ Another study found no increased rates of rerupture or other complication with same-day weight bearing versus 4 weeks of NWB for conservatively treated Achilles tendon ruptures.⁴⁰

Recent investigations have published promising results with early weight bearing after procedures that have long been thought to require long periods of NWB. Mann et al. reported a >95% fusion rate in 21 first metatarsophalangeal joint fusions using a titanium plate and full weight bearing at 2 weeks.⁴¹ Another study showed no increased rate of nonunion with early full weight bearing in a multicenter study of 367 patients undergoing a modified Lapidus arthrodesis.⁴²

Weightlessness, as seen in space travel, has been shown to have a detrimental effect on muscle. Costill et al. reported that a 17-day space flight resulted in an 11% decrease in peak muscle power, a decrease in muscle fiber diameter, and a 21% decrease in force when the muscle was contracted at peak power velocity.⁴³ The single fiber muscle diameter decreased 20%. This research helps us understand the atrophy and deconditioning that occurs in our patients when they are made to be NWB for periods much longer than 17 days after certain procedures or injuries.

We favor a postoperative protocol that allows for early weight bearing whenever possible. We recognize there are times when this is not possible, such as comminuted intra-articular fractures. However, in the sports population, early weight bearing can have such a positive impact that we try to tailor our surgical and nonoperative approach to allow early protected weight bearing. Research suggests that early loading of damaged soft tissue can enhance collagen fiber realignment and healing.^{26,27,28,44,45} Using a removable walking boot allows the patient to progress to weight bearing immediately after injury. Being in a walking boot instead of an ankle cast allows the patient to take the boot off to begin rehabilitation activities.

In the instances where immediate weight bearing is not feasible, we are using an antigravity treadmill (AlterG®) to aid in rehabilitation (Fig. 30.3). The antigravity treadmill allows



Fig. 30.3 Athlete on the Alter-G™, anti-gravity treadmill.

walking and running with significantly reduced weight. This allows the athlete to normalize gait and promote muscle and tendon function while providing the necessary protected weight bearing in some situations.

GAIT EVALUATION

The evaluation of a patient's gait immediately after injury and before return to activity can provide a clinician with valuable information on how abnormalities in ambulation contribute to the rehabilitation and prevention of injuries. Often abnormal gait mechanics can predispose the other joints of the lower extremity and back to overload and pain. Restoring normal gait after acute injuries can help to prevent these abnormal mechanics and significantly reduce the amount of time required for return to normal function. It is important that a clinician evaluates the entire lower extremity and its function during gait.

Normal gait is composed of two phases, a stance phase (60%) and a swing phase (40%). The stance phase is composed of five categories, including initial contact (heel strike), loading response (foot flat), midstance (single leg support), terminal stance (heel off), and pre-swing (toe-off). The swing phase consists of initial swing (acceleration), mid-swing, and terminal swing (deceleration).⁴⁶

In acute injuries, a clinician will notice gait abnormalities because of pain, decreased ROM, strength deficits, and lack of proprioception. An antalgic gait is most common, with a decreased stance phase on the affected limb. Crutches can be beneficial for this gait pattern to help the patient remove some percentage of weight/stress on the affected limb and thus allow for a more normal stance phase. A patient may discontinue assistive devices when he or she can walk normally. It is extremely important that as clinicians we correct gait immediately to

prevent abnormal gait habits from becoming permanent. It is likely that some failure to return to full strength after a lower-extremity injury is related to adaptive gait changes that become permanent in unloading the injured extremity. One study on patients with chronic ankle instability found a 16% slower walking velocity, 7% lower step length, and a 43% wider base of support when compared to healthy controls.⁴⁷

In chronic injuries or before return to activity, a clinician should take a closer look at lower-extremity biomechanics and gait abnormalities to facilitate return to function while preventing future problems. Observation of gait should include lateral, anterior, and posterior views. It is important to observe and evaluate the foot, ankle, knee, and hip/pelvis position and biomechanics during the gait cycle. Treatment of gait deviations includes flexibility, strengthening, and proprioception. An orthotic can be an excellent adjunct to rehabilitation if the gait deviation is a result of abnormal biomechanics and structural problems within the foot.

STRENGTHENING

Muscle strengthening should be initiated once the patient has recovered 95% to 100% of the ROM of that joint. Initiating strengthening too early can cause an increase in joint stiffness, therefore decreasing the function of the joint. Isotonic strengthening, whereby the force is kept constant, is most commonly performed. There are two types of isotonic exercises: concentric, which causes muscle shortening; and eccentric, which allows the muscle to lengthen. Both phases are extremely important and should be included in a comprehensive rehabilitation program.

There are several methods of strengthening, including weights, TheraBand, and water resistance. TheraBand is a useful tool to provide dynamic resistance in all directions of the foot and ankle (Fig. 30.4). It has different levels of resistance to allow the athlete to progress. Once the athlete can complete 3 sets of 15 repetitions through a full range of movement, the next level of resistance should be started. This same concept can be used with ankle weights. Care should be taken to include



Fig. 30.4 Thera-band exercises to strengthen foot and ankle.

strengthening of the foot intrinsic muscles in addition to the primary extrinsic muscles responsible for inversion/eversion, supination/pronation, and dorsiflexion/plantarflexion.

PROPRIOCEPTION

Many rehabilitation programs often fail to pay attention to proprioception deficits. Proprioception is the ability of the body to vary the forces of muscles in response to outside forces. Muscles, tendons, and joint receptors provide this information, which affects posture, muscle tone, kinesthetic awareness, and coordination.^{48,49} When an individual is injured, the proprioceptive input to that joint is altered and diminished. Diminished proprioception can lead to a recurrence of injury because of the joint's decreased ability to respond to outside forces.

Proprioception can be improved with a number of treatment techniques. Early weight bearing can help to decrease the amount of proprioception loss. A patient can practice standing with equal weight on both feet, progressing to single leg stance, first on solid ground and then working toward standing on soft surfaces. A biomechanical ankle proprioception system (BAPS) board (Fig. 30.5) or kinesthetic awareness trainer (KAT) can be used as a patient advances through rehabilitation.

Proprioceptive training leads to quantifiable improvements in functional ankle stability. One study showed improvements in dynamic stability in all planes after 12 weeks of training on a stability board trainer in 28 young speed skaters.⁵⁰ Another study showed that after 6 weeks of balance training, individuals with chronic ankle instability demonstrated enhanced dynamic balance, inversion joint position sense, and changes in motor neuron pool excitability compared to healthy controls who did not train.⁵¹ These improvements in proprioception and stability have been proven to lead to a decreased risk of re-injury. In fact, proprioceptive training can reduce the incidence of ankle sprains in athletes with recurrent ankle sprains to the level of an athlete that has no prior history of sprain.⁵² Proprioceptive training has also been found to be cost effective in the prevention of recurrent sprains as compared to standard therapy alone.⁵³



Fig. 30.5 Athlete on the biomechanical ankle proprioception system (BAPS) board.

CARDIOVASCULAR ACTIVITIES

During the rehabilitation program, it is extremely important to keep the patient active. If the patient becomes sedentary, the cellular metabolism levels decrease and the individual will lack energy. Additionally, the patient may experience both diminished desire and blunted motivation because of a form of depression seen after injury in athletes.⁵⁴ Lastly, low levels of physical activity have been linked to dysregulated appetite and subsequent weight gain.⁵⁵ These factors consequently can present a challenge for recovery and rehabilitation. Early in the rehabilitation, we feel that it is vital to start a sensible regimen of low-resistance exercise bike or pool therapy training 3 to 4 days a week for 10 to 15 minutes with a progression by 5 to 10 minutes of training per session per week. If the bike is used, then a walking boot or protective brace is used. Pool therapy is not initiated until the sutures are removed and the wound is fully healed. This early exercise provides physiologic benefits in the form of maintained cellular metabolism as well as psychological benefits for the athlete as physical activity has been proven to reduce depressive symptoms.⁵⁶

Our experience with and observation of clinical healing and postoperative wound healing have proven that it is important to progress the patient's activity gradually. Increasing the time increments of 10 minutes a week on a bike will allow the patient to be working approximately 30 minutes per session in a 3-week span. Typically, low-impact, weight-bearing exercise will be introduced when the athlete can walk normally in a protective device and regular shoe. The rehabilitation program will begin replacing 1 day of bike with an elliptical machine each successive week until the athlete has been converted to 4 to 6 days per week of elliptical. The athlete will continue to increase low-impact, weight-bearing exercise as tolerated. We have found that when an athlete can work out on the elliptical machine 4 to 6 days a week for 30-plus minutes, it is safe to initiate running. Running should gradually replace the elliptical training each week. It is important to give the athlete a set of running guidelines that allows for a gradual progression of activity.

FUNCTIONAL PROGRESSION

A functional progression is a series of sport-specific skills that increase in the level of difficulty that an athlete must complete before he or she can safely return to competition. Yamamoto and Fragi described a functional progression in the rehabilitation of injured West Point cadets.^{57,58} The emphasis in this program was placed on restoring agility through dynamic exercise after knee injury. Kegerreis et al. added specific movement patterns and skills to the program and introduced the importance of addressing the psychological needs of the injured athlete.⁵⁹ They also addressed the scientific principles that play an important role in the functional progression and the need to break down sport-specific functions to be addressed in the order of difficulty.

The functional progression is vital to a complete sport-specific rehabilitation program. It serves to translate the gains from

clinical rehabilitation onto the playing field. Each sport has certain demands and skills that stress the foot and ankle differently. It is extremely important that the athlete advance one step at a time without pain or apprehension. Once the athlete has completed the list of activities in order without pain or apprehension, he or she may return to full sport activity.

There are several physical and psychological benefits that the functional progression will address. The functional progression promotes healing through the application of Davis' law and Wolfe's law, which were discussed earlier. It is important that the healing tissue be stressed in the way required of it before injury so that the tissue will be ready to fully accept preinjury activity requirements. In this way, the injured tissue and bone will be stressed in a controlled, functional manner leading to further tissue and bone healing. In addition, the functional progression breaks up the monotony of traditional rehabilitation and allows the athlete to begin performing activities related to function. Psychologically it allows the athlete to increase self-confidence and mentally prepares him or her to return to sport. As the athlete completes each step, confidence will increase and apprehension will decrease, allowing the athlete to enter the competitive environment at the level of function needed for playing standards.

PHASES OF REHABILITATION

The cornerstone to appropriate rehabilitation is an accurate diagnosis, so that an appropriate rehabilitation program can be established efficiently and safely. For any injury or condition, the rehabilitation can be divided into three general phases. Each phase has specific goals, and, although there is a time frame assigned to each phase, advancement from one phase to another should be based on the patient's achieving the prescribed goals rather than on time. A clinician must be willing to adapt and modify the exercise program for each patient. As the rehabilitation landscape is ever-changing, it is important to stay up to date with current trends.

One interesting field that we have begun utilizing in clinical practice is global positioning systems (GPS) tracking devices (Fig. 30.6). These devices are worn by our football players on their shoulder pads and give us instant, real-time data on velocity, acceleration, and change of direction. We use this information relative to baseline data we have collected on the player to compare where he is in his rehabilitation. This allows us a real-time feedback tool that can provide us data as to how the affected limb is reacting to certain situations. This is invaluable data in making decisions to allow athletes to return to the field safely despite previously accepted return to play norms. This objective data provides a real comparison to pre-injury levels, allowing us to know where deficits remain so we can tailor the final phases of rehabilitation to the specific needs of the athlete. This gives both the surgeon and the athlete the confidence that they are returning to play at a level at or near previous levels prior to the injury. While there is still much research to gather in regards to its application in sport, this emerging technology is another tool that we have to better help the rehabilitation of our athletes.

Phase I

Phase I emphasizes pain modulation and inflammatory control of the soft tissues. Controlling pain and inflammation will allow patients to be better able to perform their rehabilitation exercises. Restoration of normal ROM and joint accessory motions, including glide, roll, and spin, are stressed in this phase. Early return of pain-free ROM will enhance the rehabilitative process and allow the patient to begin isolated and functional rehabilitation exercises in phase II with greater effectiveness. Once a patient has minimal pain and has normal to near-normal ROM, he or she may be advanced to phase II.

Phase II

Once inflammation is decreased, pain has subsided, and ROM is near normal, phase II may begin. Foot and ankle flexibility with functional strengthening are initiated and are the focus of this phase. In addition, cardiovascular conditioning and proprioceptive training also are started at this time. The goals of this particular phase are to improve flexibility, restore strength, and begin light, sport-specific functional training. A patient may be progressed to phase III when he or she is ready for a gradual return to activity and participation in sports.

Phase III

Emphasis in phase III is on functional return to activities of daily living (ADLs) and previous activity/sport participation. Advanced activity-specific exercise should be implemented with special attention to mechanics of the activity. Proper mechanics, as well as maintenance of flexibility and strength, can prevent further chance of re-injury. To ensure safe return to sport, athletes should perform a functional progression. External supports such as braces, straps, taping, and orthotics may be used at this time to allow the patient to participate in his or her activity pain free.



Fig. 30.6 Catapult™ GPS system.

REHABILITATION OF ACHILLES TENDON REPAIR (SEE CHAPTER 9)

Over the past 10 years, functional rehabilitation of Achilles tendon ruptures has become more prevalent. In the past, treatment of Achilles tendon ruptures has focused on immobilization, initially with long-leg casting and ultimately transitioning to short-leg casting. In recent years, however, early weight bearing with accelerated rehabilitation and earlier motion has become the standard of care.^{60,61} The functional, accelerated rehabilitation allows the athlete to return to sports more quickly and readily, without a reported increase in re-rupture rates or complications.⁶² Despite the best advances in surgery and rehabilitation of these injuries, athletic performance after a complete Achilles rupture is often affected.^{63,64}

Mendelbaum et al. initiated an early motion protocol in 29 patients after surgical repair of the Achilles tendon. They reported 93% of the patients returned to full activity by 6 months and functional deficits were less than 3%. This protocol focused on early motion at 3 days postsurgery with weight bearing initiated 2 weeks post repair.⁶⁵ Others have advocated a similar early, protected motion protocol using a walking boot with a dorsiflexion block at -20 degrees. This alternative can result in a quick return to sports activity in a reliable, motivated patient.⁶⁶ Early weight bearing after surgical repair of an acute Achilles tendon rupture improved health-related quality of life in the early postoperative period and had no detrimental effects on recovery. Similar results have been shown with the newer percutaneous repair techniques.⁶⁷

Over 80 Achilles tendon ruptures have been performed over the past 5 years at Andrews Sports Medicine. Our protocol has evolved over time and we now allow full weight bearing at 2 weeks in a walking boot with ROM exercises initiated after the first postoperative visit. We subsequently begin strengthening exercises at 1 month with a stationary bike program and toe raises initiated by 6 weeks. After 8 weeks, the patients are out of protective devices and back in to regular footwear. Once the athlete has reached this stage, the focus turns to slowly regaining full motion and strengthening of the gastroc-soleus complex. Using this protocol, we have only had a single rerupture of the tendon, which is consistent with, or better than, accepted published protocols. The following is an example of our rehabilitation protocol.

Initially, the patient is placed in a postoperative splint in slight equinus position to relax the soft tissues and remove any tension from the incision. After 10–12 days, the patient returns to clinic and sutures are removed. Once the incision is healed, we transition the patient into a walking boot with two ¼ inch heel lifts stacked in the boot. We initiate a weight bearing progression protocol that is outlined in Table 30.1. Compression and elevation are stressed to the patient during this period as edema control is paramount to early progression. Formal physical therapy is begun. We initiate an exercise bike program wearing the walking boot with active and active assist ROM exercises. We avoid passive ROM exercises past neutral in order to avoid stretching out the repair.

We also utilize a home therapy program. The home exercise program includes toe curls, active plantarflexion, resistive-band

TABLE 30.1 Progressive Weight-Bearing Protocol

Weight-Bearing Progression Protocol	
Day 1–2	25 lbs of pressure in boot with two crutches
Day 3–5	50 lbs of pressure in boot with two crutches
Day 6–8	75–100 lbs of pressure in boot with transition to one crutch
Day 9–11	100–125 lbs of pressure in boot with one crutch
Day 12–14	150 lbs to full weight bearing with transition to no crutch
	Wear boot in times of excessive walking, ankle brace should be worn when out of boot if instructed by physician

plantarflexion, and seated calf raises. We use the concept of early protected motion and resistance training, which encourages stronger tendon healing and protects against disuse atrophy. Exercises are performed at a higher frequency with a low load to continuously stimulate the tendon to heal. It is extremely important to avoid ankle dorsiflexion activity or a heel cord stretch to protect the tendon from overstretching.

