PRIMARY CARE OF FOOT AND ANKLE INJURIES IN THE ATHLETE

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PRIMARY CARE OF THE INJURED ATHLETE, PART I

PRIMARY CARE OF FOOT AND ANKLE INJURIES IN THE ATHLETE

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There has been a growth in the number of athletically related injuries to the foot and ankle and a coincident increase in public interest for specialists to provide the necessary treatment. [14] **Most of these injuries are sprains and strains that can be treated by the primary physician; however, more serious and potentially debilitating injuries can present as common sprains and strains. This article gives the treating physician a general overview of the more common foot and ankle problems seen in the athlete.**

LATERAL ANKLE SPRAINS

Lateral ankle sprains are the most common injury in sports, especially basketball, soccer, cross-country running, and dance and ballet. [8] [27] [28] [32] [40] [42] [45] Studies in Norway and Finland reported that acute ankle sprains accounted for 16% and 21% of all athletic injuries, respectively. [42] [60] In

basketball, ankle sprains account for 45% of all injuries, and in soccer 17% to 31% of all injuries are ankle sprains. [24] [27] [60]

Proper diagnosis of musculoskeletal injury depends on a working knowledge of anatomy. Therefore, it is imperative that the primary

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physician, whether an emergency physician, family physician, pediatrician, or orthopedist, understands the ligamentous and topographic anatomy. With this knowledge, the lateral ankle ligaments include the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL). The ATFL blends with the anterior lateral capsule, is 1.5 to 2.0 cm long, originates at the anterior distal fibula, and inserts on the body of the talus just anterior to the articular facet. The CFL lies just deep to the peroneal tendon sheath. It originates from the anterior distal border of the distal malleolus just below the ATFL (not on the apex of the tip of the fibula as often espoused), runs 10 to 45 degrees posterior to the longitudinal axis of the fibula, and inserts on a small tubercle posterior and superior to the peroneal tubercle of the calcaneus. The PTFL is rarely completely torn and more rarely reconstructed. Its exact anatomy is described elsewhere. [14]

Injury to the lateral ligaments of the ankle typically involves the unloaded foot and ankle (or, more accurately, just at the moment of loading) with a plantar flexion and inversion force. In plantar flexion the ATFL is taut and the CFL is relatively loose, whereas in dorsiflexion the converse is true. Also, the ATFL has a lower load to failure than the CFL (138 N versus 345 N). These observations help to explain the greater frequency of injuries to the ATFL, followed by the CFL, and the relative infrequency of injuries to the PTFL. The joint stabilizing function of the ligaments is most important in the unloaded ankle joint, because in the loaded extremity the bony configuration contributes greatly to its stability. [45] [64] Most of these injuries are midsubstance, but bony avulsion of the talus or fibula does occur and should be noted on routine radiographs. Clinical presentation of a "typical" ankle sprain usually involves the athlete describing a popping or tearing sensation with pain and loss of support. With specific questioning, the athlete describes the characteristic inversion, plantar flexion, or internal rotation mechanism. The immediate swelling can be very localized or more diffuse. Patients with a complete tear of one or more ligaments will often describe difficulty bearing weight. On examination, an anterior drawer test (performed in plantar flexion to isolate the ATFL) will elicit pain in athletes with a ATFL injury, and if a complete tear exists, anterior subluxation of the talus will present with a suction sign (depression over the ATFL with anterior subluxation). An inversion or varus stress on the calcaneus with the ankle held in neutral or slight dorsiflexion will induce pain or demonstrate instability (or both) in patients with a CFL injury. It has been our experience that meticulous fingertip examination of all potentially injured structures leads the physician to the correct clinical diagnosis.

Our radiographic routine is anterior-posterior (AP), lateral, and

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oblique (mortise) views of the ankle. Standing radiographs can provide physiologic stressing of the foot and ankle that is important in identifying ligamentous instability, such as diastasis of the mortise between the tibia and fibula. We very seldom use MR imaging, typically only in the more chronic setting to rule out an occult osteochondral lesion (OCL) of the talus, or when a tendon injury is suspected. A CT scan is used only when more specific bony anatomy is needed for surgical definition or to define occult fractures. CT is our radiograph of choice for a defined OCL of the talus that is seen on plain radiographs. Stress radiographs of the ankle (talar tilt, stress anterior drawer) are typically obtained under anesthesia only when surgical reconstruction is undertaken in chronic recurring lateral ankle instability to document the nature (tibiotalar, subtalar, or combined) and the degree of the instability.

There are numerous classification systems for lateral ankle sprains. We favor a practical system based on the presence or absence of instability by anterior drawer testing. If the ankle is unstable, treatment by immobilization with a walking boot or cast for at least 3 weeks is preferable. In a stable ankle, symptomatic treatment is adequate, and a stirrup brace is quite useful. Physical therapy can be started immediately, consisting of edema control and protected range-of-motion (ROM) exercises. Achilles stretching and peroneal strengthening are usually not tolerated well until after the first week of recovery.

Operative treatment for an ankle sprain is, in most instances, reserved for the patient with chronic recurring instability who has failed adequate rehabilitation. We prefer an anatomic reconstruction as described by Brostrom [9] and modified by Gould. [29] Because reconstruction is as predictable in the chronic setting as in the acute setting and because 90% of these patients will not experience chronic instability, we believe nonoperative treatment is most reasonable. Rarely, we recommend surgical treatment for the acute sprain, in which case we use the following criteria: (1) a severe sprain with marked instability in a young competitive athlete with stress radiographs demonstrating greater than 15 degrees of talar tilt and greater than 10 mm of anterior translation on anterior drawer (acute repair of the ATFL and CFL is performed in this setting); (2) an acute sprain in an athlete with a history of chronic instability; (3) the presence of an acute osteochondral fracture on ankle radiographs requiring surgical intervention; or (4) widening of the ankle mortise as described later in this article. Our specific surgical technique and postoperative care are described elsewhere. [14]

CHRONIC LATERAL ANKLE SPRAINS

Chronic symptoms following a lateral ankle sprain require special attention and careful evaluation. Patients with chronic instability typically

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present with either an acute sprain, which the athlete relates as a recurring problem, or with a complaint of looseness of the ankle and frequent "turning under" or "giving away." These athletes rarely complain of pain. The complaint of pain should raise suspicion for another

diagnosis or an associated injury. Evaluation should include a careful review of the mechanism of injury and symptom complex, a careful discriminating examination localizing the point(s) of maximal tenderness and type of instability, and radiographs of the ankle or foot (or both) depending on the examination. We routinely obtain stress radiographs (talar tilt, drawer, and stress Broden's view [14]) in the office in the chronic setting to assess the exact area(s) of instability. If plain radiographs suggest an associated OCL lesion of the talus, a CT scan is ordered. If our examination suggests associated intra-articular disease but the plain radiographs and examination are not diagnostic, then we proceed with a bone scan to help differentiate between a bony and soft-tissue cause. Examination of the athlete with chronic lateral instability should reveal clear, increased excursion on the anterior drawer (assuming the opposite ankle is stable) and increased inversion on varus stressing as compared with the opposite, uninjured ankle. Failure to demonstrate this instability should make the examiner question his or her diagnosis. More than one office visit may be necessary to fully appreciate the dynamics of the particular athlete's disorder and situation. During this familiarization process, we typically initiate an aggressive physical therapy program including proprioception training, aggressive Achilles tendon stretching, and progressive resistance exercises for peroneal strengthening. Occasionally, this will be all that is needed to relieve the athlete's symptoms. We recommend a lace-up brace or velcro strap or taping for these athletes during all athletic activity for the remainder of their career.