The second phase of rehabilitation begins approximately 6 weeks after repair. At this time, an increase in weight-bearing exercise is allowed, and proprioception retraining with an emphasis on normal gait is initiated. Athletes at this time are instructed in a program to wean out of the boot into an athletic shoe with one 9/16th inch felt heel lift. Our goal is to wean the patient out of the boot over 2 weeks with normal pain-free gait.

During this phase, it is important to stress to the athlete not to overtrain or attempt to push the therapy faster than the tendon can handle. While many of the athletes are motivated at a level higher than the average patient, it is stressed that protocol compliance during this phase of the rehabilitation is important. At this point, the athlete is weaned from weight-bearing protection into regular footwear, which can provide a false sense of comfort. During this phase, calf raises are initiated, balance and strengthening exercises are progressed, and elliptical/anti-gravity treadmill programs are started. Once the goal of a normalized gait is achieved, proprioceptive strengthening exercises become important. After 8 weeks, dorsiflexion exercises past neutral are started with slow increase toward full ROM over the next 4 weeks. During this period, sitting and standing strengthening exercises are progressed, while jumping and explosive exercises are prohibited. It is important to avoid forced dorsiflexion, as the final few degrees of motion will come over the following months as the patient progresses back to normal activity. The final phase of rehabilitation starts approximately at the 3-month mark. Patients will continue to work on balance, ankle strength, and unilateral calf raises. At this time, full lower-extremity strengthening will be initiated. Exercise will include step-downs, leg press, knee extensions (Fig. 30.7), and hamstring curls that can be advanced per patient tolerance. Weighted calf raises typically are initiated around 4 months. Explosive exercises are avoided until at least 4 months after surgery. These types of sudden motion, i.e., jumping and landing, put the tendon most at risk for re-rupture.



Fig. 30.7 Athlete using knee extension exercise to strengthen lower limb.

TABLE 30.2 Foot and Ankle Return to Sport Test

Knee to Wall	Determine loaded and functional dorsiflexion ROM	Pain free and 90% of the unaffected side
Y-Balance	Assess core control, balance, proprioception, ROM, strength	Pain free and <4 cm in anterior reach
Jump for Height	Assess single leg jump height to determine power	Pain free and 90% of the unaffected limb
Running Agility	Challenge running/change of direction in multiple areas	Bilateral times within 90% of each other

Data from Clanton TO, Matheny LM, Jarvis HC, et al. Return to play in athletes following ankle injuries. *Sports Health*. 2012;4(6):471-474.

Once an athlete is capable of double leg heel rise, walking without altered gait, and is using the elliptical for 30 minutes on a daily basis, he or she may begin light jogging outside of the anti-gravity treadmill (usually 3–4 months after the repair). Sport-specific activities are begun 4 months from surgery. Agility drills should be advanced gradually per patient tolerance using the functional progression as discussed above. Once the athlete is nearing return, we use objective, validated, functional testing to confirm the athlete is truly ready to return to play.⁶⁸ (Table 30.2) Return to sports normally occurs at 5 to 8 months after surgery.

REHABILITATION AFTER LATERAL ANKLE RECONSTRUCTION (SEE ALSO CHAPTER 15)

The goals of ankle sprain rehabilitation, or rehabilitation after lateral ligament repair, are to put the ankle in a healing position that avoids elongation and tension on the lateral ligaments. In each case, stress shielding of the healing ligaments followed by integrating functional rehabilitation with graduated stress application and strengthening will allow the ligament to appropriately heal. Ultimately, our goal of athletic rehabilitation is to return the athlete to sport as quickly and efficiently as possible without compromising the athlete, or the injured limb, while avoiding the potential for future problems.

Immobilization is often used in the early rehabilitation phase. Use of splints, casts, or removable walking boots are common in the acute phase. Immobilization, particularly with a CAM walking boot, puts the ankle in a stable, neutral position and helps to avoid compromising positions. These immobilization devices will allow the athlete to weight bear with the mortise in its most stable position.

After ankle reconstruction, historically, immobilization for prolonged periods of time was the standard treatment. More recently, however, early, protected motion has become the norm. With early mobilization of the ankle, appropriate stress can be applied to the ankle that will allow the ligaments and soft tissues to heal in an oriented manner that will promote both motion and stability. Functional exercises have been shown to be important to stress the healing tissues without compromising their integrity.³⁸ The following goals are important for any rehabilitation program: decreased swelling, pain, and initial inflammatory response and protection of the joint so that a secondary inflammatory response does not develop from overly aggressive rehabilitation. Similarly, ROM, muscular strength, power, and endurance must be returned to preinjury levels so that full, asymptomatic functional activities may be performed.

A similar approach can be used following a lateral ankle reconstruction. For the reliable athlete with close medical monitoring and sturdy tissue at the time of reconstruction, there may be a place for intermittent immobilization with early weight bearing and specific ROM exercise. Overall the objective is to obtain as normal an ankle as possible.⁶⁹

In our practice, the patient is initially placed in a postoperative splint for 10–12 days to allow the soft tissues to quiet down, with the athlete relying on elevation and decreased mobility to help control the swelling. Once the incision is healed and sutures are removed, the athlete is transitioned into a CAM walker boot and the first phase of rehabilitation is begun. Initially the focus is on swelling control, ROM in dorsiflexion and eversion, and restoring the normal gait cycle. Once these exercises are commenced, under supervision of a physical therapist or athletic trainer, light strengthening is begun. Care is taken to avoid compromising positions of the ankle, specifically plantarflexion and inversion. Swelling control is emphasized, both through the use of elevation and cold compression devices.

A home exercise program is also utilized during this period. Typically, our athletes utilize TheraBands for early ROM exercises and strengthening. The athlete is instructed on this program to ensure that ROM limits are set and the patient is understanding of the need to avoid inversion and plantarflexion. Cardiovascular exercises are begun, specifically using the stationary bike, while wearing the walking boot.

Partial weight bearing is typically started 2 weeks from surgery. A progressive weight bearing program is implemented to avoid excessive stresses placed across the foot and ankle (Table 30.1). This progression allows the athlete to reintroduce stress across the foot and ankle. Once full weight bearing is achieved, we will begin weaning out of the walking boot, which will allow the athlete to return to normal shoes. We have the athlete wear a brace at all times during this phase to protect the ankle from accidental injury. During the boot wean phase, ROM exercises

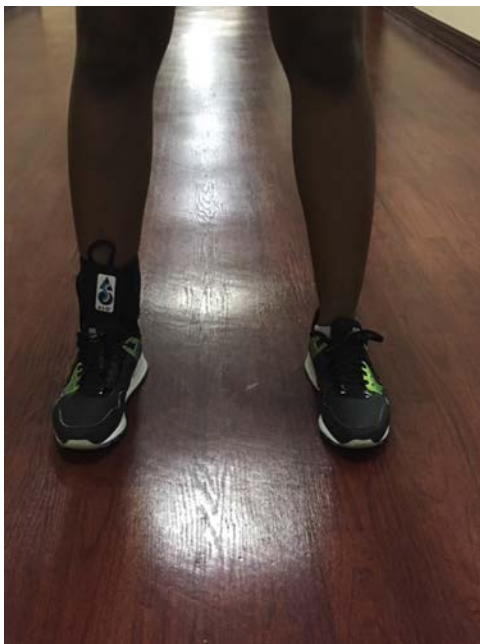


Fig. 30.8 Athlete in Ankle Stabilizing Orthosis (ASO) brace.

and strengthening exercises in all planes are commenced. Our main goal during this phase is to restore full, unrestricted ROM. Once the athlete is out of the boot, more aggressive therapy is begun.

Desensitization massage is an important part of the early rehabilitation program. Because of the highly innervated foot and ankle, the patient often will experience some surface hypersensitivity after surgery. It is important to stimulate this tissue with light massage and tactile stimulation to reeducate and desensitize the tissue to normal pressure and touch. This can be accomplished with a light massage 3 to 5 minutes several times a day.

The second phase of rehabilitation begins 4–6 weeks post-operatively. At this time patients are instructed to wean out of the boot into a stirrup or ankle stabilizing orthosis brace (Fig. 30.8; Table 30.3). Our goal is to gradually wean the athlete out of the boot within a 2-week time period and obtain a normal, pain-free gait.

Exercises in the second phase include ROM/strengthening in all four directions, aggressive heel-cord stretching, calf raises, and proprioception exercise. Dorsiflexion and inversion strengthening still are performed with Thera-tubing. Aggressive peroneal strength is accomplished by having the athlete lie in a lateral position with ankle weights hung over the end of the foot and the toes pointed to isolate the peroneal tendons. The athlete then everts the foot and ankle to strengthen the tendons. We have found this to be a very effective means of maximizing peroneal strength. Bilateral calf raises are initiated with progression to single calf raise. We like to have the patient work on eccentric phase of calf raise by going up on both and lowering slowly on the injured side. Once the patient has no difficulty with the eccentric phase of the exercise, he or she may add the concentric phase of the exercise. Proprioception exercise should begin with one-foot balance, with progression of balance with

TABLE 30.3 Progressive Boot Wean Protocol

Boot Wean Protocol

Day 1–2	1 hour in morning/1 hour in afternoon
Day 3–5	2 hours in morning/2 hours in afternoon
Day 6–8	3 hours in morning/3 hours in afternoon
Day 9–11	4 hours in morning/4 hours in afternoon
Day 12–14	Progress to full time weight bearing out of boot

opposite hip/leg exercise. Cardiovascular exercise should be advanced from the bike to an elliptical machine (4–6 weeks after surgery) and eventually to light jogging (6–10 weeks after surgery).

The final phase of rehabilitation, which occurs between weeks 10 and 14, focuses on sport specific activities. The progression to explosive activities is dependent upon continued improvement with minimal swelling and pain-free exercise. This phase is focused on ankle and leg strengthening, restoring the flexibility of the lower limb, and proprioceptive activity. There are no restrictions placed on the athlete as long as swelling is controlled and the ankle remains pain-free. A stirrup-strap type brace is utilized and on-field activities are started. Before return to sport, the patient should successfully complete a sport-specific functional progression program to ensure safe return to competition. Return to sports participation is typically seen at 12 to 14 weeks.

REHABILITATION OF HIGH ANKLE SPRAINS (SYNDESMOSIS INJURIES) (SEE ALSO CHAPTER 15)

High ankle sprains, or syndesmosis injuries, have become more prevalent in recent years. While it is difficult to tell if this is because of better recognition of the injury by treatment providers or other variables, such as shoe wear or playing surfaces, the treatment of these injuries has been evolving over the past decade. For isolated ligamentous injuries, conservative treatment was almost always the standard treatment. Recently, however, operative stabilization of the ankle with endobutton constructs has allowed us to rehabilitate athletes at a quicker pace and has allowed quicker return to play.

As with any injury, our goal is to return the athlete to the field in a safe, efficient manner without compromising the future health of the athlete. Because of the high incidence of continued problems with chronic syndesmosis injuries, a recent trend has been for operative fixation of the more severe grade II injuries and all grade III type injuries. This allows a faster rehabilitation, enabling the athlete to return to play in less time.

In our practice, typical repair includes ankle arthroscopy to debride the joint and assess the cartilage, and stabilization with a divergent endobutton construct over a lateral plate. The first 4 days following surgery are spent in a splint for soft tissue rest. On day 4, the splint is removed and the athlete is transitioned into a CAM walker boot. Edema control modalities are initiated to limit the amount of swelling. The quicker we can decrease the

swelling, the quicker we can regain full motion, thus allowing for a shorter return to play. Weight bearing as tolerated is initiated on day 4, though it often takes the athlete several days to become fully comfortable weight bearing in the boot. Once the athlete has progressed to full weight bearing, we begin weaning from the boot. Typically, this is done over 7–10 days.

Exercise bike and anti-gravity treadmill are begun 1 week from surgery. As gait normalizes, the progression to faster pace and full weight bearing is initiated. TheraBand exercises as well as proprioceptive exercises are also performed during this phase. The goal is to limit strength loss while maintaining motion. Avoiding excessive dorsiflexion can help avoid discomfort as the rehabilitation phase is started. Once the patient has returned to normal gait with full weight bearing, we transition them to explosive cutting and jumping exercises. This typically occurs in the day 14–21 range. At this point the therapy is directed by the athlete's symptoms as long as the swelling remains controlled and gait remains normal. Once the athlete can do single leg heel rise without difficulty and a single leg hop test is completed, return to sport can be considered. Typically, this occurs between weeks 3 and 6. If the athlete can sustain a faster pace of rehabilitation, he or she is not prevented from doing so.

REHABILITATION OF ANKLE FRACTURES (SEE ALSO CHAPTER 14)

Rehabilitation of athletes who sustain ankle fractures can be both challenging and rewarding for the treating team. Treatment of ankle fractures focuses on the restoration of a congruent, stable joint while providing rigid, anatomic fixation of the fractured bones. This allows us to be confident as we progress the athletes quickly through phases of immobilization to early ROM and protected weight bearing. In minimizing immobilization, we can maximize swelling control, improve early-phase strengthening, and normalize gait quicker. This allows the athlete to return to play quicker than traditional methods with long periods of immobilization.

In the immediate postoperative period, the patient is placed in a splint for swelling control and to allow the incision to heal without complications. Patients are instructed to elevate the extremity as much as possible to help decrease swelling. Nonweight bearing with axillary crutches or a rolling knee scooter is initiated initially after surgery to reduce the risk of immediate postoperative swelling. The patient should also wiggle the toes and perform leg lifts every 3 to 4 hours while awake.

After 10–14 days, the athlete is transitioned into a CAM walker boot to allow for cold compression and physical therapy. If stable bone alignment is demonstrated on radiographs, range-of-motion exercises are started. ROM should be initiated in a manner that does not put tension on an injured or repaired ligament. For an isolated fibula or stable bimalleolar fracture, ROM can include all directions. If the patient has a medial ligament injury or syndesmosis disruption, dorsiflexion with eversion should be avoided until the ligament is healed. Range of motion and light Thera-tubing exercises are guided by pain and should be performed several times a day in high repetitions (15–20); towel stretch for the Achilles and manual plantarflexion stretch



Fig. 30.9 Single-leg heel rise.

can be started (20 seconds, 5 repetitions) if there is no contraindicating ligament injury. Early active controlled motion in this manner has been reported to improve the ultimate outcomes in ankle fractures.⁷⁰ The home exercise program will consist of toe curls, ROM in appropriate directions, resistive band in appropriate directions, desensitization massage, and a light bike program wearing the boot.

Partial weight bearing is started at 2 weeks, with progression to full weight bearing in the walking boot in 2 weeks (if the fracture is stable and does not involve a weight-bearing surface). Patients are instructed to use axillary crutches and increase weight bearing as tolerated. After the first week of partial weight bearing, the patient may begin using one crutch under the opposite arm and eventually progress to full weight bearing over the next week. Once a patient can walk normally with the walking boot (typically within 3 to 4 weeks), we begin weaning the patient out of the boot and into a stirrup brace and regular shoe over the next 2 weeks. Patients with highly comminuted fractures and those with a weight-bearing joint injury or significant cartilage injury do not follow this same protocol.

The second phase of rehabilitation begins approximately 1 month after surgery. At this time, an increase in weight-bearing exercise, proprioception, and gait training with an athletic shoe is initiated. Exercises consist of progression of Thera-band activities to include directions originally avoided because of ligament complications. Standing calf stretching, balancing exercises, double to single leg calf raises, and elliptical/anti-gravity treadmill progression is included during this phase. TheraBand exercise should continue to be high repetitions (15–20) in all directions. Single leg balance is first initiated in a regular shoe and then progressed to bare foot on a hard surface (Fig. 30.9). Our goal is approximately 60 seconds. Balance can be advanced by use of a soft surface and balance board. The patient should work aggressively with calf stretching using a stair or an incline board for 3 minutes three times a day. Bilateral standing calf raises should be initiated with progression to single-leg calf raises. Once completely out of the boot, elliptical or anti-gravity treadmill progression should be substituted for the bike

with use of the brace and athletic shoes. Patients are given a home exercise program to be performed two to three times a day. Athletes who have athletic training resources should work under the guidance of the athletic training staff.

The final phase of rehabilitation (2 months) should focus on advancing the strength of the entire lower extremity and sport-specific agility drills. The goal of this phase is the return to sport after finishing a sport-specific functional progression program. Exercises in the final phase will continue to focus on ankle strengthening, flexibility, and proprioception activity. We achieve these strengthening goals using leg press, knee extension, and hamstring curls as tolerated. Sport-specific skills should be implemented at this time, increasing the intensity of these activities as tolerated. Return to sports can be highly variable depending on the severity of the fracture pattern. It can be as early as 4 weeks after rigid fixation of an isolated fibula fracture to 8 to 12 weeks after a bimalleolar and equivalent repair. Fractures that require fixation of the syndesmosis or deltoid ligament repair can take 3–6 months before return.

CONCLUSION

Full, complete rehabilitation is the goal for the athlete. In nearly all cases, the athlete will desire 100% strength, full motion, and no limitations in function. This is the goal and challenge for the therapist, trainer, and surgeon. Each member of the team must fulfill their role to complement the others in the goal of full return to play without limitation. Our rehabilitation must take into account the understanding of muscle function and its need for motion with controlled resistance to return to functional ability. Foot and ankle rehabilitation is ever changing. It continues to progress as surgical technologies advance and therapy principles evolve. While we continue to push the envelope of rehabilitation, our principles remain the same. We base our principles and approach to rehabilitation on the basic sciences that build the foundation for treatment. It is this foundation that we use to affect progress with early motion, early weight bearing, and functional resistance. We hope that this chapter encourages you to further challenge yourself in your treatment of athletes.

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Principles of Rehabilitation for the Foot and Ankle

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INTRODUCTION

This chapter provides an overview of the rehabilitation principles, appropriate progression of exercise, and return-to-play criteria when dealing with foot and ankle injuries. Rehabilitative protocols and progressions must be based primarily on the physiological responses of the tissues to injury and on an understanding of how various tissues heal. The foot and ankle often are injured during sporting events, recreational activities, and occupational accidents. Injuries to the foot and ankle may be acute or chronic in nature and often cause considerable disability in athletes. Garrick and Requa¹ reported that foot and ankle injuries represented more than 25% of the 1600 athletic injuries in their series.^{2,3} It has been suggested that the sprained ankle is the single most common injury in sports.^{2,4-7}

The foot and ankle serve as the junction of the body to the weight-bearing surface. This elegant collection of tissues, each with a variety of specialized functions, allows efficient, upright stance and locomotion.⁸ Athletic populations have unique and strenuous demands. Even with minor injuries, improper or incomplete rehabilitation can lead to significant impairment. A detailed, focused approach to rehabilitation of the foot and ankle is crucial to the athlete. Fortunately, most competitive athletes have access to daily evaluation and monitoring of progress, as well as skilled assistance to help them comply with rehabilitation protocols. Recent technologic and procedural advances contribute greatly to the treatment of the competitive athlete. Principles of rehabilitation must continue to advance and keep up to date with technologic and procedural advances. A proper

and advanced approach to rehabilitation can provide an environment conducive to a complete, full, and functional recovery.

CRYOTHERAPY/REST, ICE, COMPRESSION, AND ELEVATION

Rest, ice, compression, and elevation (RICE) are almost universally accepted as best practice by athletic trainers and other health care professionals immediately after acute foot and ankle injuries.⁹ Rest is always important to allow the inflammatory process to run its course, but initiating initial treatment principles in combination will allow for a faster recovery process. There are several cold agents to choose from, including the cold pack, ice bags, cold whirlpool, and ice immersion. There are other popular commercial ice units that include Aircast Cryocuff (DJO, Vista, CA) and Game Ready (Concord, CA). These units help provide cold and compression at the same time. The primary objective of ice is to reduce swelling and help manage pain. It has been found that pain is inhibited by cold through a decrease in nerve conduction velocity. As the temperature decreases, there is a corresponding decrease in sensory and motor nerve velocity, eventually causing synaptic transmission to be blocked.¹⁰ In our experience, as well as in the research, we have found the ankle and foot Cryocuffs to be an inexpensive and effective way to provide compression and cold.¹¹

The application of cold is most effective immediately after injury or within the first 72 hours. Hocutt et al.¹² found that patients with grade III ankle sprains that were treated with ice

in the first day returned to functional activities such as running and jumping after 6 days, whereas those treated on the second day went 11 days before they could run or jump. In contrast, those who received heat in the first day had a recovery time of 14.8 days. Ice should not be used longer than 30 minutes, especially in areas of superficial nerves as it may cause a transient nerve palsy.¹³

A contraindication to cryotherapy is individuals with hypersensitivity to cold. Cold should be avoided in patients with Raynaud's syndrome or peripheral vascular disease (see [Chapter 12](#)). Cold therapy also must be monitored closely in postoperative patients who have wet dressings because the combination of wet dressings with cold application can decrease the skin temperature to a dangerous level.

Elevation decreases hydrostatic pressure to decrease fluid loss, and also assists venous and lymphatic return through gravity.¹³ Physiologically, the application of cold agents also results in arteriolar vasoconstriction, a decrease in local metabolism, and an elevation in pain threshold. Patients should be encouraged to elevate as much as possible over the first 24–48 hours following injury.

Compression has been commonly used both immediately post injury and during the rehab process to decrease edema. It typically involves the application of external pressure in a circumferential or focal manner to the tissues or surrounding tissues injured. Elastic wraps and tape have commonly been used over the years to apply immediate compression. Over the years there have been several compression modalities utilized for both injury management and recovery in later stages of rehab.¹³

RANGE OF MOTION/MOBILIZATION

There always has been an interesting rehabilitation dilemma between the need for early range of motion (ROM) and the need to immobilize tissues to decrease swelling, protect injuries, and protect against pathologic motion. This section discusses the advantages of early ROM and mobilization.

Galileo first recognized the relationship between applied load and bone morphology. In 1892, Julius Wolff, a German anatomist, was the first to link these two vital concepts in his landmark thesis, *The Law of Bone Transformation*. Wolff explained that every change in the function of a bone is followed by certain definite changes in internal architecture and external confirmation in accordance with mathematical laws; stated simply, “form follows function.”

Application of early motion on ligament healing demonstrates that the ligament hypertrophies to compensate for decreased tensile strength of the individual fibers. Obviously the amount of tension and stress must not overcome the ultimate load to failure of the tissue and must not lead to fatigue or plastic deformation. Wolff's law also may apply to these soft tissues, and physiologic stress may allow more functional and stronger healing of soft tissues. Experimental studies of ligaments after injury indicate that exercise and joint motion stimulate healing and influence the strength of ligaments after injury.^{14–19}

Some of the early research on restoration of early ROM was performed in the hand and the knee. These historical papers

revealed insight on how early ROM decreases complications and actually enhances the healing process. Early mobilization may result in an earlier return to work and daily activity, less muscle atrophy, and better mobility compared with immobilization by casting.^{17,20,21} The value and benefit of early motion was investigated in the area of rehabilitation after flexor tendon repairs of the hand. The obvious need for full motion in the hand prompted investigation into safe rehabilitation practices, which would eliminate postoperative adhesions and stiffness but allow reliable healing of the tendon. Gelberman et al.^{22,23} noted an improved healing response, improved strength, and a more normal pattern of vascularity to the healing tendon with protective early mobilization. Several other studies also noted that early ROM decreased adhesions around the repaired tendon and had a positive influence to the healing tissue.^{24,25} Early motion after flexor tendon repair has become standard today.

Over the past several decades, there have been significant studies in the area of rehabilitation after knee injury and surgery. The focus of knee rehabilitation has centered on obtaining full symmetrical ROM following a knee injury or surgery. Obtaining full knee extension was one of the most important criteria in allowing the anterior cruciate ligament to heal anatomically and yet still avoid a knee flexion contracture. Close observation of patients who were doing well demonstrated that early ROM was not detrimental to the ligament (and in fact could be advantageous to proper ligament healing/strengthening) while allowing an earlier and safe return to function.²⁶ Early motion and weight bearing led to a significant decrease in muscle atrophy and decreased complications from arthrofibrosis with an earlier return to function.

Robert Salter and associates²⁷ investigated the effect of joint motion on cartilage nutrition. Early continuous passive motion in synovial joints allows and promotes cartilage nutrition and health. Salter et al.²⁷ demonstrated that small cartilage defects actually could heal with continuous motion, further supporting the benefit of motion on articular cartilage nutrition and healing.

These advances in hand and knee rehabilitation gave us reason to approach the foot and ankle with a similar approach. Thus early mobilization of the foot and ankle following injury is our currently favored treatment method when applicable. This method specifically avoids or reduces immobilization. We have followed the principle that unnecessarily protracted immobilization can prolong the recovery period. Early mobilization can expedite the return to work and resumption of athletic activity while potentially decreasing the risk of complications.

Eiff et al.²⁰ used a prospective randomized study to determine which treatment for first-time ankle sprains, early mobilization or immobilization, is more effective. They reported that, in first-time lateral ankle sprains, although both immobilization and early mobilization prevent late residual symptoms and ankle instability, early mobilization allows earlier return to work and may be more comfortable for patients. Active and passive ROM is useful to regain motion in cardinal and diagonal planes. Passive ROM allows the muscles to relax while working the mobility of the joint. Active ROM requires independent muscle action and incorporates muscle re-education. It is important to

work ROM in the direction opposite of the mechanism of injury (i.e., we allow dorsiflexion (DF) and eversion (EV) and avoid plantarflexion (PF) and inversion (IN) initially after a grade II or III lateral ankle sprain). Once the injury has healed, ROM should include all directions.

In addition to active and passive ROM, joint mobilization should be incorporated in the rehabilitation program. Accessory movements, termed *joint play*, are not volitional but accompany voluntary movements or occur passively in response to the ground or other forces. The amount of joint play is a function of ligament and soft-tissue compliance as well as bony configuration.²⁸ Mobilization techniques involve oscillation, distraction, and gliding movements of the joints in the planes of accessory motions. The range of mobilization is always advanced in a graded manner but always stays within the physiologic limits of the joint.²⁸

PROTECTION/IMMOBILIZATION

There is much discussion with regard to immediate, short-term protection of foot and ankle injuries. These devices can be utilized in both conservative and surgical management of injuries. A protective device allows the foot and ankle to be rested in an optimum position for healing, while allowing for early rehabilitation principles. Some of the more common methods consist of elastic wrapping, taping/strapping, semi-rigid pneumatic ankle brace, nonrigid functional ankle brace, and a removable walking boot.

A device we like is the Aircast walking boot with built-in Aircast Cryocuff (Fig. 31.1). The device allows patients to weight bear immediately, work on ROM by removing the boot, and use a continuous cold/compression device. Once the ankle has healed, a more functional brace is used for return to activity (2–4 weeks after injury). We particularly stress the use of the boot at night for the first 3 to 4 weeks to keep the foot and ankle complex in a 90-degree DF position during sleep, when the relaxation of muscular control and the forces on the heel

passively place the complex in a PF and IN position. The rigid boot counteracts this relaxed position.

PROTECTED WEIGHT BEARING

Early weight bearing has been shown to increase the stability of the lateral ankle ligaments after injury while decreasing the amount of muscle atrophy. Protected weight bearing provides a safe and earlier return to activity when appropriate by decreasing joint stiffness, muscular strength deficits, and proprioception dysfunction. We favor a postoperative protocol that allows for early weight bearing whenever possible. We recognize there are times when this is not possible, such as in hindfoot fusions. However, in the sports population, early weight bearing can have such a positive impact that we try to tailor our surgical and nonoperative approach to allow early protected weight bearing.

An intriguing area of research that is revealing to us is the investigation of weightlessness. Costill et al.²⁹ examined the effect of a 17-day space flight (essentially, total weightlessness) on muscle. They reported that there was an 11% decrease in peak muscle power, a decrease in muscle fiber diameter, and a 21% decrease in force when the muscle was contracted at peak power velocity. More specifically, Costill et al.²⁹ examined single muscle fiber changes after weightlessness. The single fiber diameter decreases were 20% after 17 days suspended leg weightlessness (for example crutch-assisted non-weight bearing) and demonstrated similar profound muscular atrophy.

Research suggests that early loading of damaged soft tissue can enhance collagen fiber realignment and healing.^{16,17,19,30,31} Using a removable Aircast walking boot allows the patient to progress to weight bear immediately after injury (Fig. 31.2). Being in a walking boot instead of an ankle cast allows the patient to take the boot off to begin rehabilitation activities. The walking boot provides more support than elastic wrapping, taping, and other semirigid bracing systems, and it also allows the patient the ability to apply cold compression simultaneously.



Fig. 31.1 Aircast Cryocuff and walking boot.



Fig. 31.2 Boot.

GAIT EVALUATION

The evaluation of a patient's gait immediately after injury and before return to activity can provide a clinician with valuable information on how abnormalities in ambulation contribute to the rehabilitation and prevention of injuries. Often abnormal gait mechanics can predispose the other joints of the lower extremity and back to overload and pain. Restoring normal gait after acute injuries can help to prevent these abnormal mechanics and significantly reduce the amount of time required for return to normal function. It is important that a clinician evaluate the entire lower extremity and its function during gait.

Normal gait is composed of two phases, a stance phase (60%) and a swing phase (40%). The stance phase is composed of five categories, including initial contact (heel strike), loading response (foot flat), midstance (single-leg support), terminal stance (heel off), and preswing (toe-off). The swing phase consists of initial swing (acceleration), midswing, and terminal swing (deceleration).³²⁻³⁴

In acute injuries, a clinician will notice gait abnormalities because of pain, decreased ROM, strength deficits, and lack of proprioception. The majority of the time, a patient will present antalgic with a decreased stance phase. If a patient is unable to walk without antalgia, a clinician should educate the patient on normal gait mechanics using assistive devices; for example, crutches. A patient may discontinue assistive devices when he or she can walk normally. It is extremely important that as clinicians we correct gait immediately to prevent abnormal gait habits from becoming permanent. It is likely that some failure to return to full strength return after a lower-extremity injury is related to adaptive gait changes that become permanent in unloading the injured extremity.

In chronic injuries or before return to activity, a clinician should take a closer look at lower-extremity biomechanics and gait abnormalities to facilitate return to function while preventing future problems. Observation of gait should include lateral, anterior, and posterior view. It is important to observe and evaluate the foot, ankle, knee, and hip/pelvis position and biomechanics during the gait cycle. Treatment of gait deviations includes flexibility, strengthening, and proprioception. An orthotic can be an excellent adjunct to rehabilitation if the gait deviation is a result of abnormal biomechanics and structural problems within the foot.

STRENGTHENING

Muscle strengthening should be initiated once the patient has recovered 95% to 100% of the ROM of that joint. Initiating strengthening too early can cause an increase in joint stiffness, therefore decreasing the function of the joint. Working isometrically, isotonically, or isokinetically can achieve strengthening. Isotonic strengthening, which is most commonly performed, uses concentric and eccentric contractions. Concentric contraction causes muscle shortening, whereas in an eccentric contraction the muscle lengthens while maintaining a load. Both phases are extremely important and should be included in a comprehensive rehabilitation program.



Fig. 31.3 Dorsiflexion.



Fig. 31.4 Plantarflexion with tubing.

There are several methods of strengthening, including weights, Thera-Band, and water resistance. Thera-Band is a useful tool to provide resistance in all directions of the foot and ankle. It has different levels of resistance to allow the athlete to progress. Once the athlete can complete 3 sets of 15 repetitions through a full range of movement, the next level of resistance should be started. This same concept can be used with ankle weigh (Fig. 31.3 through 31.6, 31.7, A and B).

PROPRIOCEPTION

Many rehabilitation programs often fail to pay attention to proprioception deficits. Proprioception is the ability of the body to vary the forces of muscles in response to outside forces. Muscles, tendons, and joint receptors provide this information, which affects posture, muscle tone, kinesthetic awareness, and coordination.^{32,33} When an individual is injured, the proprioceptive input to that joint is altered and diminished. Diminished proprioception can lead to a recurrence of injury because of the joint's decreased ability to respond to outside forces.

Proprioception can be improved with a number of treatment techniques. Early weight bearing can help to decrease the



Fig. 31.5 Inversion.



Fig. 31.6 Ankle eversion with tubing.

amount of proprioception loss. A patient can practice standing with equal weight on both feet, progressing to single-leg stance. A biomechanical ankle proprioception system (BAPS) board or kinesthetic awareness trainer (KAT) can be used as a patient advances through rehabilitation (Fig. 31.8).