When chronic lateral instability remains despite nonoperative treatment, we prefer an anatomic reconstruction as described earlier (Brostrom and Clanton/Shon). If there is concern for intra-articular disease in addition to the lateral instability, we perform an arthroscopic examination of the ankle prior to the ligament reconstruction and treat the intra-articular disorder at that time. We have noted excellent results with the anatomic reconstruction and are pleased with this approach in the athlete because it does not sacrifice tendinous structures, and it allows for regaining essentially normal ROM. This is especially important in dancers [31] and skaters. Reconstruction of the CFL ligament can be difficult and tedious, but if the tissues are reasonable and if good firm sutures can be placed in the substance of the ligament, we have found excellent stability is restored to the subtalar joint as well as lateral ankle with this approach. In a few instances, we have noted that the ATFL and CFL are so attenuated that a strong reconstruction cannot reliably be obtained. In this instance, we augment the reconstruction

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with a portion of the peroneus brevis in the fashion of a modified Chrisman-Snook or modified Evans procedure. [12] [14] We also recommend this same augmentation procedure for the following athletes: (1) those with generalized ligamentous laxity; (2) those who have failed a previous anatomic reconstruction; and (3) the very large athlete (exceeding 250 pounds) who does not require extremes of motions (as in football linemen).

Two situations deserve attention in athletes with chronic instability or failed previous attempts at lateral reconstruction. The first is the athlete with a cavovarus foot. This is defined as a varus hindfoot, where the alignment of the calcaneus to the long axis of the lower leg is neutral or in varus when viewed from behind the standing athlete. These athletes need a calcaneal osteotomy

[21] to place the hindfoot in 5 to 7 degrees of valgus in addition to the lateral reconstruction procedure. The second anatomic variant is tarsal coalition in conjunction with chronic lateral instability. Careful assessment of subtalar motion must be undertaken to rule out this anomaly prior to treatment. Rigidity of the subtalar joint places added stress on the lateral ankle ligaments. Resection of the coalition needs to be undertaken prior to definitive treatment of the ankle instability. Failure to recognize these associated bony abnormalities will result in almost certain surgical failure, regardless of the reconstructive procedure attempted.

WHEN AN ANKLE SPRAIN IS NOT AN ANKLE SPRAIN

Because lateral ankle sprains are such a common injury in the athlete, there is an understandable tendency to view all injuries around the hindfoot and ankle as an "ankle sprain." Also, the athlete is often unable to recall or describe the exact mechanism of injury, thus clouding the history. Several aspects of the athlete's symptoms are particularly suggestive of an injury other than, or in addition to, an ankle sprain: (1) the inability to bear weight on the foot and ankle (especially after encouraging the athlete to do this); (2) *any* deformity to the foot or ankle; (3) severe midfoot swelling or *any* blistering of the skin (suggesting significant skin stretching usually associated with joint subluxation, dislocation, or fracture); (4) in the chronic setting, the principal complaint being pain rather than instability or giving away; (5) the athlete who continues to hurt excessively 3 to 4 weeks following sprain. These are certainly not the only warning signs to look out for, but we have found them helpful. We will now discuss these other injuries to rule out when examining each athlete suspected of having a lateral "ankle sprain."

Atypical Ankle Sprains

We group the following four injuries about the ankle as "atypical sprains" because they represent a different mechanism of injury, their prognosis is more guarded, and the severity of injury is typically greater than for the routine lateral ankle sprain.

Medial Ankle Sprain

Medial ankle sprains involve injury to the medial, deltoid-shaped, talocrural ligaments (deltoid ligament). Most often, deltoid ligament injuries are associated with concomitant injury to the lateral ligaments or fibula. Isolated deltoid ligament injuries do occur but constitute less than 10% of all ankle sprains. It is rare to have chronic deltoid insufficiency, but subtle cases may be overlooked. The deltoid is composed of both superficial and deep portions. The superficial portion is thought to be one structure with many insertions. The most anterior portion inserts on the tarsal navicular both medially and plantarward, and it acts as a strut for the spring ligament (calcaneonavicular ligament). This anterior portion is thought to contribute to resisting abnormal external rotation to the midfoot, and it may be important in stabilizing the midfoot against collapse in abduction.

Athletes who suffer an isolated deltoid injury typically have a forced eversion injury such as landing from a long jump with the foot abducted or landing on another player's foot, forcing eversion. More commonly, deltoid injury involves a concomitant fibula fracture, syndesmotic injury, or severe lateral ligament injury. Evaluation must also exclude posterior tibial tendon injury, [54] flexor hallucis longus injury, and posterior tibial and saphenous nerve traction injury. Careful local palpation to identify tender structures as well as checking specific tendon function aids in this process. A squeeze test and external rotation test (see Syndesmosis Sprains) must be performed on each athlete to rule out syndesmotic injury. Radiographic evaluation suggestive of a medial sprain includes 2- to 3-mm widening of the medial clear space, displaced lateral malleolus fracture, or a syndesmosis disruption. A valgus AP stress radiograph can also demonstrate an isolated deltoid disruption, but this is quite uncommon. An MR scan is the definitive radiograph for deltoid disruption, but it is rarely needed.

Treatment of an isolated deltoid sprain without instability (grade I) involves functional management in a stirrup brace, with the recognition that return to sports is generally more delayed (3 to 6 weeks) than a lateral sprain (1 to 3 weeks). Grade II to III isolated deltoid sprains are extremely rare. We have not found it necessary to perform operative repair in these athletes; however, immobilization is longer (6 to 8 weeks)

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and more comprehensive (walking boot or half-cast [56]) because external rotation must be prohibited to allow healing of the anterior deltoid. [53] We believe that there may be more of a rotational component to the injury than has been previously proposed, but this has not been proved. We perform open repair of the deltoid ligament anytime we are unable to obtain anatomic alignment of the medial clear space on the mortise view. This is most commonly due to a concomitant fibula fracture with malalignment.

Chronic deltoid insufficiency is an uncommon problem, but it is very disabling when it occurs. Most commonly, we have seen this insufficiency with a malunion of a fibula fracture. The malunion does not allow the talus to "sit in" the ankle mortise normally and results in the deltoid healing in a lengthened position. Conservative measures can be attempted such as taping, rehabilitation, casting, or orthoses (ankle-foot-orthoses). These measures typically are not satisfactory for the athlete. Operative repair involves a derotational and lengthening fibular osteotomy and either imbrication of the deltoid (if tissues are adequate [20]) or reconstruction with the flexor digitorum longus tendon or a portion of the posterior tibial tendon. [36] [39] [41] [70]

Syndesmosis Sprains

Injury to the syndesmotic ligaments of the ankle can present with a range from pain along the interosseus membrane without diastasis between the tibia and fibula at the ankle (grade I) to frank ligament rupture with diastasis (grade III, Fig. 1) . A great deal of information exists regarding syndesmosis injury related to ankle fractures; however, there is very little information regarding "isolated" syndesmosis injuries. Syndesmotic injury results in considerably more impairment than does the lateral ankle sprain. This is another injury that can be readily

underdiagnosed if specific attention is not paid to the physical examination. More provocative tests for the syndesmotic injury include (1) stabilizing the lower leg with one hand while applying an external rotation force to the ankle (external rotation test), and (2) compressing the proximal tibia and fibula while asking about pain at the ankle (squeeze test).

The incidence of syndesmosis sprains in athletes is controversial. Two recent studies noted that 18% of ankle sprains in professional athletes were classified as syndesmosis sprains, [7] and as many as 32% of professional football players demonstrated calcification of the distal tibiofibular syndesmosis. Hopkinson and coauthors, [34] however, noted only a 15% incidence of syndesmosis sprains among 1344 ankle sprains in military cadets. We have seen about one to two cases of syndesmosis sprain without fracture per year over the last 10 years.