CARDIOVASCULAR ACTIVITIES

During the rehabilitation program it is extremely important to keep the patient active. If the patient becomes sedentary, the cellular metabolism levels will decrease and the individual will lack energy, and may experience both diminished desire and blunted motivation because of a form of depression seen after injury in athletes. This consequently can present a challenge for recovery and rehabilitation. Early in the rehabilitation, we feel that it is vital to start a sensible regimen of low-resistance exercise bike or pool therapy training 3 to 4 days a week for 10 to 15 minutes with a progression by 5 to 10 minutes of training per session per week. If the bike is used, then a walking boot or protective brace is used. Pool therapy is not initiated until the sutures are removed and the wound is fully healed. By initiating early activity during the rehabilitation program, the cellular metabolism will be maintained. The early exercise also provides psychological benefits for the athlete. Physically it allows an active blood flow to the involved extremity, and psychologically it helps to keep the patient motivated and counteracts the potential for depression.

Our experience with and observation of clinical healing and postoperative wound healing have proven that it is important to progress the patient's activity gradually. Increasing the time increments of 10 minutes a week on a bike will allow the patient to be working approximately 30 minutes per session in a 3-week span (Table 31.1). Typically, low-impact weight-bearing exercise will be introduced when the athlete is able to walk normally in a protective device and regular shoe. The rehabilitation program will begin replacing 1 day of bike with a StairMaster/elliptical machine (Table 31.2). We allow an additional day of StairMaster or elliptical each successive week until the athlete has been converted to StairMaster or elliptical 4 to 6 days per week. The athlete will continue to increase low-impact weight-bearing exercise as tolerated. We have found that when an athlete can work out on the StairMaster or elliptical machine 4 to 5 days a week for 30-plus minutes, it is safe to initiate running. Running should gradually replace StairMaster/elliptical each week. It is important to give the athlete a set of running guidelines that allows for a gradual progression of activity (Table 31.3).

ADDITIONAL TREATMENT CONSIDERATIONS

Personalized blood flow restriction training (PBFR): PBFR training involves decreasing blood flow to working muscles in order to promote hypertrophy and prevent disuse atrophy of muscles. The technique utilizes the application of pneumatic cuff, similar to a blood pressure cuff, on the proximal aspect



Fig. 31.7 A, Resisted eversion using Thera tubing. B, Side-lying eversion with tubing.



Fig. 31.8 Biomechanical ankle proprioception system (BAPS) board for balance and range of motion.

TABLE 31.1 Cardiovascular Exercise				
CARDIOVASCULAR EXERCISE				
	Boot	Brace		Shoe Insert
	Exercise Equipment	Minutes		Times Per Week
Week 1	<input type="checkbox"/> Stationary Bike <input type="checkbox"/> Stairmaster <input type="checkbox"/> Elliptical	10	15 20	3 4 5
Week 2	<input type="checkbox"/> Stationary Bike <input type="checkbox"/> Stairmaster <input type="checkbox"/> Elliptical	15	20 25	3 4 5
Week 3	<input type="checkbox"/> Stationary Bike <input type="checkbox"/> Stairmaster <input type="checkbox"/> Elliptical	20	25 30	3 4 5

of an upper or lower extremity. A selected pressure is used to provide venous occlusion to the distal aspect of the limb.^{35,36} PBFR creates an anaerobic environment. At the lower oxygen tension level the body recruits muscle fibers normally reserved for more strenuous exercise. In return, the mechanical stress on

TABLE 31.2 Wean out of Boot									
Wean off Crutches									
Week 1	Use both crutches and bear as much weight as tolerated								
Week 2	Use one crutch on side opposite of your injury								
Wean out of Boot									
	Daytime (8–4 pm)				Evenings (4 pm On)				
Week 1	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	
	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	
Week 2	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	
(Mon, Wed, Fri)	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	
(Tues, Thurs, Sat, Sun)	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	
	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	
Week 3	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	<input type="checkbox"/>	Boot	Shoe	<input type="checkbox"/>	
	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	<input type="checkbox"/>	Brace	Insert	<input type="checkbox"/>	
Cardiovascular Exercise									
Boot	Brace			Shoe Insert					
	Exercise Equipment		Minutes			Times Per Week			
Week 1	<input type="checkbox"/>	Stationary Bike	10	15	20	3	4	5	
	<input type="checkbox"/>	Stairmaster							
	<input type="checkbox"/>	Elliptical							
Week 2	<input type="checkbox"/>	Stationary Bike	15	20	25	3	4	5	
	<input type="checkbox"/>	Stairmaster							
	<input type="checkbox"/>	Elliptical							
Week 3	<input type="checkbox"/>	Stationary Bike	20	25	30	3	4	5	
	<input type="checkbox"/>	Stairmaster							
	<input type="checkbox"/>	Elliptical							

the muscle fibers leads to up-regulation of the muscle hypertrophy-signaling cascade (Fig. 31.9, A through C).^{36,37} This can be used during all phases of rehab to help enhance strength gains. An important application of PBFR training has been to treat patients in the rehabilitation phase following a period of injury or deconditioning after injury or surgical intervention. Postoperative rehabilitation can require prolonged treatment to achieve preinjury muscular strength. Furthermore, some surgical interventions require delays in high intensity training to allow postoperative healing of repaired or reconstructed

TABLE 31.3A Distance Running Progression (Distance Goal)**Methodist Sports Medicine—The Orthopedic Specialists
Distance Running Progression****Distance Goal**

Your injury has resolved enough to let you begin running again, but it is necessary to return to previous mileage gradually. The following guidelines will help to ensure a safe return “to the road.”

- Make sure that you stretch before and after running.
- Keep the running surface as soft, smooth, and level as possible.
- Emphasize form.
- Ice the involved area 20–30 minutes after running.
- Follow the mileage guidelines listed below. Do not progress to the next step if the previous one caused pain or compensation.

Previously Running 15–20 Miles per Week

		DAY							Total Miles
		1	2	3	4	5	6	7	
Week	1	.5	0	.5	0	.75	0	1	2.75
	2	0	1	0	1	0	1.5	0	3.5
	3	1.5	0	2	0	2	2	0	7.5
	4	2	0	2.5	2	0	3	0	9.5
	5	3	0	3.5	3	0	4	0	13.5
	6	4	3	0	4.5	4	4	0	19.5

Previously Running 20–35 Miles per Week

		DAY							Total Miles
		1	2	3	4	5	6	7	
Week	1	.5	0	.75	0	1	0	1	3.25
	2	0	1	0	1.5	0	2	0	4.5
	3	2	0	2.5	2	0	3	0	9.5
	4	3	3	0	4	3	0	2	15
	5	0	4	4	0	5	4	3	20
	6	0	6	5	0	5	5	4	25

TABLE 31.3B Distance Running Progression (Time Goal)**Methodist Sports Medicine—The Orthopedic Specialists
Distance Running Progression****Time Goal**

Your injury has resolved enough to let you begin running again, but it is necessary to return to previous mileage gradually. The following guidelines will help to ensure a safe return “to the road.”

- Make sure that you stretch before and after running.
- Keep the running surface as soft, smooth, and level as possible.
- Emphasize form.
- Ice the involved area 20–30 minutes after running.
- Follow the time guidelines listed below. Do not progress to the next step if the previous one caused pain or compensation.

Previously Running 15–30 Minutes per Day

		DAY							Total Minutes
		1	2	3	4	5	6	7	
Week	1	5	0	5	0	8	0	8	26
	2	0	10	0	10	0	12	0	32
	3	12	0	15	0	15	12	0	54
	4	15	0	18	15	0	20	0	68
	5	20	0	25	20	0	25	0	90
	6	30	25	0	30	25	25	0	135

Previously Running 30–45 Minutes per Day

		DAY							Total Minutes
		1	2	3	4	5	6	7	
Week	1	5	0	5	0	8	0	10	28
	2	0	10	0	12	0	15	0	37
	3	15	0	15	0	15	12	0	57
	4	20	0	20	15	0	25	0	80
	5	25	25	0	30	25	25	0	135
	6	30	30	30	0	35	0	40	165

**Fig. 31.9** Blood Flow Restriction (BFR).

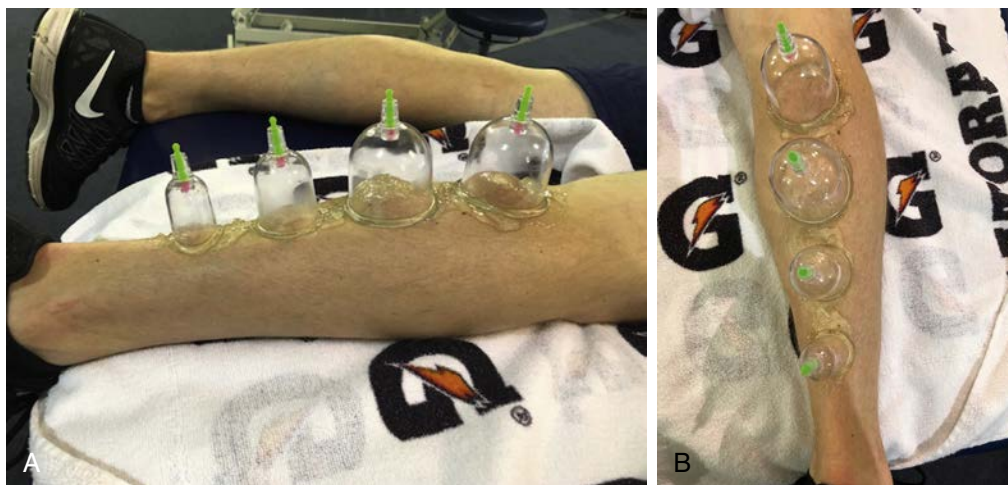


Fig. 31.10 Cupping.

joints. BFR therapy needs to be discussed and approved by the Ortho MD prior to utilizing on the athlete/patient.

A lower-extremity exercise program would be utilizing the BFR in conjunction with an exercise the patient is capable of doing during the rehab process.^{38,39} The occlusion can be set between 60% and 80% depending on patient comfort. Eighty percent is the highest setting used on the lower extremity. The patient will then complete 30,15,15,15 reps with a 30-sec rest between sets or to failure. Some examples of exercises can include Thera-Band ankle strengthening, calf raises (seated or standing), and body weight squats.

The Owens Recovery Science provides education and certification to utilize PBFR training. (www.owensrecoveryscience.com)

Myofascial Decompression Techniques (MDT; cupping) has been used for recovery, pain relief, and soft-tissue manipulation (Fig. 31.10, A and B). The biomechanical and neurophysiological effects of myofascial cupping remain theoretical. Myofascial cupping appears to temporarily exaggerate the condition so as to “kick-start” the physiologic changes necessary to “reset” the tissue.⁴⁰ By propagating the tissue’s hypoxic state, more lactate is produced, thereby increasing the acidity.⁴⁰ In skeletal muscle, the added acidity has been shown to combat fatigue⁴¹ and stimulate nitric oxide release, resulting in improved microcirculation and blood flow via vasodilation.⁴²

Myofascial Decompression Technique is reported by many clinicians to reduce pain and increase pressure-pain thresholds in athletes with myofascial pain and/or localized myofascial trigger points with the effects comparable to other instrument-assisted soft-tissue mobilization (e.g., Graston) and ischemic compression techniques.⁴³ It is usually utilized in Phase I and Phase III rehab as an additional soft-tissue therapy. Typically a practitioner will utilize cupping in conjunction with movement to help decrease trigger activity and improve the quality of movement. Myofascial Decompression Technique therapy should be discussed and approved by the Ortho MD prior to utilizing on the athlete/patient.

Practitioners should take a course before applying MDT therapy to patients. (www.MyofascialDecompression.com)

Functional dry needling is a technique utilized, by qualified practitioners allowed by state law, for the treatment of pain and

movement impairments. The term “dry” needle is used because the needle is inserted in the skin without medication or fluids. It has been most commonly used to treat myofascial trigger points. A trigger point is a taut band of skeletal muscle located within a larger muscle group, which may cause pain and cause muscular dysfunction (Fig 31.11, A and B).

Dry needling will cause the body to release calcitonin gene-related peptide (CGRP), which causes a cascade of reactions resulting in vasodilation, increased blood vessel formation, and increased tissue repair.⁴⁴ In addition, an enkephalin release will occur resulting in a local pain response.⁴⁵ Moreover, a beta endorphin release from the brain creating an analgesic effect will result in an overall systemic response.⁴⁵

Physical therapists use dry needling with the goal of releasing or deactivating trigger points to relieve pain or improve ROM. Dry needling depends upon physical examination and assessment to guide the treatment; moreover, this allows you to test and retest after a dry needling treatment has been performed. It gives you the opportunity to show tangible changes pre and post treatment. Preliminary research⁴⁶ supports that dry needling improves pain control, reduces muscle tension, and normalizes dysfunctions of the motor end plates, the sites at which nerve impulses are transmitted to muscles. It expedites the healing process and repair of tissue while decreasing pain and increasing ROM; this response allows athletes and clients to return back to their sport or activity much quicker.

There are several schools of thought regarding dry needling. My personal experience has been through Kinetacore training. Before utilizing any type of needling, it is critical to go to an appropriate course and become certified (www.kinetacore.com). In addition, you should discuss with the patient’s surgeon before utilizing postoperative.

FUNCTIONAL PROGRESSION

A functional progression is a series of sport-specific skills that increase in the level of difficulty that an athlete must complete before he or she can safely return to competition. Yamamoto and Fragi described a functional progression in the rehabilitation

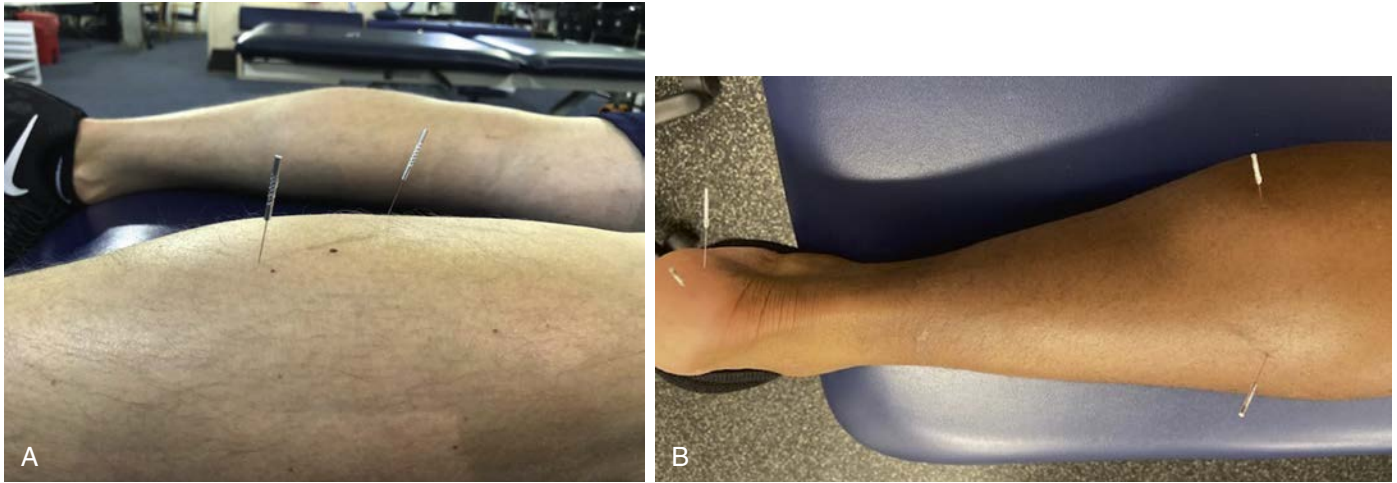


Fig. 31.11 Dry needling.

of injured West Point cadets.^{47,48} The emphasis in this program was placed on restoring agility through dynamic exercise after knee injury. Kegerreis et al.⁴⁹ added specific movement patterns and skills to the program and introduced the importance of addressing the psychological needs of the injured athlete. They also addressed the scientific principles that play an important role in the functional progression and the need to break down sport-specific functions to be addressed in the order of difficulty.

The functional progression is vital to a complete sport-specific rehabilitation program. It serves as the key element in advancing the athlete from clinical rehabilitation to athletics. Each sport has certain demands and skills that stress the foot and ankle differently. Sport-specific functional progressions need to be designed to incorporate the demands of the sport or activity that the athlete will return to. This chapter will include specific functional progressions to show the differences. It is extremely important that the athlete advance one step at a time without pain or apprehension. Once the athlete has completed the list of activities in order without pain or apprehension, he or she may return to full sport activity (Tables 31.3 through 31.7).

There are several physical and psychological benefits that the functional progression will address. The functional progression promotes healing through the application of Davis' law and Wolff's law, which were discussed earlier. It is important that the healing tissue be stressed in the way required of it before injury so that the tissue will be ready to fully accept preinjury activity requirements. As described in Davis' law and Wolff's law, injured tissue and bone stressed in this controlled manner will lead to further tissue and bone healing and strength. In addition, the functional progression breaks up the monotony of traditional rehabilitation and allows the athlete to begin performing activities related to function. Psychologically it allows the athlete to increase self-confidence and mentally prepares him or her to return to sport. As the athlete completes each step, confidence will increase and apprehension will decrease, allowing the athlete to enter the competitive environment at the level of function needed for playing standards.

TABLE 31.4 Field Sports Functional Progression

Methodist Sports Medicine—The Orthopedic Specialists Field Sports Functional Progression

Once you have completed the appropriate phases of rehabilitation it will be possible to begin a functional progression. The functional progression is an ordered sequence of activities that enable you to reacquire the skills necessary for safe and effective return to athletic endeavors.

Begin with step one. If you can do the prior step without pain or limping, you may proceed to the next step. It is very important that you perform each exercise correctly and without apprehension. When you have successfully completed each step of the functional progression you may then attempt to return to your sport. You should wear any protective equipment recommended by your physical therapist or athletic trainer.

1. Heel raises on the injured leg - 10 times
2. Walk at fast pace for 50 yards
3. Jump on both legs - 10 times
4. Jump on the injured leg - 10 times
5. Jog straight - 50 yards
6. Jog straight and curves - two laps each direction
7. Sprint - 1/2, 3/4, full speed - 40 yards
8. Run figure 8's - 1/2, 3/4, full speed - 15 yard distance
9. Cariocas (cross-overs) 40 yards - both directions
10. Backward run - 40 yards
11. Cutting - 1/2, 3/4, full speed for 40 yards
12. Position drills _____

PHASES OF REHABILITATION

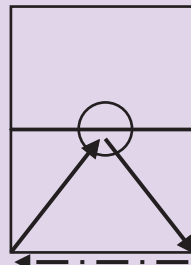
The cornerstone to appropriate rehabilitation is an accurate diagnosis, so that an appropriate rehabilitation program can be established efficiently and safely. For any injury or condition, the rehabilitation can be divided into three general phases. Each phase has specific goals, and although there is a time frame assigned to each phase, advancement from one phase to another should be based on the patients achieving the prescribed goals rather than on time. A clinician must be willing to adapt and modify the exercise program for each patient. There are a variety of rehabilitative techniques to choose from; each can have benefit to the patient. As a clinician, it is important to stay up to date with current rehabilitative trends.

TABLE 31.5 Court Sports Functional Progression**Methodist Sports Medicine–The Orthopedic Specialists
Court Sports Functional Progression**

Once you have completed the appropriate phases of rehabilitation, it will be possible to begin a functional progression. The functional progression is an ordered sequence of activities that enable you to reacquire the skills necessary for safe and effective return to athletic endeavors.

Begin with step one. If you can do the prior step without pain or limping, you may proceed to the next step. It is very important that you perform each exercise correctly, without apprehension. When you have successfully completed each step of the functional progression, you may then attempt to return to your sport. You should wear the Air-cast, Swedo, knee brace, or tape as instructed by your physical therapist or athletic trainer.

1. Heel raises on the injured leg - 10 times
2. Walk at fast pace full court - 2 times
3. Jumping on both legs - 10 times
4. Jumping on the injured leg - 10 times
5. Jog straight - full court
6. Jog straight and curves - two laps each direction
7. Sprint - $\frac{1}{2}$, $\frac{3}{4}$, full speed - full court
8. Run figure 8's - $\frac{1}{2}$, $\frac{3}{4}$, full speed - baseline to $\frac{1}{4}$ court
9. Triangle drills - sprint baseline to $\frac{1}{2}$ court, backward run to baseline, defensive slides along baseline, both directions
10. Cariocas (cross-overs) - $\frac{1}{2}$, $\frac{3}{4}$, full speed
11. Cutting - $\frac{1}{2}$, $\frac{3}{4}$, full speed - full court
12. Position drills _____

Triangle Drills**Phase I**

Phase I emphasizes pain modulation and inflammatory control of the soft tissues. Controlling pain and inflammation will allow patients to be better able to perform their rehabilitation exercises. Restoration of normal ROM and joint accessory motions, including glide, roll, and spin, are stressed in this phase. Early return of pain-free ROM will enhance the rehabilitative process and allow the patient to begin isolated and functional rehabilitation exercises in phase II with greater effectiveness. Once a patient has minimal pain and has normal to near-normal ROM, he or she may be advanced to phase II.

Phase II

Once inflammation is decreased, pain has subsided, and ROM is near normal, phase II may begin. Foot and ankle flexibility with functional strengthening are initiated and are the focus of this phase. In addition, cardiovascular conditioning and proprioceptive training also are started at this time. The goals of this particular phase are to improve flexibility, restore strength, and begin light, sport-specific functional training. A patient may be progressed to phase III when he or she is ready for a gradual return to activity and participation in sports.

Phase III

Emphasis in phase III is on functional return to activities of daily living (ADLs) and previous activity/sport participation.

TABLE 31.6 Ballet Dance Functional Progression**Ballet Dance Functional Progression
Lower Extremity**

Once you have completed the appropriate phases of rehabilitation, it will be possible to begin functional progression. The functional progression is an ordered sequence of activities that enable you to reacquire the skills necessary for safe and effective return to dance. Begin with step one. If you can do this exercise correctly without pain, you may proceed to the next step. It is very important that you perform each exercise properly, without apprehension. When you have completed each step of the functional progression, you may return to dance. Begin with one basic technique class, then slowly add other classics, then rehearsals. If you must do rehearsals sooner due to an upcoming performance, reduce the number of class hours accordingly to a minimum of one technique class.

1. 16 demi plies on both legs then single leg on injured side
2. 16 relevés on both legs, then single leg on injured side
3. Barre activities (limiting sustained releve sequences) or floor work warm-ups
4. Center practice (adagio, barre exercises done in center, standing jazz or modern combinations)
5. Pirouettes or pivoting on one leg
 - a. Promenades, retire balances (pirouette preparation)
 - b. Simple turns from fourth, fifth, or second position
 - c. Soutenu, pique, and chaine turns
 - d. Grand pirouettes (attitude arabesque turns, tours a la seconde)
 - e. Avoid fouettes or repeated relevés on one leg
6. Simple across the floor combinations
7. Jumping
 - a. Double leg low impact — sautés, changements echappes, soubresauts
 - b. Double to single leg — assembles, jetes, glissades, pas de chats, sissonnes
 - c. Batterie (add beats and speed to basic jumps)
 - d. Single leg — repeated single-leg jumps temp levés
8. Pointe work at barre following above progression
9. Pointe work in center- simple relevés, soutenus, echappes, piques
10. Fouettes or repeated turns on one leg not en pointe
11. Grand allegro/traveling leaps
12. Complex pointe work- fouettes, hops en pointe, grands pirouettes

Advanced activity-specific exercise should be implemented with special attention to mechanics of the activity. Proper mechanics, as well as maintenance of flexibility and strength, can prevent further chance of reinjury. To ensure safe return to sport, athletes should perform a functional progression. External supports such as braces, straps, taping, and orthotics may be used at this time to allow the patient to participate in his or her activity pain free.

**An Updated Rehabilitation of Achilles
Tendon Repair**

The research suggests that the incidence of Achilles tendon rupture is increasing and the majority (68%) of ruptures occurred during sports participation (Raiken). Only 4% of Achilles ruptures have an associated Achilles tendinopathy, and two-thirds of ruptures report no pain prior to the rupture (tallun).

The rehabilitation after an Achilles repair is an example of progression toward a more functional recovery. Recently, rehabilitation after an Achilles repair has progressed from long-leg casting to short-leg casting to the use of intermittent

TABLE 31.7 Contemporary Dance Functional Progression**Contemporary Dance Functional Progression**
Lower Extremity

Once you have completed the appropriate phases of rehabilitation, it will be possible to begin functional progression. The functional progression is an ordered sequence of activities that enable you to reacquire the skills necessary for safe and effective return to dance. Begin with step one. If you can do this exercise correctly without pain, you may proceed to the next step. It is very important that you perform each exercise properly, without apprehension.

When you have completed each step of the functional progression, you may return to dance. Begin with one basic technique class, then slowly add other classes, and then rehearsals. If you must do rehearsals sooner due to an upcoming performance, reduce the number of class hours accordingly to a minimum of one technique class.

1. 16 plies (mini-squats) on both legs, then single leg on injured side
2. 16 heel raises on both legs, then single leg on injured side
3. Floor work warm-ups
4. Standing warm-ups and combinations in center without jumps
5. Turning and pivoting on one leg
 - a. Simple turns in place
 - b. Traveling turns across the floor
 - c. Attitude fouettes or similar repeated turns in one second
 - d. Pitch turns, illusions
 - e. Avoid fouettes or similar repeated turns on one leg
6. Simple across the floor combinations
7. Combinations including floor work and transitions to floor (falls, rolls)
8. Jumping
9. Fouettes or repeated turns on one leg
10. Large traveling jumps across the floor (split leaps, stag leaps)

immobilization and early weight bearing. Mandelbaum et al.⁵⁰ have established an accelerated rehabilitation protocol for the Achilles repair. Their protocol involves early ROM at 72 hours and early weight bearing at 2 weeks post repair. This functional approach allows the competitive athlete to return to sports more quickly without a reported increase in complications.

At Methodist Sports Medicine, over 300 acute Achilles repairs have been performed over the past 22 years using an ankle-block anesthetic, no casting, intermittent immobilization with a removable boot, and cryotherapy. Patients have been full weight bearing by 2 weeks, and ROM is started at the first postoperative visit, along with a bike program and sitting toe raises. We use the concept that early-protected ROM and weight bearing encourage strong tendon healing and protect against disuse atrophy. The goal of the surgery is to return full strength, power, and function in the shortest and safest amount of time. The re-rupture rate has been consistent with that of less accelerated protocols (<2%). The general rate of return to strength is 75% by 3 months and 90% by 6 months. This is an example of our rehabilitation program that is consistent with all accelerated programs.

Immediately postoperatively the patient is placed in an Aircast walking boot with a built-in Cryocuff. The walking boot also has one 9/16-inch felt heel lift placed inside to put the foot/ankle in a slight equinus position for healing. (We will use two heel lifts if the repair is 3–8 weeks after the tear.) The patient is

**Fig. 31.12** Toe curls.

instructed to be non weight bearing for the first 5 to 7 days and is appropriately trained in axillary crutch use (or rolling cart for the involved knee if preferred by the patient) for walking and negotiating stairs. This decreases the risk of early postoperative swelling and allows appropriate initial wound healing.

The immediate postoperative protocol consists of rest, elevation, and continuous daytime Cryocuff use. The patient also is instructed to wiggle toes and perform leg lifts every 3 to 4 hours in the first postoperative week.

Dressing changes and rehabilitation will begin 1-week postoperatively. Physical therapy will consist of a home exercise program, gradual progression of weight bearing, and a light bike program to maintain cellular metabolism. The rehab program will start with isometric calf contractions, toe curls (Fig. 31.12), and leg raises in all four directions. After the first 7–10 days the program will include active PF, resistive-band PF (see Fig. 31.4), and sitting calf raises (Fig. 31.13). We use the concept of early-protected motion and resistance training, which encourages stronger tendon healing and protects against disuse atrophy. Exercises are performed at a higher frequency with a low load (see phase I exercise prescription) to continuously stimulate the tendon to heal. It is extremely important to avoid ankle DF activity or a heel cord stretch to protect the tendon from overstretching.

Partial weight bearing is started at 1 week with a gradual progression to full weight bearing at 2 to 3 weeks postoperatively. The first week of rehabilitation allows partial weight bearing in the walking boot with axillary crutches and the amount of



Fig. 31.13 Seated calf raise.

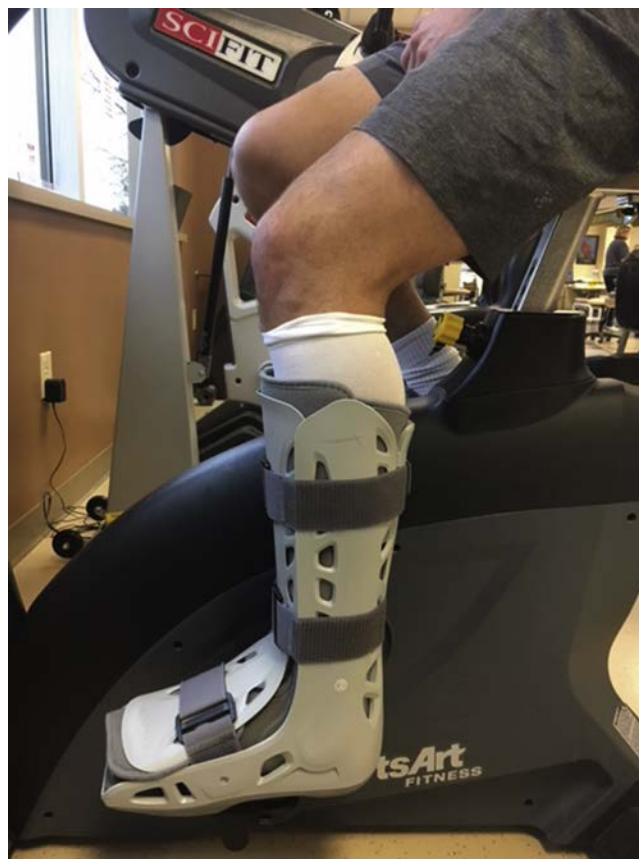


Fig. 31.14 Biking in boot.

weight bearing is increased as tolerated by pain and swelling. After the first week, the patient may begin using one crutch under the opposite arm and then progress to full weight bearing when the athlete is able to walk normally.

A bike program is initiated in the first week using the walking boot. The program consists of 10 minutes three times the first week and increases by 10 minutes per week and to 4 days over the first month. We progress this slowly to give the incision/wound time to heal without increasing the moisture or swelling to the ankle. Once clinical wound healing has occurred, a patient can be more aggressive with cardiovascular activity (Fig. 31.14).

The second phase of rehabilitation begins approximately 6 weeks after repair. At this time, an increase in weight-bearing exercise is allowed, and proprioception retraining with an emphasis on normal gait is initiated. Athletes at this time are instructed in a program to wean out of the boot into an athletic shoe with one 9/16-inch felt heel lift. Our goal is to wean the patient out of the boot over 2 weeks with normal pain-free gait.

Exercises in the second phase consist of balance, standing calf raises, and elliptical/StairMaster progression. Single-leg heel raise performance is critical for an athlete to return to function. Long-term deficits can lead to altered biomechanics and therefore to a decrease in performance (Silbernagel, Olsson). Fifty percent of patients can perform a full single-leg heel raise by 3 months. Single-leg balance (Fig. 31.15) is first initiated barefoot on a hard surface with a goal of

approximately 60 seconds. Once that is achieved, balance is progressed to a soft surface with other possible variations (i.e., ball toss). Patients will begin bilateral calf raises (Fig. 31.16) with a progression to single calf raises (Fig. 31.17). Thera-Band exercise is performed in all directions to incorporate the entire ankle. However, DF past neutral is not allowed. Once completely out of the boot, 1 day of elliptical/StairMaster may be substituted for the bike each week, so that over a 4-week period the athlete transitions into full cardiovascular workouts with a StairMaster/elliptical 4 to 5 days a week. It is important to avoid passive DF or Achilles tendon stretching to protect the Achilles repair from stretching out. We have found that normal DF will return naturally without being aggressive with DF motion.

Swimming is permitted at the 6-week mark for conditioning. A weight-bearing pool program can be initiated at 8–10 weeks depending on the progress of the patient. This program will start at chest-high water and advance to waist-high water as the patient tolerates. The program will consist of light activities, jogging, and eventually running (Tables 31.3, 31.8, 31.9).

The final phase of rehabilitation starts approximately at the 3-month mark. Patients will continue to work on balance, ankle strength, and unilateral calf raises. At this time, full lower-extremity strengthening will be initiated. Exercise will include stepdowns (Fig. 31.18), leg press (Fig. 31.19), knee extensions (Fig. 31.20), and hamstring curls that can be advanced per patient tolerance. Weighted calf raises typically are initiated around 4 months.



Fig. 31.15 Single-leg balance with sissel.



Fig. 31.17 Single-leg calf raise.



Fig. 31.16 Bil calf raises.