The syndesmotic ligaments consist of the anterior inferior tibiofibular

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Figure 1. Ankle radiographs of an athlete with a syndesmosis injury. *A,* Non-weight-bearing radiograph with subtle widening of medial clear space *(arrow). B,* Stress radiographs of same athlete's ankle demonstrating diastasis and obvious widening of medial clear space *(arrow).*

ligament (AITFL--most commonly injured portion of the ligament and source of anterolateral ankle impingement), the posterior inferior tibiofibular ligament (PITFL--least injured portion of the ligament), and the interosseus ligament (IOM--primary bond between the tibia and fibula).

Routine radiography is essential in this injury to assess for fractures; bony avulsions (10% to 50% occurrence primarily off the tibia) [5] [7] [26] ; and, most important, the mortise alignment of the tibia, talus, and fibula. We recommend weightbearing radiographs to allow for a physiologic stressing of the ankle joint. If there is a high index of suspicion for a syndesmotic injury and the weightbearing radiographs are negative for diastasis, then stress radiographs either under anesthesia or with a high ankle block are undertaken to rule out a "latent" diastasis. [23] We use the same radiograph criterion in the stressed and unstressed radiographs to assess the mortise, as described originally by Harper and Keller. [33] The mortise is abnormal if (1) the tibiofibular clear space is greater than 6 mm on AP and mortise views; (2) there is less than 6 mm of overlap of the tibia and fibula at the fibularis incisura on AP view; and (3) less

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than 1 mm of tibia and fibula overlap on the mortise view. If there is any question, we obtain comparable radiographs of the opposite, uninjured ankle for comparison, and we would consider greater than 1 mm of side-to-side difference as abnormal. If there is still a question, a bone scan or MR imaging can provide more definitive information.

Treatment of the syndesmotic ankle sprain *without* fracture depends on the extent of the injury. Our classification is described elsewhere. [14] For the purpose of this short discussion, we will present primarily the acute injury. For the sprain with negative routine *and* stress radiographs (stable without ligament tears), we treat with a fracture brace initially (typically 3 to 4 weeks, then a stirrup brace for an additional 6 weeks), cold therapy until the swelling is reduced, and weight-bearing initially with crutches, with weaning off of the crutches as tolerated. Follow-up weightbearing radiographs must be obtained at 2 weeks to confirm continued stability of the mortise. Return to sports is often as long as 3 months from initiation of treatment. For the sprain with negative routine radiographs *but* widening of the mortise on stress radiographs (latent diastasis), we recommend operative stabilization. We use two 4.5-mm screws placed across three cortices with the ankle in maximal dorsiflexion. The two syndesmosis screws are placed through a four-hole one-third tubular plate that is affixed to the lateral fibula with two unicortical screws. The foot is immobilized in neutral to slight internal rotation for 4 weeks and then placed in a fracture walker for weight-bearing initiated at 4 weeks with progression to full weight-bearing at 6 weeks. Range-of-motion exercise is initiated at 2 weeks. We use a removable half cast or a fracture walker from 2 to 6 weeks. The two syndesmosis screws are removed at 12 to 16 weeks. The plate is left in place to decrease the risk of a stress fracture through the two syndesmosis screw holes. Return to sports is typically 6 months from initiation of treatment. For the frank diastasis evident on routine radiographs, we treat with operative stabilization, the same as for the latent diastasis. Occasionally, it is necessary in the more severe diastasis to repair the deltoid medially, because the anterior deltoid is particularly important in counteracting the external rotation forces.

Subacute and chronic syndesmosis sprains are beyond the scope of this article. We have discussed our approach to these difficult problems elsewhere. [14]

Maissoneuve Fracture

Syndesmosis sprains can also occur *with* fractures. The sports medicine physician must be aware of the Maissoneuve variant of the syndesmosis injury. This injury typically involves complete rupture of the deltoid ligament, the AITFL, the interosseus membrane and a proximal

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fibula fracture. This injury is inherently unstable and must be recognized. However, a routine non-weight-bearing ankle radiograph will not demonstrate the proximal fibula fracture and spontaneous reduction can often occur. Treatment of this serious ankle injury is the same as the frank diastasis *without* fracture. We do not think there is a place for non-operative treatment in this variant of the syndesmosis sprain.

Subtalar Sprains

Only recently has subtalar instability/sprains been noted as a specific isolated area of pathology. [65] The specific ligaments involved in the lateral subtalar joint are the CFL (spanning both the ankle and subtalar joints), the inferior extensor retinaculum (used to augment the Brostrom ankle reconstruction), the lateral talocalcaneal ligament (TCL), the cervical ligament (just anterior to

the TCL), and the interosseus ligament. It is essentially impossible from the history alone to distinguish between lateral ankle instability and subtalar instability. Both conditions give the athlete the sensation of the ankle "turning under" or "turning over" during sports. Patients with chronic subtalar instability will note that they look at the surface when walking, that they are uncomfortable walking on uneven surfaces. Runners with this problem will not run at night. Physical examination of the athlete with subtalar instability varies only slightly from that in ankle instability; note that the conditions can coexist. The anterior drawer should be negative in isolated subtalar instability but positive with ankle instability; however, a subtle finding with subtalar instability is increased rotation of the calcaneus under the talus on an anterior drawer test. The varus stress test will usually demonstrate laxity comparable to that of the opposite side, but this also can be subtle. The definitive diagnosis in our hands is made by stress radiographs of the subtalar joint (loss of parallelism to the posterior facet of the subtalar joint with greater than 3 degrees of angulation on the stress Broden view and/or anterior translation of the calcaneus on the talus during the anterior drawer lateral projection [14]). These findings occur only in the more severe cases of subtalar instability. Less severe forms are difficult to demonstrate, and this can still be a diagnosis of exclusion.

Our approach to the acute subtalar sprain is similar to that for an acute ankle sprain, consisting of peroneal strengthening, Achilles tendon stretching, proprioceptive training, and the use of a brace. For the patient who presents with chronic instability, we still will attempt these conservative measures because we have seen athletes return to sports in the chronic setting after these nonoperative measures. In the chronic setting, however, these measures are often inadequate to regain stability for the athlete. [61] Our experience has been that most individuals who

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present for surgical treatment can be treated with the Brostrom procedure, provided the calcaneofibular ligament is addressed and the Gould procedure is used as reinforcement. Rarely, we will add a peroneus brevis transfer as described earlier.

A related condition is sinus tarsi syndrome. The pathophysiology is thought to involve scarring or degenerative changes in the soft-tissue elements of the sinus tarsi. We contend that, to make this diagnosis, the team physician must rule out subtalar instability and any nerve entrapment pain (superficial peroneal nerve [SPN] or lateral branch of deep peroneal nerve [DPN]), which can mimic sinus tarsi syndrome. Examination should confirm a lack of instability and a lack of pain over the SPN and DPN. Radiographic assessment should also demonstrate normal stress radiographs. We have not typically utilized subtalar arthrograms in our diagnostic algorithm, but the literature does note a relationship between sinus tarsi syndrome and a loss of microrecess projections within the subtalar joint. [47] [66] As we have become more discriminating and specific with our examination, we have made this diagnosis less frequently; however, in the patient who does fit the picture of sinus tarsi syndrome, we rely heavily on injection of anesthetic and cortisone into the sinus tarsi. If this does not give at least temporary relief, then the diagnosis must be questioned. In some athletes, one to three injections can give permanent relief. If pain

recurs after temporary relief with injections, we use surgical intervention similar to that described by Regnauld, [58] with preservation of the interosseus and cervical ligaments.