Once an athlete is capable of using a StairMaster/elliptical machine for 30 minutes 5 days a week, he or she may begin light jogging (usually at 3–4 months after the repair). A treadmill progression can be started at this time. Once an athlete can complete a treadmill progression with good running mechanics and no symptoms, they can advance to field or court agilities. Agility drills should be advanced gradually per patient tolerance. Before return to sport, the patient should successfully complete a functional progression to ensure a safe return to competition. Return to sports normally occurs at 5 to 8 months after surgery. Typically, at 6 months and 1 year postoperative the patient will undergo isokinetic strength testing in our clinic. It is our desire and experience that almost every patient achieves 80% of PF strength, which is the critical level of strength for return to sport. In our experience, DF strength on the operative side is typically stronger due to the altered strike patterns postoperative. (Fig 31.21, A and B)

Rehabilitation After Open Reduction and Internal Fixation of Lisfranc

The rehabilitation process after a Lisfranc open reduction and internal fixation (ORIF) begins by having the patient non-weight bearing in an AirCast boot for 6 weeks to allow for healing. Crutches or a “roll-a-bout” may be used with an AirCast boot to help the patient maintain the non-weight-bearing status.

Phase I of the rehabilitation will be from day 1 postop to 6 wks. The goals for phase I are to allow healing, control swelling, increase ROM, light subtalar strengthening, and eliminate

TABLE 31.8 Weight-Bearing Pool Training Program**Phase I (warmup 2 lengths)**

Jogging
 High knees
 Butt kicks
 A skips
 B skips
 Shuffle
 Carioca
 Clawing exercise - 2 set 8
 Quick feet scissors - 2 set 10
 Quick fit long stride - 2 set 10
 Quick feet hip rotation - 2 set 10
 Jogging/running - 4 x 10 sec

Phase II (plyo series)

Straight leg plyos (ankle emphasis)
 straight leg - 4 x 8
 single leg - 4 x 6 each
 Diagonal Bounding one 1 leg with stick (bounding over a hurdle)
 3 set 6 (3 per side)
 Waist-deep jumps (2 foot landing-stick)
 3 x 4 jumps/30 sec rest
 Waist-deep jumps (1 foot landing-stick)
 2 x 4 (alternate feet)
 Waist-deep rebound jumps (2 foot landing)
 3 x 4 jumps/30 sec rest

Phase III (conditioning)

Chest-deep running
 8–12 set 30 sec with 15–30 sec rest (start at 30 and progress to 60 resistance)
 Waist-deep running
 8–12 set 30 sec with 15–30 sec rest (start at 50 and progress to 80 resistance)

incision sensitivity. Gentle active ROM into DF, PF, IN, EV, Achilles tendon stretch with a towel (Fig. 31.22), toe curls, and desensitization massage all begin at 1-week postoperative. We use an Aircast Cryocuff in the boot to provide cold and compression. Patients utilize either crutches or a roll about for ambulation in order to maintain their non-weight-bearing status. At the 2-week status postop we will begin seated calf raises, tubing DF, PF, IN, and EV. If the incision is healed at 2 weeks, aquatic therapy can be added as well as icing with a vasopneumatic device.

**Fig. 31.18** Four in step down side view.**TABLE 31.9 Deep Pool Training Program****Program I**

Rep 1- 20 sec run – 20 sec rest
 Rep 2- 18 sec run – 20 sec rest
 Rep 3- 15 sec run – 20 sec rest
 Rep 4- 12 sec run – 20 sec rest
 Rep 5- 10 sec run – 20 sec rest
 Rep 6- 8 sec run – 20 sec rest
 Rep 7- 6 sec run – 20 sec rest
 Rep 8- 18 sec run – 20 sec rest
 Rep 9- 12 sec run – 20 sec rest
 Rep 10- 8 sec run – 20 sec rest
 Rep 11- 20 sec run – 20 sec rest
 Rep 12- 15 sec run – 20 sec rest
 Rep 13- 10 sec run – 20 sec rest
 Rep 14- 6 sec run – 20 sec rest
 Rep 15- 18 sec run – 20 sec rest

Program II

15 reps 20 sec run – 15 sec rest

**Fig. 31.19** Single-leg press.

Phase II begins at 6 weeks and is when weight bearing in the boot can be initiated. The patient will utilize crutches with their boot for ambulation until they are able to ambulate pain free and limp free in the boot. In our experience, the patient is able to wean off the crutches on average in 7–10 days. Proprioception retraining is initiated in the boot. This allows the patient to gain confidence by weight bearing through the affected extremity as well as reactivate the proprioceptors. Biking is also begun in the boot. The patient may progress toward 30 minutes in the boot as their foot tolerates. The patient is instructed in a schedule to

wean out of the boot over 2 weeks into a shoe and carbon fiber plate. At 8 weeks s/p, the patient may bike in an athletic shoe and carbon fiber plate. The patient may gradually add time on the bike using pain as their guide. Once the patient is able to achieve 30 minutes of biking pain free, the StairMaster/elliptical (upright, low impact cardio) is substituted. Aggressive Achilles tendon stretching is initiated in the shoe and CFP at this time (Fig. 31.23). Standing bilateral calf raises in progressing to single-leg calf raises in shoes/CFP begin at 6–8 weeks in order to aid the patient in proper push off as well as strengthening the gastrocnemius.

Phase III begins at 12 weeks. Typically, a functional progression to the patient's particular sport is initiated. The patient must have full ROM, flexibility, and strength in order to begin the functional progression. The patient must also be able to tolerate >30 minutes of cardiovascular exercise on the elliptical or StairMaster on 5 consecutive days prior to starting the functional progression. The functional progression is used to allow the patient to gradually and safely return to sport. Removal of hardware is performed no earlier than 3 months after the initial operation.

Fifth Metatarsal Open Reduction and Internal Fixation Rehabilitation

Phase I of rehabilitation after the ORIF of the fifth metatarsal goals are to allow for fracture site healing, obtain full subtalar and talocrural ROM actively and passively, control swelling, and eliminate incision site sensitivity. The patient will remain



Fig. 31.20 Single-leg knee extension machine.



Fig. 31.21 A, Dorsiflexion cybex. B, Plantarflexion cybex.



Fig. 31.22 Achilles tendon stretching with strap.

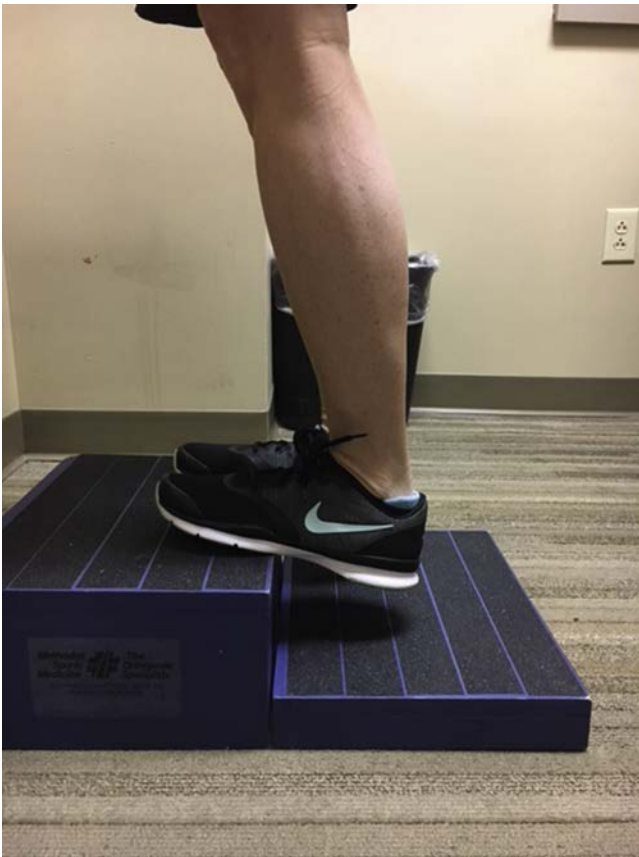


Fig. 31.23 Step stretch side view.

non-weight-bearing for the first 2 weeks postop. The patient will utilize a short Aircast boot and either crutches or a roll-about to maintain their non-weight-bearing status on the operative side. Active ROM DF, PF are initiated at week 1. Seated calf raises, toe towel curls as well as desensitization massage also begin at 1-week s/p. Beginning at 2-week s/p, the sutures are removed. If the incision is healing well, aquatic therapy and use of a vasopneumatic device are allowed. The patient will begin to gradually initiate weight bearing using crutches and the boot. In our experience, patients typically can wean off the



Fig. 31.24 C splint.

crutches and be full weight bearing in the boot in 7–10 days. Proprioceptive retraining is started in the boot. This will help the patient become more confident in weight bearing on the surgical limb as well as reactivate proprioceptors in their foot and ankle. Biking in the boot should begin, and the patient may increase the time on the bike as their foot tolerates and should use pain/swelling as their guide. Thera-tubing into eversion is initiated to begin strengthening the peroneals.

At 6 weeks postop, phase II of the rehabilitation process begins. The patient will wean out of the boot into a running shoe with a semi-rigid carbon-fiber plate over 2 weeks. The patient can begin biking in their shoe/plate and rework up to 30 minutes. Standing bilateral calf raises are initiated to help the patient with toe-off during gait as well as recover strength in the gastrocnemius. The patient will use pain as their guide in order to progress toward a single-leg calf raise. Peroneal strengthening should be advanced in this phase using a side-lying eversion exercise. See [Fig. 31.7, A and B](#). Advanced proprioceptive training on uneven surfaces, ball tossing, or kicking with contralateral foot should also be initiated.

Phase III of the rehabilitation involves return to sport (approximately 8–12 weeks). A fifth metatarsal clamshell splint is made for the patient to wear during all sport activities. The splint is made of perforated aquaplast by the physical therapist. The patient is to wear the splint directly on their skin with their sock/cleat over it. The splint provides support to the screw fixation and decreases stress on the lateral foot and fifth metatarsal ([Fig. 31.24](#)). If x-rays show good healing, the functional

progression may be started as early as 8–10 weeks. If the x-rays do not show complete healing, return to play is delayed until 10–12 weeks postop. The sport-specific functional progression must be passed without pain or compensatory patterns in order to be released to play.

The carbon fiber plate is discontinued for everyday activities at 12 weeks postop. The athlete is to continue to use the plate for all sporting activities until 6 months postop.

High Ankle Sprains

Phase I (7–10 days)

We place the patient/athlete in full walking boot. We teach proper gait mechanics in the walking boot. We emphasize heel to toe to avoid hip rotation and turn out of the leg. If the patient still has antalgic gait pattern, we utilize axillary crutches or one of strand crutches. The goal would be to move from two crutches, to one crutch and eventually full weight bearing as soon as the patient can walk normal. The idea of early weight bearing will help stimulate the lower extremity muscles to help diminish any additional strength and atrophy concerns. The boot allows for protected weight bearing and will allow the ligament to heal and avoid rotational stresses.

Early rehabilitation goals focus on pain and swelling reduction while starting some specific ROM and exercises. During the first 3 days we will focus on cryotherapy (Game Ready, ice, etc...) and compression. We will also use Hivamat, soft-tissue massage, and other compression modalities to help promote lymphatic drainage and decrease swelling. The early rehab consists active and passive DF, towel toe curls, seated calf raises without resistance to comfort, and tubing for DF. During the week we will add PF ROM and tubing as the patient tolerates. Some additional exercises to help the lower extremity are multidirectional leg raises, knee extensions/leg curls, and core work. We will start the patient on a bike program once they can walk full weight bearing in a boot.

The home exercise program consists of active ROM for DF and PF. Passive DF or calf stretching can be started with a stretch strap or towel.

Phase II (10–21 days)

The plan is to discontinue the boot for activity and work into a brace. There are several bracing options that can be used from a custom AFO to a lower-profile DonJoy velocity or Ultra. Proper gait instruction is always necessary to help decrease substitution patterns. The patient can still rest in the walking boot if soreness persists.

Rehabilitation goals will be to focus on restoring normal joint mobility, ROM, strength, and proprioception. Introduction of grade II–III joint mobilizations can help improve any subtalar joint stiffness from the walking boot. It will also help improve the active range of motion (AROM) and passive range of motion (PROM) of the ankle. Range of motion will focus on DF, PF, and IN. The patient will work on eversion and progress as tolerated, with some attention to external rotation of the foot/ankle on the lower leg. Calf flexibility is critical to rehab and prevention of other issues. Passive stretching can be performed several ways from a wall stretch,

TABLE 31.10 Treadmill Warm-Up & Progression

Move incline to 1.0		
Warm-Up: This is done consecutively		
1 Min Each Speed		
3.0 mph, 3.5, 4.0, 5.0		
Progression starts at 6.0 mph and bouts are 30 sec run with a 30 sec rest in between.		
We increase by .5 mph every run up to 10.0 mph, then the run time decreases to 15 sec run with 30 rest. The top speed that we like to see for Lineman is 12.0 mph and up to 14 mph.		
If the athlete is running better but has not made it to the top speed but has run a couple of times, the .5 increments can be skipped at the lower speeds. We also like a 2 min cool down at a brisk walk or light jog.		
3.0 – 1 min	7.5 – Run	30 Rest
3.5 – 1 min	30 Rest	10.5 – Run
4.0 – 1 min	8.0 – Run	30 Rest
4.5 – 1 min	30 Rest	11.0 – Run
5.0 – 30 sec	8.5 – Run	30 Rest
5.5 – 30 sec	30 Rest	11.5 – Run
6.0 – 30 sec	9.0 – Run	30 Rest
6.5 – Run	30 Rest	12.0 – Run
30 Rest	9.5 – Run	2 Min Cool Down 3.5 mph
7.0 – Run	30 Rest	
30 Rest	10.0 – 15 Sec Runs Start	

stair stretch, or utilizing a slant board. Stretch times should range between 3 and 5 minutes with either using a sustained stretch or booted stretching if sore. Strengthening will follow the same pattern with Thera Band, manual resistance, or sand weights for DF and IN. Weight bearing exercises will focus on calf raises (double to single), single-leg balance progression step-downs, and bosu squats to begin functional training. Additional lower-extremity strengthening can be started in a regular shoe that includes leg press, squats, lunges, multi-hip strength, and core.

At this point we will advance into more cardio and a functional progression to return to running activity. The progression starts with elliptical or StairMaster in a regular shoe with a brace. Training time will start at 15 minutes and advance 5 minute every 2 days as tolerated. We will also introduce pool agilities and running at this time. The patient will begin in chest-high water and then progress to waist-high water. Typically, the patient will do well with these activities. Once they tolerate back-to-back stairmaster/elliptical for 20–30 min or pool running, we will advance them to a treadmill progression (Tables 31.10, 31.11). Our goal is pain-free normal running mechanics at 12–14 mph before we consider advancing to field drills.

Phase III (3–6 weeks)

The focus will continue to be on full restoration of ROM and strength. Aggressive joint mobilizations and stretching to help get end ROM back. Calf stretching can be accomplished by

TABLE 31.11 Functional Progression Guidelines**Phase I: Elliptical Progression**

Elliptical 20–30 minutes

2 consecutive days without setbacks → progress to Phase II

Phase II: Treadmill Progression

Treadmill warm-up

Progressive speed intervals 7–9 mph, increase in .5 mph increments 30 second run/30 second rest

Progressive speed intervals 9.5–14 mph, increase .5 mph increments 15 second run/30 second rest (7–10 mph), 10 second run/15 second rest (10–14)

If player completes 12–14 mph without hesitation and pain, move to Phase III

Phase III: Field Functional Progression

Jog 2 x 50 yards

Perform warm-up exercise

High knees 2 x 10 yards

Butt kicks 2 x 10 yards

A skips 2 x 10 yards

B skips 2 x 10 yards

Back pedal 2 x 5 yards

Lateral shuffle 2 x 10 yards both directions

Cariocas 2 x 15 yards both directions

Double-leg jump x 10

Single-leg jump x 10

3 x 40 yards accelerations (50–75-full)

2 x 30 yards arc left 10, arc right 10, sprint straight 10

2 x 5 yard zig zags for 25 yards

W drill both directions

Triangle drill both directions

Individual period specific to position/sport

If player can complete all aspects of progression without hesitation and pain progress to Phase IV

Phase IV

Conditioning/Return to Practice

a slant board, wall stretch or a simple stair stretch (see Fig. 31.23). Calf stretching should range from 3 to 5 minutes. Plantarflexion stretching can be accomplished by sitting back on your heels. Strengthening should still be addressed in all directions. We also like to include working on resistance to IN of the ankle on the lower leg in both a DF and PF position. Also, advancement back to weight room exercises that include squatting, lunges, and dynamic step-ups in multiple directions. Some advanced functional strengthening will include shuttle jumps, mini tramp jumps, slide board, box jumps, and landing.