Peroneal Tendon Subluxation and Dislocation

Subluxation or dislocation of the peroneal tendons is another uncommon but easily misdiagnosed injury. In the acute setting it is often thought to be a lateral ankle sprain. The subluxation or dislocation can almost always be traced to a single traumatic event. Snow skiing (71%) is the most common sport of injury, and football (7%) is a distant second. [14] Congenital and habitual dislocations have been described but will not be discussed here.

The peroneus longus and brevis are positioned posterior to the distal end of the fibula in the retrofibular sulcus. The tendons are contained in a fibroseus tunnel, with the superior peroneal retinaculum as the most important stabilizing soft-tissue structure. [52] We use the classification system of Eckert and Davis. [22] In grade I injury, the retinaculum is subperiosteally stripped off the posterolateral distal fibula. In grade II injury, the retinaculum is stripped off with a cartilaginous ridge. In grade III injury, the retinaculum is stripped off with a bony rim avulsion. Tears of the retinaculum typically do not occur. Attritional

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tears of either the longus (less common) or brevis (more common) do occur and need to be recognized if surgery is required.

The mechanism of injury involves a sudden dorsiflexion stress with a violent reflex contraction of the peroneal musculature. [22] The patient is often at a loss to explain exactly what happened. Careful examination will reveal maximal tenderness and swelling posterior to the fibula or along its posterior border (or both). Ligamentous examination is negative. A hallmark of the examination is extreme discomfort or apprehension during attempted eversion of the foot against resistance. It should be recognized, however, that rarely do the tendons frankly dislocate with this office maneuver. In the chronic setting, the athlete often complains primarily of instability or a potential giving away or slippage around the ankle with little discomfort. The foot and ankle can, and often do, appear entirely normal in the chronic case. Asking the athlete in the chronic setting to maximally dorsiflex and evert the ankle will often reproduce the subluxation or dislocation, confirming the diagnosis.

Radiographs are typically normal unless there has been a bony rim avulsion, which is present in 15% to 50% of cases. [57] Because of the high rate of recurrence in the athletically active population, even with adequate nonoperative treatment, the authors favor surgical treatment for the acute injury. In the acute setting, our surgical approach, regardless of the grade of injury, involves direct repair of the retinaculum and periosteum back to bone, using slowly absorbing O or OO suture through three or four drill holes in the posterolateral fibula. In the acute setting, we have not found it necessary to address a shallow or convex fibular groove. The postoperative management involves non-weight-bearing in a splint for 7 to 10 days, after which the athlete is placed in a walking splint for 4 to 6 weeks. Return to sports is typically 3 to 4 months.

In the chronic setting, nonoperative treatment has little to offer the athlete other than reduction of the inflammatory symptoms, which tend to recur after treatment is discontinued. Our approach to the chronic subluxing or dislocating patient involves a groove-deepening procedure and repair or reconstruction of the superior peroneal retinaculum. [14] [62] [71] The postoperative rehabilitation is the same as that for the acute repair, except that return to sports is slightly more prolonged at 4 to 6 months.

Occult Fractures About the Ankle

Several avulsion and chip fractures about the ankle can initially appear like a typical lateral ankle sprain; however, the treatment and the prognosis can be quite different than that for a sprain. These fractures include the anterior process of the calcaneus, the lateral process of

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the talus, the posterior process of the talus (os trigonum), and osteochondral lesions of the talus.

Anterior Process Fractures of the Calcaneus

The anterior process of the calcaneus is the superiormost projection of the calcaneus articulation with the cuboid. Key ligamentous attachments to the anterior process include the superficial inferior extensor retinaculum, the cervical ligament, and the bifurcate ligament with its projections to the cuboid and navicular. The mechanisms of injury with anterior process fracture are similar to that for lateral ankle sprains (plantar flexion and inversion). Another fracture pattern also exists involving a compression force across the calcaneocuboid joint in association with forceful abduction of the foot (the "nutcracker injury"). Physical examination will reveal swelling and tenderness 1.5 to 2.5 cm distal and slightly inferior to the lateral malleolus. This is somewhat more inferior and anterior than that seen with a lateral ankle sprain. The anterior process is best visualized on the lateral view of the foot or ankle (Fig. 2) and the oblique view of the foot, which avoids overlapping with the talar neck. The fracture cannot be appreciated on AP view of the foot or ankle, nor on mortise view of the ankle. CT is the optimal examination assessing the actual fragment size. In difficult to diagnose cases, a bone scan is helpful. Small fractures with little displacement and little joint involvement can be treated nonoperatively with a stirrup brace or short term in a walking fracture boot. If chronic symptoms develop, then debridement of the small fragment usually alleviates the pain. If the fragment is large and displaced and has significant joint involvement (40%), then we advise open reduction and internal fixation (ORIF) with minifragment screws. Early accelerated midfoot and hindfoot ROM exercises need to be initiated to prevent stiffness.

Lateral Process Fracture of the Talus

The lateral process of the talus is the wedge-shaped lateral portion of the talar body where it extends from the inferior aspect of the fibulotalar articulation to the undersurface of the talus. Thus, this structure incorporates two articular surfaces: (1) the fibulotalar recess, also called the "lateral gutter of the ankle joint," and (2) the posterior facet of the subtalar joint. Fracture of this process is thought to result from inversion and dorsiflexion, which can result in a compressive

force being delivered to the lateral talus. The athlete may complain of significant pain with ambulation and significant ecchymosis. Examination will reveal minimal tenderness at the ATFL, a negative anterior drawer, pain on varus stressing, tenderness just distal to the tip of the fibula (similar

Figure 2. Anterior process fracture of calcaneus. *A*, Fracture line of anterior process of calcaneus *(fine arrow)* with attached bifurcate ligament *(heavy arrow)*. *B*, Lateral radiograph of ankle noting anterior process fracture of calcaneus *(arrow)*.

to the area of tenderness for a CFL injury), and sometimes palpable crepitation. Close attention to the AP and mortise views of the ankle will often reveal the fracture (Fig. 3) . This fracture is not well seen on the lateral ankle view. A 45-degree internal rotation view with the foot in equinus will better demonstrate the fracture if the mortise view is inconclusive. CT with frontal plane cuts is the imaging method of choice to assess the size of the fragment and any comminution (see Fig. 3) . Similar to the anterior process fracture, small undisplaced fractures can

Figure 3. Athlete with lateral process fracture of talus. *A*, Anterioposterior radiograph of ankle demonstrating lateral process fracture of talus noted just below tip of fibula *(arrow)*. *B*, CT scan of same athlete's ankle demonstrating size of lateral process fracture of the talus *(arrow)*.

be treated symptomatically with weight-bearing fracture braces and rehabilitation. Chronic pain can develop from nonunion of even small fragments and these require debridement. [18] [35] The only notable ligament attaching to the lateral process is the lateral talocalcaneal ligament. It is not particularly important in providing stability; therefore, debridement usually does not lead to instability. Large fragments can involve up to 40% of the subtalar joint, and we recommend ORIF with small fragment screws or K-wires or both. It is very important to begin early motion of the ankle and subtalar joint because stiffness is common. Despite optimal treatment, the athlete needs to understand that chronic lateral pain can develop after this fracture.

Posterior Process Fracture of the Talus (Os Trigonum)

The posterior process of the talus is made up of the posterolateral and posteromedial tubercles, with the latter being more prominent and more commonly symptomatic. Acute fractures have been described; however, our experience has been almost exclusively with chronic impingement in the plantar flexion athlete (ballet, skating). A separate

ossicle is found in 10% of the population [10] and can be asymptomatic. Symptomatic impingement results from repeated posterior compression in plantar flexion, as in the ballet dancer assuming the pointe or demipointe position. The athlete will complain of posterior ankle pain rather than anterolateral pain as with chronic lateral ankle instability. The athlete with a painful os trigonum will not report instability but will note pain as the principal complaint. Examination will reveal no instability but pain either posterolateral or, more commonly, posteromedial. The pain is located anterior to the Achilles tendon and can be well localized with careful palpation. A period of nonoperative treatment consisting of anti-inflammatory medications, resting from the extreme of plantar flexion, taping or bracing to limit extreme plantar flexion, and judicious cortisone injections can be effective in relieving the symptoms. If these nonoperative measures fail, we excise the fragment, approaching it [68] from the most symptomatic side. If surgery is undertaken, one must avoid the sural nerve on the lateral side and the posterior tibial neurovascular structures on the medial side. A stirrup brace is recommended during the first 4 to 6 weeks of recovery. We have not significantly immobilized these athletes postoperatively and have not had any complications. Return to sports is possible after 6 to 8 weeks.

Osteochondral Lesions of the Talus

Osteochondral lesions (OCL) *of the talus* is the preferred term now for a number of conditions termed *osteochondritis dissecans of the talus, osteochondral fractures,* and *talar transchondral fracture*. Osteochondral lesions typically appear in the younger athlete and the young adult. The exact cause is still not defined but is believed to be related to trauma, either from an acute injury or repetitive microtrauma. The location of the lesions is either posteromedial or anterolateral (2:1, medial to lateral [49]). Evaluation of children and adolescents with ankle pain must include this diagnosis. It is notable in these younger athletes that their complaint centers more around pain than the ankle giving away. The athlete describes pain "inside" the ankle rather than anterolateral as with an ankle sprain. Aching and stiffness are common complaints, with the pain decreasing with rest. This disorder is more commonly seen in a chronic setting and can be associated with chronic lateral instability, further supporting the theory of repeated microtrauma as the cause. Examination occasionally reveals an effusion. An anterolateral lesion will have palpable tenderness along the anterolateral dome of talus best appreciated with the ankle in plantar flexion. The examination must differentiate talar dome pain and AITFL pain. A posteromedial lesion is more difficult to palpate. Extreme plantar flexion of the ankle can often deliver the posteromedial lesion anterior enough to palpate tenderness

just in front of the tibial lip and medial malleolus. Alternatively, one can maximally dorsiflex the ankle and locate tenderness posterior to the medial malleolus. These athletes often have subtle lateral instability and occasionally have frank instability. Also, there have been reported cases in which normal-appearing radiographs later demonstrate an osteochondral lesion. Therefore, even

if initial radiographs were negative for an OCL, if the athlete has continued "pain," then we repeat plain radiographs with a plantar flexion mortise view. Our approach to the athlete with chronic ankle pain with negative radiographs is to first perform a bone scan with coned-down views of the foot and ankle. [50] Our radiograph of choice for a defined osteochondral lesion is a CT scan because it gives the best bony detail, and we believe MR imaging is overly sensitive for the extent of the lesion. If the plain radiographs are negative, then we recommend MR imaging to define the lesion. Berndt and Hardy [4] first classified OCL of the talus based on standard radiographs. We utilize both this classification and a classification based on CT. Stage I represents a small compression fracture without displacement. On CT scan the lesion is often noted to be cystic, and at arthroscopy the cartilage is noted to be intact. Stage II represents an undisplaced osteochondral fragment. CT evaluation will demonstrate significant subchondral bone attachment with sclerosis but without displacement. Arthroscopic evaluation will typically demonstrate a rent in the cartilage surface. Stage III represents an osteochondral fragment that is loose but hinged in place in its talar bed. CT evaluation will demonstrate the osteochondral fragment and its displacement within the donor bed but not a frank loose body. Arthroscopy will confirm that the fragment is loose but still hinged by cartilage. Stage IV represents an osteochondral loose body. CT evaluation will document the location of the loose body and the bony defect. Arthroscopic evaluation will confirm the osteochondral defect and the loose body. Typically, unless there was an acute fracture, the loose fragment will not perfectly match the donor defect.

Treatment centers around the patient's symptoms. An acute osteochondral fracture is treated surgically with ankle arthroscopy, anatomic reduction with absorbable pins if the fragment is large, and excision if the fragment is small, followed by toe touch weight-bearing for 4 to 6 weeks with immediate ROM. Return to sports is typically in 3 months. We have seen very few acute osteochondral fractures, and this is not the norm for OCL of the talus. If the athlete is not having mechanical symptoms, has minimal to no effusion, and has pain controlled by activity modification, then we first attempt nonoperative treatment (usually stage I or II). We recommend cross training, ROM exercises, limiting all sports activity to nonimpact and nonpainful activity for 2 months, and then functional progression. We have not used casting in these

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athletes because we believe cartilage nutrition produced by ROM activity is important for healing and cartilage nourishment. For the patient who does not demonstrate improvement with this approach, or for the athlete who has mechanical symptoms, such as locking or catching and persistent effusions, we recommend operative treatment. We perform all of our operative treatment arthroscopically. For stage I lesions, we perform antegrade drilling of the defect, which is usually identified by softness under intact cartilage. The athlete is kept toe touch weight-bearing 4 to 6 weeks, then gradually returned to weightbearing over 2 to 3 weeks. Return to sports is in about 3 months. For stages I to IV, we debride the fragment and drill the defect and rehabilitate the same as for stage I. We have not used retrograde drilling or subchondral bone grafting.

MIDFOOT INJURIES

Injuries to the midfoot in the athlete vary from metatarsal and tarsal stress fractures to mild tarsometatarsal sprains to frank fractures and Lisfranc dislocations. Similarly, the treatment, recovery, and prognosis range from simple to complex. The more severe injuries can result in career-ending injuries and long-term degenerative arthritis. Thus, the physician attending to these athletes must be able to distinguish the precise injury, recognize its severity, and institute appropriate initial and definitive treatment.

Lisfranc Ligament Injuries

The tarsometatarsal (TMT) joints consist of the base of the five metatarsals and their articulations with the three cunieforms and the cuboid. In the athlete, injuries to these joints are often radiographically subtle and easily overlooked (Fig. 4) . Therefore, a high index of suspicion must be maintained. The most common Lisfranc joint injured in the athlete is the second TMT joint. In this injury pattern, the base of the second metatarsal is displaced dorsally and laterally, resulting in a diastasis between the first and second metatarsals. It should be noted that the second metatarsal base "sits" more proximal in the shorter second cuneiform, and the second TMT joint stability is the key to the structure of entire transverse and longitudinal arch of the foot. In fact, Cain and Seligson [11] reported that no significant dislocation of the TMT joints can occur unless the second is dislocated. Dislocations of the Lisfranc joints typically occur dorsally and laterally because the plantar ligaments are much stronger than the dorsal ligaments. [16] The mechanism

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Figure 4. Standing anterior posterior radiographs of the foot noting subtle diastasis at the Lisfranc joint. *A*, A 3- to 4-mm widening between medial cuneiform and base of second metatarsal *(arrows)*. *B*, Normal joint space at Lisfranc joint in uninjured foot of same athlete *(arrows)*.

of injury in sports is typically an indirect axial load on a foot in the equinus position. More specifically, in football, a player's foot is planted on the ground in a extreme plantarflexed position and another player lands on the heel, producing the extreme axial load required to collapse the foot. [1] Similarly, injury can occur when the athlete's foot accepts an axial load when landing in the extreme pointe position, such as in the performing arts or skating.

The athlete usually complains of severe midfoot pain with the inability to bear full weight. At times, the athlete relates symptoms of paresthesias also. If there has been a complete dislocation, there will be marked swelling and, in some cases, fracture blisters. In the athlete, the injury is typically less severe than the pattern seen from motor vehicle accidents. In the athlete, this injury ranges from a sprain without any instability of the joint to moderate subluxation and occasionally frank dislocation. The hallmark of the examination is tenderness directly over the involved TMT joints. Swelling is related to the degree of the injury. Stressing of the TMT joint elicits severe pain from movement of the involved ray(s).