We will continue to focus on cardio as we progress the patient to field work. The field work may include basic field agilities, cutting, ladders, hurdles, and cone work. A field test will be conducted once the patient has become comfortable with the field work. This test will include full-speed work, change of direction, and sport-specific drills. If an athlete can complete all of the field progression at full speed and look normal, they will be cleared to return to full sport and activity. Time table will change based on the demand of the sport and even the position in the sport (i.e., offensive line versus wide receiver).

Surgical Intervention for Syndesmotc Disruption With Ligament Damage

Phase I (1 month)

The patient will be placed in a walking boot and will be non-weight bearing for the first month postop. The goal will be to allow the repair structure time to heal without placing weight on the ankle. Weight bearing too early can put too much stress on the ankle joint, causing the joint to spread. We will focus on controlling soreness and swelling with ice or game ready. We will monitor the wound and follow basic wound care guidelines.

During the first 2 weeks, we can start active toe ROM, light desensitization massage, multidirectional leg raises, and core work. At weeks 3–4, we can start some AROM for ankle DF/PF. We can add some light towel/strap stretching for the Achilles and calf complex. We will start some seated calf raise without weight to stimulate the calf some. At 2 weeks the athlete can start bike conditioning in the boot. When the wound is healed, deep-water conditioning can be started without flippers.

Phase II (2–3 months)

We will begin AROM and PROM for the ankle in all directions. We will monitor eversion or external rotation of the ankle on the lower leg. The goal will be to restore normal joint mobility during this phase. Grade II–IV joint mobilizations to help advance ROM. Calf stretching can be accomplished by a slant board, stair, or basic standing wall stretch. Multidirectional ankle strengthening can be started with tubing or manual resistance. Advance as tolerated and monitor pain and soreness with IN. If the athlete is uncomfortable with IN resistance, gradually add it into the rehab program based on soreness. We want the exercises to be comfortable.

The athlete can advance the weight bearing status during this phase. They will progress from two crutches to one crutch in the first week and then to full weight bearing during the first 2 weeks (4–6 weeks). The athlete will continue to utilize walking boot during this time. Over the next 2 weeks (6–8 weeks) the athlete will begin to wean out of the boot into a brace. Some of the more common braces include DonJoy velocity, Ultra, ASO, and the Arizona brace (Fig. 31.25). There are many other brace options that can be considered. The brace used will depend on the desired support and protection needed as determined by the physician. Weight-bearing exercises include calf raises, step-downs/ups, and proprioception activities. We will start with double-leg calf raises and progress to single-leg calf raises as tolerated. The athlete will focus on good eccentric control of the calf raise. Step-downs/up will be initiated at this time to focus on some single-leg control and strengthening. The step-up is generally easier for the athlete and will help improve push-off and power through the foot. Step-downs will focus more on balance and control as the athlete lowers through good hip, knee, and ankle control. Balance exercise will include a progression from balancing in the walking boot to a regular shoe and then finally barefoot. We will also challenge the balance surface (hard to soft).

At this time the athlete can start a general lower extremity (LE) strengthening program. The program can include shuttle



Fig. 31.25 Elliptical in ultra brace.

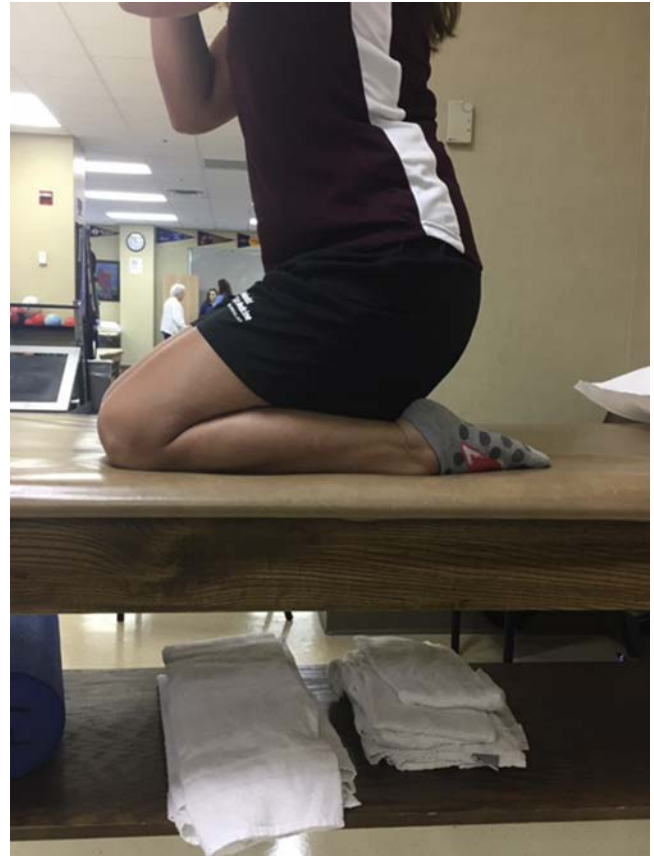


Fig. 31.26 Sit on heels.

press, leg press, knee extension, leg curls, and multidirectional hip strength. During the early part of this phase they can start these activities and work in the boot. They can progress into a regular shoe as they move through the wean-out-of-boot program. Cardio exercises should include bike, StairMaster, elliptical, and pool activities if available. A general progression on the cardio equipment would be 3–4 times per week, starting at 15 minutes. We will advance both time and resistance as tolerated. We like to get athletes to 4 days of 30 minutes on cardio equipment before advancing to treadmill work. A treadmill progression can be initiated in this phase if the athlete is ready. The treadmill progression will include an interval speed progression.

Phase III (3+ months)

During this phase we want to make sure that we restore normal joint mobility and ROM. Aggressive stretching for both DF and PF (see [Figs. 31.23 and 31.26](#)) should be continued throughout the process. Basic ankle strengthening should be continued with tubing, manual resistance, and other weighted activities. Single-leg balance and strength work should be initiated and advanced as tolerated. Balance can include different surfaces, catching a ball, bosu squats, and opposite-side hip tubing while balancing. Slide board can be initiated to improve single-leg push-off and stability.

Strengthening activities should be monitored for good form and ankle mobility. Corrective exercises should be

completed prior to advancing into heavier lifts to ensure symmetry and balance. The exercises can include a variety of squats, double/single-leg deadlifts, lunges, cleans, and squats. In addition, a plyometric progression can be started that should include landing mechanics and jump mechanics. This progression should move from double-leg to single-leg work.

Functional activities and field work should be initiated once the athlete has passed a treadmill progression for time, volume, and running mechanics. Field drills should begin with basic agility drills and ladder work. Ladder drills will help with ankle position and quickness. If the athlete tolerates those activities well, then change of direction, deceleration, and arc running should be initiated. The final progression will include sport-specific drills and function. Once an athlete has completed all phases of the field progression, they will be cleared for sports activity. Isokinetic testing is performed in DF, PF, IN, and EV to evaluate symmetry between the involved and non-involved sides (see [Figs. 31.21, A and B, 31.27, A and B](#)).

Once an athlete has returned to full activity, a maintenance program should be carried out until he or she feels a 100%. This program should continue to focus on joint mobility, aggressive ROM (especially DF), ankle strengthening, and balance. Sometimes athletes will need some extended warm-up time prior to sport participation. We will continue to utilize taping or bracing until completely comfortable.



Fig. 31.27 A, Ankle cybex eversion. B, Ankle cybex inversion

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The Team in the Care of the Athlete

David A. Porter

OUTLINE

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When we set out to be a team physician or to take care of the athlete with foot and ankle problems, it is a noble deed. However, it is important to remember, we are just one part of the *TEAM* that takes care of the athlete. We play a critical role both diagnostically and therapeutically, but we cannot misunderstand our role, misplace where we fit in the care, or in any way minimize the role so many others have in the care of the athlete.

From a distance, it can appear glamorous to be involved in the care of some of the greatest athletic specimens who have ever walked this earth. It can be incredibly rewarding to be a part of a great outcome and see the results literally played out on a field or court right before you and the world. There is so much at stake for the player and his/her team, owner, and administration. However, I always remember what my mentor for my Masters and PhD in Exercise and Sports Performance Physiology, David L. Costill, PhD, told me, “You will likely be remembered more for the mistakes you make than the successes you enjoy!” We are supposed to be successful, but when an outcome is not so great (and we all have them), increasingly the media and fans react harshly and unsympathetically.

Just like most facets of medicine, taking care of the athlete is a lot more about work, tireless and selfless service, and not so much about glamour! That’s okay. We still get plenty of moments when an athlete will say to us, “Thanks, Doc, for keeping my wheels a-turning, so I could do all I wanted to do!” That really is reward enough. Put all the marketing, recognition, fame, and fortune aside, and the personal involvement we get to have in the lives of these amazing athletes, more importantly, these amazing people and families, is why we chose this field to begin with.

I have thought a lot about all we present in this book regarding the science, studies, innovation, and analysis that we do, all so that individuals can have a better outcome and have a “normal” life (even when “normal” can mean trying to help an athlete get back to a level only experienced by less than 1% of the population). I thought it only appropriate that we take a little interlude and talk just a bit about *HOW* we care for and about the athlete. This is my feeble attempt to discuss our role and

how consultants team physicians interact in the care of the athlete. I have chosen to go to those we actually serve and provide care for in this endeavor. The remainder of the chapter is taken from interviews I conducted with an athletic trainer, general manager, agent, professional player, colleague, and a generous parent. I have interjected stories and anecdotes that I personally have experience that support and give example to the points made by the kind folks I interviewed. So please understand some of what you read is a bit editorialized by me, and I think it will be obvious to you what are my examples and what are the **key words** enlightening our understanding from the contributors. I hope you enjoy it and can agree with much of it, or at least be challenged by their thoughts, concerns, and needs.

BILL POLIAN (FORMER NFL GENERAL MANAGER AND EXECUTIVE)

Competence: The doctor has to be good! He or she must be right most of the time! Specifically, these are athletes, and it’s not enough just to know about their foot and ankle condition from an orthopedic standpoint; we have to know the sports medicine context. In its unique demands and contexts, sport creates extreme demands on the same structures, causing them to be affected differently. The sports medicine physician and health care provider need experience and understanding of what goes on for that specific athlete, realizing that each athlete and position is different. I remember when a ballet dancer came to our clinic for a lateral ankle sprain. She was diagnosed appropriately, given appropriate guidance regarding the natural history, was explained the anatomy of the injury, and was sent to physical therapy where she was given a large bulky brace and told to come back in a month if it wasn’t better. She went home and was in tears. She had a performance in 3 weeks, which was before her next visit with her doctor to clear her to dance. She cried as she put on the brace the first and only time as she saw clearly she could not dance “in that thing!” Now, our approach is different. We get our dancers in with a physical therapist who is a former

dancer, use a low-profile support for the ankle, start them on Barre work immediately, see them back every 1 to 2 weeks to ensure they are clear and safe to dance, and work directly with the therapist to follow their recovery.

Honesty: How bad is it? What is the prognosis? How quickly can the team count on the player? What does the future hold? We can't just be optimistic. We need to explain the worst-case scenario and the realistic outline on how to plan for this week, next week, and the weeks to come! These are the questions the team asks and wants to know about. The need for prognosis and timetable is critical both at the recreational, collegiate, and professional levels. Not only are we setting practical expectations for the athlete and the team, but by giving an honest prognosis, we are giving the staff and administration time to logistically change their program if necessary. The next man or woman up needs clarity so he or she has time to prepare, while the coaching staff and management need time for roster adjustments, if needed. The list goes on and on, which is why honesty is essential to building a strong "team" and reliable approach. There was a time when one of our physicians had to tell a player that his spine condition would not allow him (the player) to safely continue to play his collision sport. Mr. Polian mentioned how impressed and thankful he was with how the physician handled the honesty of the situation but with empathy and compassion (Fig. 32.1).

Neutrality: We can't just be a fan. Though we love cheering for our teams or players, we must remain objective when treating athletes. We have to be able to keep a level of detachment from the desire to see our patients "get back quickly" and do what is best for their long-term good. Essentially, we should have passion for the athlete, not for the score. We are physicians, surgeons, and health care professionals, not coaches, owners, or even just fans. Our professionalism and skills are tools that allow us to perform in the best interest of the athlete. Usually everyone can tell when our interests are divided, which sets us up for failure. A distrust can form between us and the patient/athlete if they feel we have our own agenda. They need to know that our number-one priority is their well-being and not the name on the front of their jersey or the boost in our name or recognition.

Empathy: Can you understand the significance to the athlete? Can you see the situation as they see it? It is easy to just

see a problem when dealing with any medical issue, leading to the mind set of "that part is broken, and it's my job to fix it." Though that mindset is technically not wrong, the situation is so much more involved and delicate than that. When athletes end up in our offices, they have just experienced something that has the potential to change the trajectory of their entire career path and possibly their long-term health. They will be worried about the next step, stressed about wanting to return to play, and they may even be scared. Not only is it our job to treat their physical needs, but we must also treat the needs/fears/concerns that x-rays and magnetic resonance imaging (MRI) do not show. To do that, we must empathize with them. We need to be able to understand what this means for the athlete and the additional stresses/concerns/worries that come along with injuries. They are not just a fractured foot or a sprained ankle, so we should not see ourselves as just a technician there to fix an injured part. We need to remember that every injured foot, ankle, or toe is attached to a real human being.

Selfless: It not about the "doc," it's about the athlete. The proliferation of interest in the health of competitors and players, and its impact on play and entertainment, is astounding! Getting your name in the paper for treating a well-known athlete can seem almost dreamy, but it can turn into a nightmare when "things go a bit wrong." Again, this is another area where reminding ourselves of the importance of our professional relationship is critical. The athlete came to us for medical care, the team is counting on us to give the highest quality of care. Marketing of ourselves, at a minimum, plants a seed of self-interest but can lead to a deterioration of the entire patient-physician relationship.

Communication: Check your pride at the door. Communication is not about which "team member" gets all the glory; it's about upholding the highest ideals of collaboration and treatment. So, get the language right and do it in a way that works. Speak a common language and develop a communication method that works for all involved. If communication is lacking the first few times, do not just give up, but work at understanding a common language for whomever you're communicating. Be creative; analogies can help the non-athlete a great deal. For example, an osteochondral lesion (OCL) of the talus can be analogous to a pot-hole in the road. We all (especially those of us in the Midwest!) have experienced the bumpy ride our vehicle takes over an uneven surface or pot-hole. An athlete or parent can understand better how an OCL might cause pain or inflammation from this perspective. Talk to others on the team and see what will help them understand better. Do not fall victim to communicating in a silo (speaking in a way only a medical professional can understand). Poor communication only leads to confusion and less than ideal outcomes. Additionally, encourage the athlete to ask questions, not only so they feel integral in his or her own care, but so you may truly understand the issue at hand. This is especially important when delivering the "your career is over" message. First, we need to make sure this is the best decision for all involved and we are doing right by everyone. Then, we must make sure our message is delivered in a clear and compassionate way. Again, we must remain neutral from an allegiance standpoint and focus on the well-being of the athlete.



Fig. 32.1 Bill Polian. (Courtesy Bill Polian and the Indianapolis Colts.)

DAVE HAMMER, ATC (HEAD ATHLETIC TRAINER, INDIANAPOLIS COLTS)

Communicate: Letting the team and the head athletic trainer know what you think and getting back with them on a timely basis is critical to how the athletic trainer can interact with the athlete and the team including the athletic training staff, the coaches, and the general manager. The athletic training staff have to treat the player daily and at times more than once a day. The coaches need to know who they can count on for practice and the upcoming game. The general manager needs to know if there is a need to replace the player on the roster. At a particular point in the season, is this an injury that will require the player to be replaced permanently or temporarily? Typically, we as consultants or team physicians will need to be able to contact the responsible person with the team within 24 hours of being asked to review information or obtaining the information. If the player is seen by you in consultation, a phone call is expected the same day as the consult. As you can see, this can involve a lot of calling. When I was a fellow with Don Baxter, MD, in Houston, TX, we were consulted on a Division I athlete from my home state. He asked me to handle all the calls and keep track of how many calls were made. From the time of the first call until the time we performed the surgery and got the rehab set up post-op, there were nine calls I had made or fielded. It helps to somewhat enjoy communicating, as it may often involve calls at night, on weekends, or in a car as you travel. Communication is key! It builds trust, reliability, and clarity (Fig. 32.2).