Radiographs (weight-bearing, if the athlete is able) are essential to

diagnose the initial amount of diastasis (see Fig. 4) . Radiographs are not normal unless there is absolute anatomic alignment of each TMT joint viewed on lateral, AP, and oblique views. Meyerson and coworkers [48] have noted that a small chip of bone off the lateral aspect of the medial cunieform is highly suggestive of a Lisfranc dislocation and should be recognized as such. Other subtle findings on plain radiographs are increased space between the medial and intercuneiform (middle cuneiform), fractures at the base of the second metatarsal, loss of any varus to the first TMT joint alignment (compared with the opposite side), and *any* dorsal subluxation of the first metatarsal on the medial cunieform noted on the lateral radiograph. Stein [63] reported the following constant anatomic relationships, and we use these criteria for determining a negative radiograph: (1) the medial border of the fourth metatarsal forms a continuous line with the medial border of the cuboid; (2) the lateral border of the third metatarsal forms a straight line with the lateral border of the lateral cuneiform; (3) on AP view, the medial border of the second metatarsal forms a continuous straight line with the medial border of the middle cunieform; (4) the first metatarsal aligns itself medially and laterally with the medial cunieform. If any of these anatomic alignments are interrupted, then a Lisfranc disruption has occurred.

Treatment depends on the degree of the injury. Essentially, any disatasis noted on plain radiographs (greater than 1 mm) requires operative reduction and screw fixation in the athlete. We recommend open reduction of the joint, debridement of any bony chips, anatomic joint alignment, and stabilization with 4.5-mm cannulated screws. Anatomic alignment is confirmed with intraoperative plain radiographs. We use a non-weight-bearing splint for 2 weeks and then transfer the athlete to either a fracture walker or a half cast and allow ROM exercises and picking up objects with the toes. It is important to encourage ROM of the toes even while in the splint. The athlete is progressed to partial weight bearing in a fracture walker after 6 weeks. Protected weight-bearing is continued until the screws are removed at 12 weeks. Return to sports is typically at 4 to 5 months.

Injury to the tarsometatarsal ligaments without instability is treated nonoperatively. Radiographs must be scrutinized with rigid adherence to the above criteria. Comparison views are necessary if any question exists regarding instability (see Fig. 4) . CT is beneficial for questionable cases. We treat the stable sprain with cold therapy, custom or off-the-shelf inserts, extended steel shanks (either within the shoe or as an insert), arch taping, and cross training. If the athlete is unable to bear weight comfortably, then we immobilize with a fracture walker. As symptoms resolve, we progress from exercise in water to biking in a stiff shoe to closed chain exercises as the athlete can tolerate before the

athlete returns to sports. Most athletes will benefit from a stiff-soled shoe and semirigid inserts for 12 months. Injury to the TMT joint ligaments can also involve the lateral joints, a so-called lateral Lisfranc injury. In this case, injury involves a forced supination to the foot, with injury to the 4 to 5 TMT joints. Frank dislocation is unusual, and the injury can be treated similar to the medial Lisfranc without diastasis. In these athletes, we add a cuboid pad to relieve the lateral TMT joint ligaments.

Navicular Stress Fracture

Navicular stress fractures have recently become more prevalent. Whether this is due to an increase in awareness of the fracture or an increase in work load on athletes who now train all year around is not known. Stress fractures in the foot are common in the running athlete; however, navicular stress fractures have been noted primarily in the more explosive athletes, such as basketball players, hurdlers, high jumpers, and sprinters, and less so in football players and long-distance athletes. The athlete describes an insidious onset of symptoms, with aching in the medial foot or ankle. Occasionally, there will be a history of an aggravating injury but, uniformly, direct questioning will uncover prodromal symptoms. On examination, the athlete has pain on palpation of the navicular, especially on the lateral border of the naviculum, lateral to the anterior tibial tendon. We have found it helpful to locate the naviculum by noting the insertion of the posterior tibial tendon on the navicular tuberosity and following the contour of the bone laterally. It is important to differentiate this injury from a Lisfranc sprain, a medial ankle sprain, and a posterior tibial tendon injury. Radiographs of the foot should confirm the diagnosis (Fig. 5) . This fracture is not well visualized on a lateral ankle radiograph; therefore, it can be missed if only ankle radiographs are obtained. If the history of pain is recent, the radiographs may be inconclusive. In this instance we use a bone scan. If the bone scan shows uptake in the midfoot, we obtain a CT scan with 2-3 mm contiguous cuts through the navicular (see Fig. 5) . If the bone scan is negative, the injury is considered a soft-tissue problem. If the CT scan demonstrates a stress fracture, it can be either complete or incomplete. An incomplete fracture will be located on the dorsal talar side of the naviculum.

The literature is sparse regarding treatment. Khan and coworkers [38] recommend a non-weight-bearing cast for 6 weeks and report near-uniform resolution with this approach. Our experience has not been so satisfatory. For the patient with only a stress reaction (positive bone scan with a negative CT scan), we remove the athlete from the offending activity--for example, no jumping or sprinting for 3 to 4 weeks--and

Figure 5. Athlete with complete navicular stress fracture. *A*, Radiograph with complete navicular stress fracture *(arrow)*. *B*, CT scan of same athlete more clearly demonstrating navicular stress fracture *(arrow)*.

use an arch support and insert an extended steel shank into the shoe to decrease the midfoot stress. Cross-training is allowed and aqua running allows the track athlete to continue to work on from without weight bearing. For the athlete with a complete fracture (see Fig. 5) , short leg casting for 6 weeks has not resulted in reliable healing. In the preseason or in-season athlete with a complete fracture, we have tried to get the athlete through the season with the same approach as used with a stress reaction. After the season, these athletes usually require operative intervention with a distal tibial onlay corticocancellous bone graft and two 4.0-mm cannulated screws. Return to sports after this surgery is 6 to 9 months. For athletes with an incomplete fracture, we have them use an arch support (custom or off the shelf) in a fracture walking boot for 4 to 6 weeks or a half cast with crutches. This allows the athlete to continue to perform ROM exercises and counteract the disuse changes that occur with continuous cast immobilization. These athletes can be offered an external bone growth stimulator also.

Metatarsal Fractures

Fifth Metatarsal Fractures

Fractures at the base of the fifth metatarsal are the most common metatarsal fractures that induce concern in the sports medicine physician.

This is because transverse fractures at the metaphyseal diaphyseal junction have a high incidence of nonunion. The injury is commonly termed the "Jones fracture" after Sir Robert Jones, [37] who described the injury in his own foot. Vascular compromise and the rigid stability of the base fragment with increased mobility of the shaft fragment distally contribute to the poor healing and high nonunion rate. Fortunately, most fractures of the fifth metatarsal are metaphyseal tuberosity avulsion fractures that typically heal uneventfully. A useful classification of these fractures is described by DeLee. [19]

The mechanism of injury is similar for each of these fractures and involves violent indirect forces. The athlete usually describes a plantar flexion, inversion, and adduction force such as landing from a jump. The vertical load on the foot at the moment of impact places severe stresses on the lateral foot, resulting in either an avulsion of the tuberosity or the classic Jones fracture. The history of any prodromal symptoms (aching in the lateral foot) is particularly important with the Jones fracture. This history with radiographic evidence of medullary sclerosis confirms prior stress reaction or stress fracture. This scenario portends a high nonunion rate, and the injury must be treated more aggressively. Examiantion will reveal point tenderness on the base and absence of tenderness over the lateral ligaments of the ankle. Careful assessment of the peroneal tendons will rule out tendon tears. Radiography will confirm the fracture, its location, and thus its classification.

We treat the tuberosity fracture with a removable fracture boot, early rehabilitation, and return to sports on a symptomatic basis with a stiff-soled shoe, supportive taping, and a cuboid pad to relieve pressure on the fracture site. Return to sports is in 3 to 6 weeks. An intra-articular

tuberosity fracture with significant displacement (2 to 3 mm) would merit ORIF with minifragment screws, but we have not had experience treating this fracture in the athlete.

Fracture at the metaphyseal diaphyseal junction can be either an acute fracture without prodromal symptoms and no medullary sclerosis, or an acute fracture on an existing stress fracture, or a chronic stress fracture without an acute event. In the competitive athlete, we treat each of these injuries the same. We believe these fractures require intramedullary stabilization to create stability for reliable healing, to allow accelerated rehabilitation, and to decrease the time lost from sports. We use a 4.5- or 5.5-mm ASIF cannulated cancellous screw placed by using image intensification. Postoperatively, the foot is splinted for 2 weeks. Partial weight-bearing is then initiated in a walking boot. The athlete works on ROM, mild strengthening, and pool therapy. The athlete is allowed to return to sports when pain at the fracture site and over the incision allows, typically 6 to 8 weeks. We have not used bone grafting except when there has been a prior failed surgical procedure. In the middle-aged

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recreational athlete who suffers an acute fracture and prefers a nonoperative approach, we treat with 4 weeks of non-weight-bearing immobilization followed by 4 weeks in a fracture walker. This approach can work well in this population.

Second Metatarsal Stress Fractures (Dancers' Fractures)

We treat these fractures nonoperatively as described by O'Malley et al. [51]

HEEL PAIN AND ACHILLES TENDON RUPTURE

We subdivide heel pain into posterior heel pain and plantar heel pain. Posterior heel pain typically involves either insertional Achilles tendinitis or acute Achilles rupture. Plantar heel pain is very common, and plantar fascilitis or calcaneal apophysitis is the usual diagnosis.

Posterior Heel Pain

Insertional Achilles Tendinitis

Insertional Achilles tendinitis can be either acute or chronic. It is common in runners and impact-loading athletes and is associated with jumping, as in basketball, dancing, and racquet sports. Acute Achilles tendinitis is commonly associated with a recent history of an increase in the work load of the athlete, such as sudden increase in mileage for a runner, or increase in intensity, or a change in the exercise environment, such as initiating hill work, changing the running surface, or changing footwear. The athlete reports aching or burning pain in the posterior heel and on examination has localized tenderness at the bony insertion or 1 to 3 cm above the insertion. There can also be associated retrocalcaneal bursitis. Radiographs are typically normal except for the occasional prominent posterior superior calcaneus (Haglund deformity [30]). Acute Achilles tendinitis usually responds to correcting the training error, Achilles tendon stretching, heel lift, anti-inflammatory medication, and a short period of cross-training. Occasionally, the acute

process is so severe that the athlete limps with ambulation. We then recommend a short period of immobilization in a fracture boot in conjunction with the preceding protocol. Icing is also beneficial in the acute setting. We do not operate on acute Achilles tendinitis.

In the chronic setting, the acute process has never been eliminated, or, more commonly, the process has become recurrent and episodic.

Clinically, the athlete reports pain at the posterior heel. Often the pain is worse after exercise and may become constant. With chronicity, the tendon becomes thickened, the pain is usually localized to the posterolateral heel, and the gastrocsoleus complex is tight. With the knee straight, the athlete is unable to get the foot to neutral dorsiflexion. Radiographically, there is usually a Haglund deformity, and often calcific spurring occurs at the bone-tendon interface. Nonoperative treatment is similar to that in the acute setting. Pathologically, the tendon demonstrates chronic degeneration rather than inflammation. We also recommend a heel wedge in the chronic setting. If acute inflammation exists in the chronic setting, we immobilize in a walking fracture boot until the acute process has resolved, usually 2 to 4 weeks.

If the nonoperative protocol fails to resolve the symptoms after 3 to 6 months, then operative intervention may be necessary. In the younger athlete (less than 50 years of age), we prefer a single posterior tendon splitting approach directly over the area of point tenderness. [46] We resect the area of tendon degeneration and the calcific deposit and remove the Haglund deformity through the single incision. Postoperatively, we splint the ankle in neutral for 2 weeks, then place the athlete in a walking boot or half cast for an additional 3 to 4 weeks. We allow cross-training, early motion, and gentle Achilles tendon stretching at 2 to 4 weeks, depending on the amount of insertion detached. In the older athlete (greater than 50 years of age), if we find greater than 50% of the tendon is degenerative, then we perform a flexor hallucis longus tendon transfer. [69] The rehabilitation is more prolonged, [69] and often the strength returns to only 70% to 80% of the uninvolved side. If in the older athlete the degeneration is localized and involves less than 50% of the insertion, we treat it the same as in the younger athlete.

Acute Achilles Tendon Rupture

The acute Achilles tendon rupture has been well described. The injury must be ruled out in any athlete with an acutely swollen ankle, especially if the athlete reports a "pop" in the posterior ankle or reports a feeling of being shot or kicked in the heel. Squeezing the calf with the athlete prone will demonstrate a lack of plantar flexion (Thompson test [67]). An ankle sprain is the most common initial diagnosis in athletes with a neglected Achilles tendon rupture, [55] so this diagnosis can and will be missed. In the athletic population, we favor open repair with early motion. [43] For the neglected rupture less than 12 weeks from the time of injury, we are able to perform a primary repair and utilize the interposed vascular scar as reconstruction material. [55] Rehabilitation is somewhat slower, but the long-term results can be similar to those for

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the acute repair. If the injury is greater than 12 weeks from the time of surgery, then we perform a flexor hallucis longus tendon transfer. [69]

Plantar Heel Pain

Plantar Fasciitis

Plantar fasciitis involves chronic inflammation of its origin at the plantar medial calcaneal tuberosity on the anteromedial aspect of heel. In the chronic setting, entrapment of the first branch of the lateral plantar nerve (FBLPN) can contribute to the pain syndrome. In proximal plantar fasciitis, the athlete notes pain at the plantar medial heel that is worse after rest and with the first steps in the morning. The pain actually lessens with activity only to recur after rest. In the chronic setting, the athlete can complain of radiating pain up the medial side of the heel and occasionally across the lateral side of the foot. It is important to question the athlete regarding lateral swelling of the heel that would suggest a stress fracture of the calcaneus and radicular symptoms suggestive of a discogenic source of pain. In the more chronic setting, some athletes will unconsciously walk on the lateral side of the foot to relieve the plantar medial pain and present with lateral foot pain. Physical examination will demonstrate pain at the plantar medial origin of the fascia, and if the FBLPN is involved, tenderness will also be noted at the proximal superior abductor hallucis muscle. Associated findings include a tight Achilles. There is no consistent alignment abnormality noted with plantar fasciitis, but hindfoot valgus with a pronation deformity increases the stress on the medial plantar fascia. Tenderness with side-to-side compression indicates a stress fracture. A positive straight leg raise indicates a herniated disc. Radiographs are taken to rule out a calcaneal stress fracture and to assess for calcaneal spurs.