Be Definitive: Take a stand so there is a directive to act. For any of us or our ATC/PT colleagues to treat and have a directive to care for the athlete, we need a firm diagnosis. I remember during my training, Frank Mannarino, MD, of Dayton, OH (Sports Ortho MD), was always asking me when I came out of the athlete's exam room, "What is the diagnosis?" I wanted to tell him all that I had found out, but what was important was, *could*



Fig. 32.2 Dave Hammer, ATC. (Courtesy of and permission by Dave Hammer and the Indianapolis Colts Used with permission of the Indianapolis Colts.)

I get the right diagnosis? It taught me to interview the athlete and examine in a way that leads to a diagnosis. We are not the reporter of the athlete's complaints but specialized analysis experts who are discovering what has gone awry. It goes without saying that we have to give the player and his team a clear prognosis, an expectation within a reasonable range of when the athletes will be able to practice and play again. Being correct in this way over time builds confidence in us and helps everyone set realistic goals.

Be Realistic and Honest: Don't say what you think someone wants to hear. We often want to please everyone, and there is a temptation to say what we think the player wants to hear. Then when we talk to management, coaches, athletic trainers, etc., we say what they want to hear, and it leads to mismatched expectation and frustration. Consistency in communication is critical to create a unified approach. Someone in the process may not be as pleased, but honesty, in my opinion, it serves everyone best. It is difficult but necessary to be honest with everyone. There are different goals sometimes, and different impacts, but I have found keeping it straightforward, realistic, and consistent serves everyone best and creates peace and unity.

Be Effective: Be right more often than wrong. I realize everybody is wrong occasionally, but we can't always be wrong. This area is not an area of just effort and goodwill. Keeping up professionally, using your intellect, attending professional meetings, having good, smart, and up-to-date colleagues are invaluable in order to be effective. Some of this may be giftedness that we can't fully change, but training ourselves, being life-long learners, and humbly seeking advice, in my experience, can go a long way in augmenting our natural abilities. Evaluate your outcomes on a long-term basis by following up; having your patients assess their subjective outcome is also a great way to continue to hone our skills and process for outcomes (rehabilitation, surgical approaches, return to play process, etc.). As we all know, we can often learn more from our mistakes and poor outcomes as long as we are honest about it and face up to those that don't do as well.

MARK BARTELSTEIN (PROFESSIONAL AGENT)

Collaborative: We are in a profession of the best, taking care of the best. Realize we need to work with the others in our field and related fields, strength training, athletic training, nutritionist, exercise scientist, etc. Can we put our egos aside? It is often more of an art than just a science. Injuries can be unique, so it is important to talk to other professionals and get a good amount of input from other experts in order to get the best opinion. It cannot be "my way or the highway." Again, we must leave our pride at the door. We have to have the humility to look at the problem from all different angles, including how nonmedical people look at it. For example, can we look at a problem and ask ourselves, "Can we plate it and allow the athlete to get the same result with a quicker return?"

Communication/Accessibility: Athletes get hurt at all hours of the day and all days of the week. They need peace of mind, so don't be in a hurry. Sports medicine physicians need to understand injuries are career impacting; therefore, we must communicate every step of the way. Many agents can be very heavily involved throughout the process, so it is important to make sure they are in the know, just as much as the player, athletic training

staff, and coaching staff. Depending on the agent–athlete relationship, the agent may be the one who helps the athlete make the final decision. Obviously, having approval and permission from the player to speak to his/her agent are necessary before any conversations of this nature (Fig. 32.3).

Education: As medical professionals, it is important to educate all involved, including the agent. Weigh the risk and benefits with the athlete, agent, coaches, and other members of the team so they are able to clearly see the options available. It may be a long, thorough process, and that is okay. Those involved need to understand the situation and options so they can help make the best decision. Always make sure to explain the procedure, whether it is fixed surgically or by trying a new orthobiologic. Don't gloss over it! Other members of the team look to us, as the acting physician, to hold the knowledge of the injury, treatment, and recovery. They expect that you will educate them and lead them in the right direction. We cannot just leave them hanging with little or no explanation. "This is the way we do it" doesn't work for an explanation!

Serving the Master as the Athlete: Though there are lots of influences, we need to remain focused on the athlete as the patient. The team is the employer, which means they have a significant stake and influence on all involved. However, we have to maintain the balance. That includes giving agents a peace of mind that we are doing what is in the best long-term interest of the athlete. We can't be seen as just "employees of the team."

Competence: We all know there is an art to treating athletes, but being competent is much more than that. We have to give a reasonable timeline of treatment and recovery and be able to hit the milestones we have set! One way to do this is to remain up-to-date with techniques, innovations, and research. Alan Habansky, MD, from Muncie, IN, has been a great mentor to me and was one of the first orthopedic sports medicine physicians in the country.

He used to tell me, "You don't always have to be first to do something, but you should not be the last to do it either!" So, be competent with your skills and keep up with innovation and testing that are coming out. I find it very helpful to talk directly with those who are working through a new procedure or technique and find out what really works for them and what to be aware of also. Developing a network of colleagues around the country and the world has served my patients well with how I have continued to grow and develop as a physician and surgeon. There should be some areas that each of us are developing in our field, but we are not going to be the latest expert on every injury we treat. However, we can and should be part of the greater conversation that is going on in the medical field in which we work. Researching our own patient outcomes and pursuing academic understanding and shaping our field with our expertise takes time, effort, organization, and money. But it is necessary and worth it!

GORDON HAYWARD (PROFESSIONAL ATHLETE)

Transparency: The player wants to clearly know the diagnosis. "What is actually going on?" "What is wrong?" It is important that the athlete is not told something different than management and the athletic training staff. It usually becomes obvious if the athletic training staff has a different opinion regarding the player's return and eventual outcome. Transparency also means educating the athlete on the anatomy and normal outcome at the level the athlete can understand and is interested in. Some athletes want more than others, but an inability to clearly explain the problem and expected outcome creates some mistrust. It is important to bring "peace of mind" so the athlete has confidence his physician understands his or her problem and that the plan is clear and "makes sense." Setting a realistic expectation regarding recovery is important right at the outset of the treatment (Fig. 32.4).



Fig. 32.3 Mark Bartlestein. (Courtesy of and permission by Mark Bartlestein and Priority Sports Courtesy of Julie Magrane.)



Fig. 32.4 Gordon Hayward. (Courtesy Gordon Hayward and the Boston Celtics.)

Confirmation and Clarity: As the treatment unfolds, the patient will ask, “Am I on track?” “Am I coming along like I should?” If you can relate, confidentially, to the athlete regarding other athletes that have had a similar injury and what their outcome was, it helps build confidence and confirms the right treatment. If the doctor can set good, trackable goals, follow-up evaluations can be a time to confirm the set goals are being met. Step-by-step progress is important because the athlete cannot get back to play all at once! It takes time. This often has to be reiterated, so small goals are key in helping the athlete’s rehab because they delineate the timeline and assure progress is being made.

Empathy and Care: The athlete wants to know his doctor understands his/her situation particularly. We, as physicians and/or surgeons, see a lot of the same injuries, but the athlete needs to know he or she is not just a problem, but a person with an injury. Do you understand “my situation?” Teddy Roosevelt is famously known for the adage, “People don’t care how much you know... until they know how much you care!” We need to keep that in mind. A stoic, distant, unemotional interaction may not communicate to the athlete a genuine empathy and can put up a barrier to trust. Outcome depends on mutual respect and cooperation. Cooperation does not happen if our patients don’t believe in us and our treatment.

BOB ANDERSON, MD (ORTHOPEDIC FOOT AND ANKLE CONSULTANT TO NFL AND NBA)

Don’t let your ego get in the way: The very fact we have made it to this point in our career indicates that we have some God-given talent and ability. The years of training where we have succeeded compared to others can lead to pride and an inflated ego. We need to recognize our strengths and weaknesses. **Refer when uncertain.** I remember two situations when I was asked to see an elite high school athlete and an elite professional athlete. I understood both of the problems the respective athletes were facing, and I thought surgery was the most reliable solution in each case. But it was not the type of surgery I performed a lot, so I recommended they get the surgery with a different surgeon. The high school athlete’s family was a bit condescending toward me, saying, “I thought you could do everything.” My pride did well up inside me, but I stood my ground because I knew the athlete would be better served elsewhere. The professional athlete was actually very grateful and went to the other surgeon I recommended, with whom he has since had a great result. Much like sports teams, there are specialties even in coaching and on the field. Know where we are strong and where we need to refer.

The Player Is Patient #1: Similar to “empathy” as noted by Gordon Hayward and Bill Polian, we have to remember first and foremost that these athletes are people and patients. It could be easy to get caught up in all the surroundings of the player’s career and all the other individuals involved in their life. But remember, they are still patients that are looking to us for a correct diagnosis and a clear treatment plan to help them overcome their injury or illness. If we can keep in mind that these elite athletes are people who need our attention and expert medical care, much of the other issues fall by the wayside very easily.

Be a Team Supporter but Not a Fan: Most of us have gone into sports medicine because we are fans of sports. However, in our roll as physicians and health care providers, we have to be a supporter of the teams we cover, as well as those we provide opinions for. However, we can’t treat the patient for the hope they might provide for our teams. Rather, the treatment for the athlete must be objective, clear, and in the best interest of the athlete’s health. I have found, in the long run, this is the best way to support the team, as well. There could be a temptation to want our team to benefit from our athlete’s recovery to a point that it could “cloud our judgment.” This must not happen! Another way of saying this might be, “**Stick to what’s right for your patient**” and stand your ground to parents, coaches, and agents, among others.

Listen. Watch. Educate: To help the player and his or her family make a decision, you must first listen, watch, and then educate. We need to always remember that this athlete is a person in a unique situation with individual and team goals that are specific to his or her situation. We must **listen** to what their situation is and understand the time frames that are involved. For instance, the late-season Jones fracture might be amenable to supportive care and a wraparound orthoses for a high school student to finish his senior year. During off-season, however, the Jones fracture that is picked up on a routine image might be best served with a more aggressive surgical approach so that it does not re-fracture during the season. An in-season Jones fracture will require operative intervention, but the athlete and the team must understand the risk of returning in the same season, which could lead to a higher risk of re-fracture and possibly a second off-season surgery. We need to **watch** the interaction that the athlete has with family and team. It is important to also look closely on physical exam at predisposing factors for injury. After we understand the athlete’s situation and have done a very thorough examination, we then can **educate** the athlete, the family, the team, and all those involved regarding clear options that the athlete can then choose to follow. Good education allows all involved to understand the options and the treatment course that best fits their needs and desires. This obviously takes time. A rushed interaction and exam could provide the correct diagnosis but create a lack of trust if we have not **listened** well, **watched** well, and **educated** well (Fig. 32.5).

Offer Second Opinions: Be Content (and relieved) When an Athlete Goes Elsewhere for Surgery. Second opinions can be extremely valuable for athletes. It often seems like a bit of a hassle, but it can be an excellent way to bring clarity, confirmation, or a new approach to the athlete’s injury or illness. I remember one of the general managers that our group worked with telling us, “I love whenever our players go get a second opinion outside of our team. They always seem to come back with the even more trust in our physicians and our medical team’s approach.” There is a lot of communication required with second opinions, and all the other issues we have discussed come front and center with giving a second opinion: that is, understanding the situation of the player and the team, being empathetic toward the player, putting the player first, checking our pride at the door, and being skilled in our diagnostic and treatment acumen. We should also feel comfortable when our



Fig. 32.5 Bob Anderson, MD. (Courtesy Robert Anderson, MD.)

patients seek second opinions. It can be a relief for the patient to be treated elsewhere. I remember, when I first started in practice, I sometimes tended to second-guess myself if my patients sought other opinions. Early in my career I was seeing one of professional football players who had a significant midfoot injury. I diagnosed him with a Lisfranc instability that required surgery. The patient wanted another opinion. I actually was able to be on the phone with the second-opinion physician who fully supported my view and said this approach was correct. What started off as a bit of a fearful and insecure situation ended up giving me a great deal of support and encouragement in the direction I recommended. I ended up treating the patient, and he did very well. I have never forgotten that interaction. Bill Hamilton, MD, was the orthopedist who provided that second opinion and supported me early in my career.

We Can Clear Based on Our Specialty: As an orthopedist, we clear “orthopedically.” The physical therapist decides when the recovering patient’s strength and range of motion are appropriate. Athletic trainers clear regarding functional progression once the patients are cleared medically and orthopedically. The conditioning coach clears them to have the stamina and necessary aerobic and anaerobic conditioning to withstand practice and competition. The coaches then determine when the performance level is back to the level needed to contribute on the field. I have always said that, in general, the medical team clears the player to practice and the coaches determine who actually plays. But as you see, clearance requires a great deal of communication and clarity of responsibility. As an orthopedist, I do not have the ability to determine when one is ready to return to play (RTP). That is a team decision and is based on functional recovery and performance measures as noted.

Never Talk to the Media: Enough said! There is never really much good that can come out of talking to the media about players. Obviously HIPPA violations come into play also. Only

once have I (David Porter, MD, PhD) spoken to the media regarding an athlete that I have treated. It was a situation where a professional football player had injured his foot, and there was so much controversy in the media regarding the diagnosis and the treatment, the player and the team asked me to make a statement. We formulated the consultation in comment/statement with the player and the team so that we were all on the same page. Other than that situation, I fortunately have stayed out of the media.

LUAN, PARENT OF ELITE ATHLETES SAMANTHA AND JESSICA PESZEK

Communication: As with most things, communication is essential to treating patients, especially when the patients are elite athletes that are surrounded by an entire team of various professionals who are supporting their recoveries. After a thorough evaluation and delivering the diagnosis to the “team,” the acting physician must be able to delineate a clear plan of action so the patient, coaching staff, athletic trainers, and other members of the team are attentive to the same treatment plan outlined by the physician. Open and constant communication not only ensures the proper training and treatment of the athlete but demonstrates a mutual respect among all team members. If there was ever lack of or miscommunication, the strength and effectiveness of the team would suffer tremendously, impairing the recovery of the athlete and bringing fear and anxiety to the athlete’s parents.

Teamwork: Imagine a group of well-intended, skillful professionals all trying to accomplish a common goal, yet all trying to do it on their own. While an individual’s skill-set may complete one aspect of the goal, more than likely the overall objective would never be accomplished because key contributors to the process are missing. The same principle can be applied to treating patients, especially treating elite athletes, where multiple disciplines are involved. Teamwork is fundamental to achieving the goal of returning the player to full participation. The athlete, coaches, ATCs, parents, and physicians collaborating as an efficient and reliable team is a requirement so the athlete can progress. Should there be any miscommunication, lack of mutual respect, or any other detrimental cause to effective teamwork, the patient’s outcome would not be realized to its highest potential. For example, there was miscommunication on the severity of a stress fracture. This could lead to inappropriate training and worsening of the injury. However, had all members of the “team” made sure to clarify the situation, a setback in recovery could have been avoided (Fig. 32.6).

Accessibility: Players, families, coaches, trainers, and team managers all rely on the physician to be readily available to answer any number of questions or solve any problems that may arise throughout treatment. Accessibility comes in many forms. Can you be reached? Can you work the patient into the schedule during a hectic day at the office in a timely fashion? The answer to those questions should be “yes” the majority of the time. Elite athletes cannot miss a thing. Time is crucial not only to their recovery but to developing their skills on and off the field. Aside



Fig. 32.6 Luan and Ed Peszek, mother and father of Samantha (2015 NCAA all-around champion-UCLA, 2008 Olympic silver medalist, 2007 World Championships gold medalist) and Jessica (Division 1 Western Michigan gymnast, MAC Senior Gymnast of the Year). (Courtesy of and permission by Luan Peszek Image courtesy of Luan Peszek)

from delaying their return, inaccessibly could lead the patient to believe that there is a lack of urgency or empathy for his or her situation. An injury or setback of any kind places great stress on the player, coaching staff, parents, and managerial team. Thus physicians needs to confirm that they are dedicated to the patient and his or her needs.

Trust: Their safety is in our hands. Patients (and parents) put their wellbeing, even their careers, in the hands of physicians. They trust not only that the medical professionals are competent and highly skilled at what they do, but they also trust that

providers will do right by them. Having the best interest of the patient, and validating that dedication to his or her safety, is vital to creating a trusting patient-provider relationship. If the patient and/or the parents of that athlete feel that a doctor is just going through the motions and only sees a problem to be fixed, not a person who needs healing, a wedge of distrust can be driven between them. Trust fosters an ideal environment for healing, as there is no second-guessing or hesitation to follow the best plan of action for the quickest recovery set forth by the physician. Parents just want their children well cared for and to have a “normal” life if possible!

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