Plantar fasciitis, even in the chronic setting (up to 9 months), is treated nonoperatively. Aggressive Achilles tendon stretching (ATS) is the mainstay of curing this aggravating syndrome. We spend a significant amount of time educating our athletes on the relationship between a tight Achilles tendon and plantar fasciitis. We have found that athletes must believe that ATS will cure the ailment before they will comply with the stretching. Also, we advocate an aggressive stretching program of 2 to 4 minutes of passive stretching three or four times a day. We also use nonsteroidal anti-inflammatory medications, soft heel pads (silastic), and arch taping to help with the symptoms during athletic activity. We will occasionally prescribe custom orthotics for the in-season athlete with a medial heel wedge and first metatarsal lift to relieve the medial fascia and correct pronation deformity. Judicious

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injections with long-acting steroid preparations may benefit the in-season athlete (usually one to three per year). We have noted excellent results with this approach in approximately 95% of patients. [17]

We reserve operative treatment for athletes who have failed an aggressive nonoperative approach as outlined previously for 9 to 12 months. Occasionally, because of timing for the competitive athlete, we will proceed with an operative release of the proximal fascia and release of the deep abductor fascia after 6 months of treatment. Our operative approach is open, as described by Baxter and Pfeffer. [3] If a spur is noted on the lateral radiograph, it is resected. Postoperatively, we splint the ankle with a molded arch for 2 weeks, then begin mild stretching and ambulation with crutches in a walking boot. Crutches are discontinued after 3 to 4 weeks. Biking in the boot and running in a pool are allowed at 3 weeks, with gradual increase in impact activities over 3 to 4 weeks. Running and jumping are initiated at 8 to 12 weeks depending on symptoms. Return to sports is in 3 to 4 months.

Calcaneal Apophysitis

Calcaneal apophysitis (Sever's disease) presents in a similar fashion in the skeletally immature athlete, but the pain can be posterior or inferior. Treatment is similar to the nonoperative approach used for acute Achilles tendinitis. We rarely use a custom orthosis but prefer a Silastic heel lift. Ice is very beneficial in these athletes. Furthermore, activity limitations (cross training, low-impact aerobic training, and avoidance of aggravating activities) are often required to alleviate symptoms. These young athletes are able to resume unlimited pain-free activities by 2 to 3 months in most cases.

ANKLE IMPINGEMENT

Anterior Ankle Impingement

Anterocentral and anteromedial impingement is typically bony impingement, whereas anterolateral impingement typically involves soft tissue (see next section). Anterocentral and anteromedial ankle impingement results from repeated bony impingement between the talar neck and the anterior lip of the tibia. This repeated bony impingement stimulates the cambium layer to form osteophytes. Once the osteophytes begin to form, they often grow and increase the impingement symptoms and inhibit dorsiflexion.

The athlete with anterior bony ankle impingement complains of vague anterior pain, especially with landing and forced dorsiflexion. It

is common in basketball, soccer, and dance. On examination, the athlete's pain is reproduced by forced dorsiflexion or with squatting or pile. With experience, the spurs can be palpated, and this is especially important for the anteromedial impingement, which is not well visualized on radiographs. Anterocentral impingement is well visualized on a lateral radiograph of the ankle. The impingement can be documented with a forced dorsiflexion lateral, but this is not necessary to make the diagnosis if the remainder of the history and examination are conclusive. Anterocentral spurring can be located on the distal anterior tibia only, the superior talar neck only, or both the tibia and talus. Anteromedial impingement involves impingement between the

anterior medial malleolus and the medial neck of the talus, and it often cannot be visualized on standard radiographs.

For bony anterior ankle impingement, nonoperative treatment has little to offer. For athletes in their final competitive season, a 3/8- to ½-inch heel lift can be tried to keep the athlete from "bottoming out." Operative resection of the offending spurs is the mainstay of treatment. For spurs located on the tibia only, we use arthroscopic resection and have been pleased with our results. If there is spurring both on the tibia and the talus or just on the talus, we use a small arthrotomy. Osteophytes on the talar neck are often located within the capsule and are difficult to remove safely and quickly through the scope. We have noted little difference in recovery time using the open approach for these lesions, and the operative time is less. Postoperatively, we treat all patients the same regardless of the spur location. We immobilize the ankle for 1 week in a splint and use cold compression for pain and swelling. At 1 week we use a soft dressing and cold compression therapy for continued edema control and begin ROM exercises stressing Achilles stretching. After 2 weeks we begin ankle strengthening. At 3 to 4 weeks we allow biking and low-impact aerobic activity. Return to sports is in 2 to 3 months.

Anterolateral Ankle Impingement

Impingement of the anterior ankle on the lateral side is due to soft-tissue hypertrophy. The anterolateral dome of the talus rubs against the hypertophied and scarred anterior inferior tibiofibular ligament (AITFL). This phenomenon has been popularized by three different authors, [21] [25] [44] but we believe these are descriptions of essentially the same entity. Anterolateral impingement is most often seen in the athlete whose ankle is not aggressively rehabilitated. The most distal portion of the AITFL then becomes hypertrophic and fibrotic. The athlete complains of anterolateral localized pain. Occasionally, the athlete reports a snapping or

popping sensation with dorsiflexion of the ankle. Examination will reveal point tenderness at the AITFL and an increase in pain with dorsiflexion of the talus.

Treatment is initiated by instituting a complete ankle rehabilitation program including an aggressive Achilles tendon stretching program to "stretch out" the AITFL, as the wider anterior talus is rotated up between the tibia and fibula. For the recalcitrant case, we use a single injection of the ligament with local anesthetic and a long-acting corticosteroid preparation to confirm our diagnosis and to decrease the inflammation and impingement. Arthroscopic resection is reserved for patients not responding to this protocol. Return to sports after arthroscopic resection is in 3 to 5 weeks.

FIRST METATARSOPHALANGEAL SPRAINS (TURF TOE)

Injury to the first metatarsophalangeal (MTP) joint is common in the athlete, especially with participation on hard surfaces while wearing flexible shoes. These injuries most often involve a sprain of the ligaments of the MTP joint. First toe sprains (turf toe) commonly result from forced

hyperextension of the MTP joint. [6] [15] [59] Forced hyperextension produces tearing first in the plantar portion of the capsuloligamentous complex at its origin from the metatarsal head and neck. The initial pain and swelling can be relatively minor but usually worsen over the first 24 hours. Clanton and coworkers [13] described three grades of injury based on clinical examination. In a grade 1 stretching injury to the capsuloligamentous complex, there is localized plantar and medial tenderness with minimal swelling and no ecchymosis, and the athlete can continue to play but with some discomfort. In a grade 2 partial tear, tenderness is more diffuse and there is intense, increased swelling and ecchymosis, with limitation of motion; the athlete is unable to play at a normal level. In a grade 3 is a complete tear of the plantar plate from the origin off the metatarsal head. It is characterized by severe, diffuse pain both dorsal and plantar, ecchymosis, and restricted ROM. The athlete cannot bear weight on the medial foot and obviously is unable to play. There may be a fractured sesamoid, separated bipartite sesamoid, or dorsal metatarsal head impaction.

Nonoperative treatment is the mainstay of care for the turf toe regardless of the severity. For the grade 1 injury, we place the athlete in a more supportive shoe with an insole incorporating a stainless steel plate in the forefoot to limit hyperextension of the MTP joint. We tape the toe also for athletic participation to further prevent extension. For grade 2 injuries, shoe wear modifications are the same as with grade 1, but enforcement of activity restrictions and protection from re-injury are

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critical. Restrictions in activity vary from a few days to 1 to 2 weeks. For the grade 3 injury, the athlete will typically require protected weight bearing with crutches for a few days to 1 week. Shoewear modifications are required as described previously, with activity restrictions extending 3 to 6 weeks. Operative treatment is reserved for the unreducible dislocation or when there is a complete tear of the plantar structures and retraction of the sesamoids is noted on standing comparison radiographs.

SUMMARY

A thorough knowledge of foot and ankle anatomy is required to allow an accurate and focused examination of the injured athlete. This short review has attempted to educate the treating physician on our approach to foot and ankle injuries commonly seen in athlete. We have tried to elucidate less common injuries that present in similar manner to the more common foot and ankle sprains and strains.

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